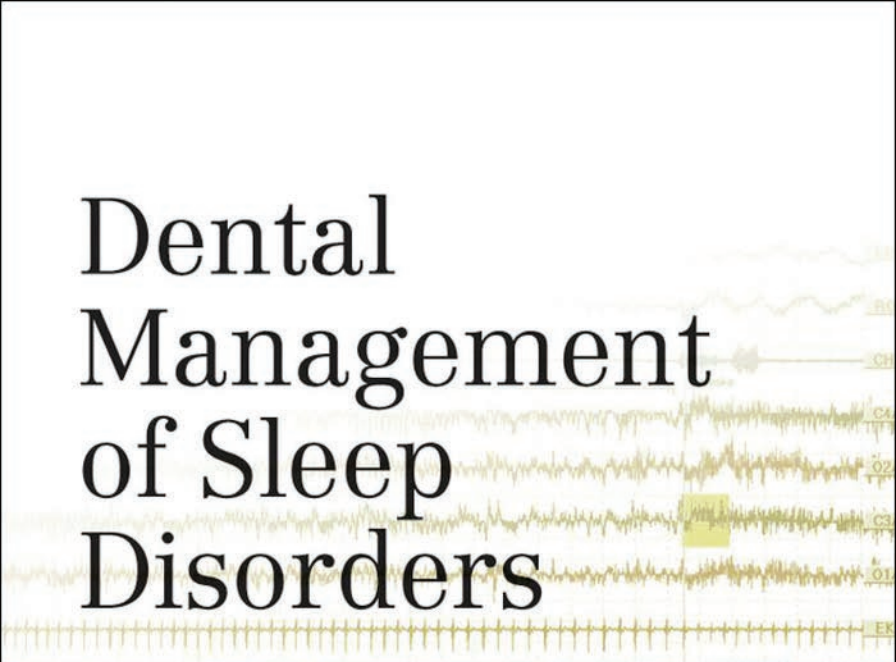


Dental Management of Sleep Disorders

A background image showing a polysomnogram (PSG) waveform, which is a series of yellow lines representing various physiological signals recorded during sleep. The waveform is overlaid on a white background.

**Ron Attanasio and
Dennis R. Bailey**

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Ronald Attanasio, DDS, MSEd, MS
Dennis R. Bailey, DDS

 **WILEY-BLACKWELL**

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Dedication

If it were not for the love, understanding, and sacrifices of our respective families, the production of this book would not have been possible. This book is dedicated to them.

Ronald Attanasio is forever grateful to his wife (Elise) for faithfulness and steadfast prayers, his children (Devon, Drew and Dani, Doron, Damaris, and D'Anna) for joy and fulfillment, his novelist brother (A.A. Attanasio) for editing contributions and keeping life's perspective balanced, his deceased parents (Joseph and Louise) for having instilled the blessings of love for life and an appreciation for dedicated work ethics, and his good friends and colleagues (Dr. David Covey and Professor Caren Barnes) for their encouragement and humor.

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Preface

Approximately 50–70 million people in the United States are chronic sufferers from sleep disorders, who have impaired health and daily functioning issues as a result of those disorders. The societal 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 to be 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.

As it relates to the recognition of health issues, the role of the dentist as well as auxiliary staff is becoming more apparent. No longer is the dentist solely relegated to only the management of dental structures and dental-related conditions. The dentist now has an ever-increasing role in the recognition of a patient who may be at risk for a sleep disorder. Epidemiologic data support the increasing awareness of the relationship of sleep disorders and other health issues. Clinical treatment and decision making now emphasize sound evidence based on documentation that relies on well-researched epidemiologic studies to assist in determining the coexistence of a sleep disorder, which in turn is impacting the health of a patient.

The dentist is more significant in comanaging these SRBD patients, especially those with snoring and/or obstructive sleep apnea (OSA). Not only does the dental practitioner have the opportunity to recognize a potential sleep disorder issue in their patients, but he or she also has the opportunity to interact with the sleep medicine field through both the referral and the provision of oral appliance therapy when indicated.

The present time is very exciting for dentistry regarding its contribution and participation in the field of sleep medicine. This book has been structured to provide the reader with an overview of sleep medicine as well as the assessment and management of the SRBD patient. There is an

ever-increasing volume of research findings regarding sleep medicine and dentistry as each year passes. It is our intent to have readers use this book as a stepping-stone along their path of lifelong learning.

Ronald Attanasio, DDS, MEd, MS
Dennis R. Bailey, DDS

Section 1

Overview of Sleep Medicine

1 Impact of sleep disorders on society

CONCEPTUAL OVERVIEW

Approximately 50–70 million people in the United States are chronic sufferers from sleep disorders, who have impaired health and daily functioning issues as a result of those disorders.¹ The societal economic impact of sleep disorders is estimated at \$16 billion annually for health care expenses and \$50 billion annually regarding lost productivity.² Sleep disorders are considered to be 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.^{3,4}

Sleep disorders can no longer be thought of in simple terms as having a poor night's sleep. There are currently a large number of different sleep disorders that may affect one's quality of life. In addition, there is a difference between the sleep state and the wake state. Sleep is not simply an altered state of consciousness, that is merely a difference of being asleep or being awake.

Sleep is a totally separate behavioral and physiological state that is unique and well documented, and it is defined as "a reversible behavioral state of perpetual disengagement from and unresponsiveness to the environment."⁵ As such, sleep is composed of a combination of rapid eye movement (REM) and nonrapid eye movement (NREM) associated with well-defined and variable brain activity.⁶ Sleep disruption and the specifically recognized sleep disorders not only may have a major impact on an individual's well-being, health status, and quality of life, but may also render significant consequences on the various areas of public health, such as

accidents, mortality, morbidity, work and other daily performance, cognitive function, and utilization of health care.

EPIDEMIOLOGY OF SLEEP DISORDERS

Epidemiology is the study of the geographic distribution and the risk factors of a particular disease,⁷ and, in particular, how that disease impacts the health status of different and varying populations. Relative to sleep disorders, the origin and onset of a specific sleep disorder are often multifactorial. As such, it is essential for there to be an epidemiological awareness of both normal and abnormal sleep and wake patterns. The ultimate goal of this greater appreciation of sleep disorder epidemiology is the improvement in people's health as well as a foundation for preventative medicine and public health.

The eventual outcome of these early epidemiologic studies of normal and abnormal sleep patterns resulted in the publication of the *International Classification of Sleep Disorders* that has since been revised into its current second edition (*ICSD-2*).⁸ The *ICSD-2* is the evidence-based standard for the classification, terminology, and diagnostic criteria of sleep disorders. Currently, the most common sleep disorders on the basis of epidemiologic studies are the following:⁹

1. Insomnia
2. Sleep-related breathing disorders (SRBD)
3. Restless legs syndrome (RLS)

The general onset of sleep disorders as well as their progression is, to some degree, dependent on age,¹⁰ the presentation of being at risk for health-related consequences, and trauma. In many instances, these disorders may appear as a health issue and perhaps even some type of emotional or psychological condition. Also, the presentation of the particular health issue is sometimes not first recognized or diagnosed as maybe having an association with a sleep disorder. As an example, SRBD patients may seek treatment and utilize more health care resources for cardiovascular disease (e.g., hypertension) prior to the recognition of the SRBD as being the possible underlying cause¹¹ (Figure 1.1).

In addition to the overall statistics of the portion of a population that suffers from the general label of sleep disorders, the prevalence of these disorders are often referenced to the epidemiologic studies pertaining to each of the classifications recognized in the *ICSD-2*. Thus, the actual distribution of a specific sleep disorder is variable and dependent on the study that is being referenced.

Relative to the three most common sleep disorders, a 1993 study is the most frequently cited reference relative to SRBD.³ In this study of 602 people, it was determined that 24% of men and 9% of women are at risk for SRBD, and it also demonstrated that 4% of men and 2% of women met the

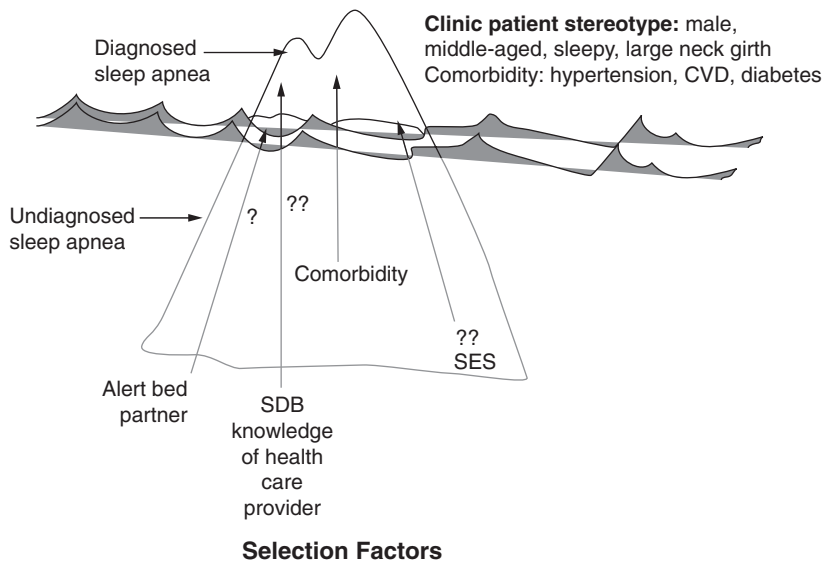


Figure 1.1 Clinical recognition and diagnosis of sleep apnea. The proportion of all cases of sleep apnea is represented by the iceberg, with clinically diagnosed cases shown in the tip of the iceberg. Clinically recognized sleep apnea represents 85% of the total prevalence of sleep apnea cases that would be candidates for treatment. CVD, cerebrovascular disease; SES, socioeconomic status. “?” and “??” indicate that these particular “selection factors” are not as well known as “comorbidity” relative to the recognition and diagnosis of sleep apnea. (Young T. Rationale, design, and findings from the Wisconsin Sleep Cohort Study. *Sleep Med Clin*, 2009; 4(1):39. Reprinted with permission.)

criteria for SRBD, who also have daytime hypersomnolence or excessive daytime sleepiness. The correlate to this is a study that indicated 93% of females and 82% of males with moderate-to-severe SRBD are undiagnosed, and 98% of females and 90% of males who have less severe sleep apnea are undiagnosed.⁴ A more recent study demonstrated that a minimum of 12–18 million people in the United States are affected by untreated SRBD.¹²

For insomnia, the prevalence may vary on the basis of the study, but, in general, it appears to be approximately 33% of the population.¹³ The percentage will vary on the basis of the defined criteria that may exist as a result of the specific study parameters.

The epidemiology of RLS is estimated to be between 2.5 and 10% of the general population.¹⁴ Many of the studies have limitations because of misdiagnoses as well as patients who may not seek medical care. With improved criteria for making a diagnosis, the recognition of RLS may actually result in an increased prevalence.

Narcolepsy is another sleep disorder that has been studied epidemiologically. At the present time, its prevalence is between 25 and 50 per 100,000 population,¹⁵ which is a prevalence similar to multiple sclerosis.

RISK FACTORS

There are a multitude of risk factors that may impact the onset as well as the progression of a specific sleep disorder. The risk factors may be different for each disorder, and there may also be overlapping risk factors that apply to a number of different sleep disorders. In general, the demands of modern-day life have impacted the quality of one's sleep as well as the required amounts that are deemed appropriate. In addition, the disruption of an individual's sleep can impact other family members, roommates, or one's bed partner. As an example, the results of a study pertaining to SRBD demonstrated that the snoring of one person significantly impacted the sleep of the bed partner to the point that the affected person had symptoms that were worse than those of the snorer.¹⁶

The risk factors frequently associated with the three most common sleep disorders are listed in Table 1.1.

HEALTH CONSEQUENCES AND COSTS

The impact of sleep disorders on one's health can be evident in a wide range of adverse health consequences, including but not limited to hypertension,¹⁷⁻¹⁹ cardiovascular diseases,²⁰⁻²⁷ metabolic disorders such as diabetes,²⁸⁻³² gastric disorders such as gastroesophageal reflux disease,³³⁻³⁶ respiratory disorders such as asthma,³⁷ emotional and psychological disorders,³⁸⁻⁴¹ and increased mortality rates.⁴² The heightened awareness that assorted health issues potentially may arise as a result of sleep disorders is improving. Similarly, an illness or health problem may conversely impact one's sleep.

The increased risks for cardiovascular disease and elevated blood pressure associated with SRBD are well-documented. The largest and frequently cited investigation, the Sleep Heart Health Study, took place between 1995 and 1998 as a cohort study with more than 6,000 people enrolled as participants.⁴³ This study found that obstructive sleep apnea (OSA) along with other SRBD are risk factors for cardiovascular disease, which is inclusive of myocardial infarction and stroke. A variety of proposed mechanisms indicate that OSA and cardiovascular disease are related, and elevated blood pressure is the more common one.

Insomnia, which is the inability to fall asleep or to maintain sleep, is often associated with emotional, psychological, or depressive disorders. In addition, it can be associated with painful conditions as well as other health issues. As a sleep disorder, insomnia is the most common and may occur in conjunction with other sleep disorders such as OSA and RLS. Everyone may have insomnia at some point in their life.

Alteration in metabolic and endocrine functions is also associated with SRBD, the most prevalent of which is type 2 diabetes. It has been found

Table 1.1 Common risk factors for the three most common sleep disorders.

Sleep apnea and sleep-related breathing disorders
Increase with age
Snoring/gasping during sleep
Associated with larger neck size
Increase with weight
Males greater than females
Airway obstruction—especially tonsils, adenoids, enlarged tongue, elongated soft palate
Hypothyroidism
Coexisting cardiovascular disease or hypertension
Allergy or asthma
Family history
Insomnia
Increases with age
Associated with anxiety and/or depression
Associated with pain, headaches, arthritis, temporomandibular disorder symptoms
Dissatisfied with one's quality of sleep
Females greater than males
Substance abuse
Perceives health as worse or bad
Level of schooling
Restless legs syndrome
Increases with age
Worsens over time—follows a chronic course
Males greater than females
Use of antidepressants
Low iron levels
Peripheral neuropathy
Pregnancy
End-stage renal disease
May coexist with sleep bruxism

that snoring alone increases the risk for type 2 diabetes independent of any other risk factors.²⁸

Health issues that impact sleep are also recognized. Classically, among these are painful conditions, increased stress, and depression. These most often lead to insomnia and a decrease in sleep time and sleep quality, and, in many instances, an associated increased time in bed.

Although sleep disorders may potentially increase the risk for a multitude of health problems, it is not an absolute that a given sleep disorder will lead to any one specific health problem. The important issue is to recognize that these health problems may have an underlying sleep disorder as a contributing factor in the development or progression of a health issue. The more common health conditions associated with sleep disorders are listed in Table 1.2.

Table 1.2 Common health issues possibly associated with a sleep disorder.

Painful conditions
Headaches
Arthritis
Fibromyalgia
Anxiety or depression
Stress
Hyperthyroidism
Pregnancy
Gastroesophageal reflux disease
Medications
Cardiovascular disease
Hypertension

There are also associations of sleep disorders with impairments in performance relative to attention and reaction time,^{44–46} which could have significant impact in occupational settings. One study demonstrated that about 20% of serious injuries from motor vehicle accidents are related to driver sleepiness and not alcohol-related.⁴⁷ Other studies have indicated the equivalence of driving when sleepy as when driving under the influence of alcohol.^{48,49}

A report by the National Sleep Foundation (NSF) indicated that (1) 60% of adult drivers stated that they have driven a vehicle during the past 12 months while feeling drowsy and (2) 37% self-reported that they fell asleep while driving at least 1–2 days/month.⁵⁰ The NSF report included an estimate by the National Highway Traffic Safety Administration that vehicular crashes related to driver fatigue result in a minimum estimation of \$12.5 billion of monetary losses to the economy. Fiscal impact on society could be higher since there is no physical test to attribute these crashes to sleepiness.

Studies have also demonstrated the association of sleep-related fatigue and work-related injuries and fatalities.^{51,52} Workers who were identified as highly fatigued had a 70% greater chance of being involved in occupation-related accidents than were workers identified with low fatigue. Another study demonstrated that workers with insomnia had a greater chance for injuries related to their occupations than workers who were identified as having good sleep.⁵³ Also, workers with disturbed sleep had twice as much chance of dying in a work-related accident than those who did not have sleep disturbance.⁵¹

Because of the increased risk for the development of health problems with sleep disorders, there may be an associated increase in health care costs as well. The presence of a sleep disorder may drive up the cost of health care in general, and it has been estimated that untreated sleep apnea may add \$16 billion in medical costs annually in the United States.²

However, at the present time, it is unclear what the actual savings in medical costs are with the management of OSA. In a study of 31 patients diagnosed with cardiovascular disease and OSA, there was a reduction in the need for hospitalization related to the cardiovascular illness when the apnea was adequately managed.⁵⁴ An association between the severity of the sleep disorder and increased health care expenditures is possible.⁵⁵

The costs of health care are increased by the lack of attention to the specific sleep disorder. A 1998 study demonstrated that 10 years prior to the actual diagnosis of OSA, the patients who were eventually diagnosed with OSA had incurred nearly double the costs for health care and had additional hospitalizations when compared to the matched controls.⁵⁶ As such, in many instances the sleep disorder actually precedes the onset of the specific health issue or consequence. If the sleep disorder is not discovered, then the attention to the related health issue takes precedent. This may then lead to an increase in expenditures for various testing, imaging, physician visits, hospitalization, and medication or prescription usage.

Along with the health issues that may arise, there is also an impact on one's quality of life. Studies that assessed parameters regarding quality of life demonstrated that patients with a sleep disorder feel that their quality of life is poorer than those without OSA.^{57,58} When the OSA is improved with treatment, both the patient and the bed partner experience an improvement in their quality of life.⁵⁹

THE ROLE OF THE DENTIST

As it relates to the recognition of health issues, the role of the dentist as well as the auxiliary staff is becoming more apparent. No longer is the dentist solely relegated to only the management of dental structures and dental-related conditions. An example is the association between periodontal disease and cardiovascular disease.

Many decades ago, the dentist's role in the overall health care of the patient was initiated by performing blood pressure screenings. Patients often saw their dentist more frequently than they saw a physician, which led to the discovery of many patients who were at risk for hypertension and yet were unaware of its existence. A commentary in the *Journal of the American Dental Association* (JADA) called for an increase in the education of the dentist in biological and medical sciences.⁶⁰ This commentary directly referenced medical topics possibly related to the dentist that appeared in JADA from 2004 to 2006, including diabetes, heart disease, cardiovascular diseases, and OSA. Another commentary noted the potential for the dentist to inform patients about possible risk factors for cardiovascular disease.⁶¹ When considering the association between SRBD and cardiovascular disease, the dentist as well as the auxiliary staff are well-poised to assist patients with their overall health.

Table 1.3 The role of the dentist.

Action	Indirect approach	Direct approach
Advise patient of potential sleep disorder	Yes	Yes
Obtain blood pressure and review medical history	Yes	Yes
Refer for further evaluation: to primary care physician or sleep specialist	Yes	Yes
Refer for sleep study	No	Yes
Based on diagnosis, actively involved with treatment	No	Yes

The role of the dentist can potentially take on one of two roles (Table 1.3). The first, referred to as the *indirect approach*, is simply the recognition of the potential existence of a sleep disorder that may be impacting the patient's health status, and the patient is subsequently advised of the situation and referred for definitive evaluation and care. The second, referred to as the *direct approach*, is when a sleep disorder is recognized and, if appropriate, the patient is referred for more comprehensive evaluation and care. This testing often involves the primary care physician and/or sleep specialist, and the care, when deemed applicable, may also include the dentist. The dental treatment typically involves the use of an oral appliance for the management of OSA. The dentist may also be involved for treatment of a painful orofacial condition that is impacting the patient's sleep.

Regardless of the role that the dentist assumes, the initial action needs to start with the recognition of the patient who may be at risk for a health problem or who may have a health problem that may have a sleep disorder as an underlying or contributing factor.

CONCLUSION

The dentist now has an ever-increasing role in the recognition of a patient who may be at risk for a sleep disorder. Epidemiologic data support the increasing awareness of the relationship of sleep disorders and other health issues. Clinical treatment and decision making now emphasize sound evidence based on documentation that relies on well-researched epidemiologic studies to assist in determining the coexistence of a sleep disorder, which in turn is impacting the health of a patient.

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Human sleep

2

CONCEPTUAL OVERVIEW

Sleep is a universal necessity of humans, and approximately one-third of the human life is spent in the sleep state. Disruption and/or deprivation of sleep typically results in adverse physiologic effects.

Despite large amounts of research and investigation into the true definition of sleep, the complete purpose for sleep is not fully understood. It is known, though, that adequate sleep is needed to maintain alertness, heal the body, and assist with memory and learning. Also, the physiologic and neurochemical activities needed for the sleep and awake states are 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.

NORMAL SLEEP

Normal sleep can be viewed from two aspects: (1) the actual distribution of sleep cycles relative to nonrapid eye movement (NREM) and rapid eye movement (REM), and (2) the neurotransmitters that affect regulation of the sleep–wake cycle. Each of these areas changes as the human progresses from infancy to elderly.

NREM and REM sleep

Structural characteristics of normal sleep can be referred to as sleep architecture, which comprises two distinct states: NREM, which is subdivided

into Stages 1–4; and REM.^{1,2} NREM and REM sleep occur in varying proportions during a sleep period, and they also alternate in a cyclical fashion with each other throughout sleep. In addition, their proportional distribution during sleep changes with age.

The typical pattern in normal sleep is for the individual to progress from wakefulness to the NREM sleep state, followed by the REM sleep state, and then cyclically alternating between REM and NREM stages. Overall, a night of sleep comprises about 75–80% of NREM sleep and 20–25% of REM sleep. When this cycling becomes irregular and/or there is a deprivation of NREM sleep stages, such as what occurs with narcolepsy, sleep disorders can develop.

Muscle activity during sleep also varies depending on REM or NREM sleep. In REM sleep, there appears to be an increase in activity in the motor centers of the brain, but there is active inhibition that is exerted on these motor neurons.

In NREM sleep, muscle activity is decreased with the maximum effect during the more restorative Stages 3 and 4.

NREM sleep

NREM sleep has historically been subdivided into four distinct stages on the basis of characteristic brain wave and physiologic activities ever since it was initially observed and measured through electroencephalography (EEG): NREM Stage 1, NREM Stage 2, NREM Stage 3, and NREM Stage 4 (Figure 2.1).^{3,4}

These four stages of NREM sleep are typically defined as follows:

Stage 1: 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. NREM Stage 1 comprises about 2–5% of the total sleep time, and it is considered to be a drowsy or 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 2: Although this begins to be a deeper stage of sleep with a reduction of heart rate and body temperature, it is still regarded to be 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 NREM Stage 1. This stage 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 and sleep spindles, the latter of which has been postulated to being associated with memory consolidation.⁵ The K-complex may appear as a result of some type of stimulation, such as noise, or it may appear spontaneously.

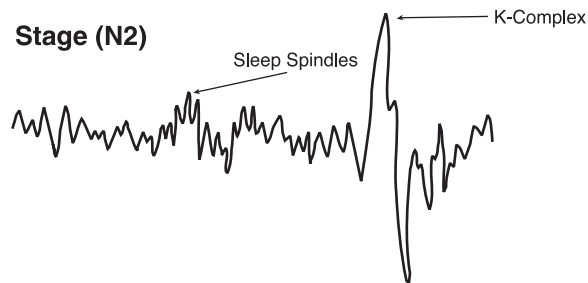
REM (Stage R)**Stage (N1)****Stage (N2)****Delta sleep (N3)****Awake**

Figure 2.1 EEG recordings of brain wave activity associated with their respective sleep stages.

Stages 3 and 4: These two NREM stages have their own unique and individually recognized brain waveforms, 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 waveform, they are also known as delta sleep. Together, they comprise about 13–23% of the total sleep time. NREM Stage 4 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 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” waveform and can also appear similar to wakefulness relative to mixed frequency. 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 an 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 their dreams during sleep.⁶ REM sleep may also be contributory to memory consolidation.^{7,8}

REM sleep may be further regarded as two phases: tonic and phasic.⁹ 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:

- Atonia (loss of muscle tone) of the skeletal muscles that appears near paralysis;
- 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,¹⁰ and sleep study reports may increasingly refer to this nomenclature as opposed to the staging that has been historically utilized in the past (Table 2.1):

- Stage W (Wakefulness)
- Stage N1 (NREM1)
- Stage N2 (NREM2)
- Stage N3 (NREM3; this replaces NREM Stages 3 and 4)
- Stage R (REM)

Table 2.1 Comparison of sleep study scoring by stages.

Previous scoring staging	Updated scoring staging
Awake	Awake
REM sleep	Stage R
NREM Stage 1	Stage N1
NREM Stage 2	Stage N2
NREM Stage 3	Stage N3
NREM Stage 4	Stage N3

Source: Adapted from Iber C, Ancoli-Israel S, Chesson AL, et al. The AASM Manual for the Scoring of Sleep and Associated Events. Westchester, IL: American Academy of Sleep Medicine. 2007.

Cycles and hours of sleep

Sleep patterns and architecture change for individuals throughout life. From infancy to elderly, these changes are dynamic and distinct, relative to sleep initiation, sleep maintenance, and the amount of time for each sleep stage.^{11,12}

During normal sleep, the human typically cycles through the NREM and REM sleep stages four to six times per sleep period. In the adult, regardless of the age, these stages occur at about 90-minute intervals. In children, these stages are shorter and occur at about 50- to 60-minute 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 hours, 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.

As we 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 (Figures 2.2 and 2.3).

As people become elderly, they often begin to adjust the time that they go to bed to an earlier and earlier hour.¹³ The result is that they have the increased potential to wake up earlier in the morning, which is a condition termed advanced sleep phase syndrome.¹⁴ In this syndrome, the individual is purposely adjusting 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.¹⁵

With the aging process, the typical sleep architecture becomes more fragmented with increased awakenings or arousals during the sleep period¹⁶ and people tend to subsequently have an increased risk for sleep disorders,^{17,18} including sleep-related breathing disorders (SRBD) and insomnia.¹⁹ With SRBD, the musculature that supports the airway

Figure not available in this electronic edition.

Figure 2.2 Architecture of sleep stages by age groups over a full night at varying hours of sleep for children, young adults, and elderly. (Kales A and Kales JD. Sleep disorders: recent findings in diagnosis and treatment of disturbed sleep. New Eng J Med. 1974; 290:487. Reprinted with permission.)

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 obstructive sleep apnea (OSA) and for the management of this sleep disorder with oral appliance therapy.

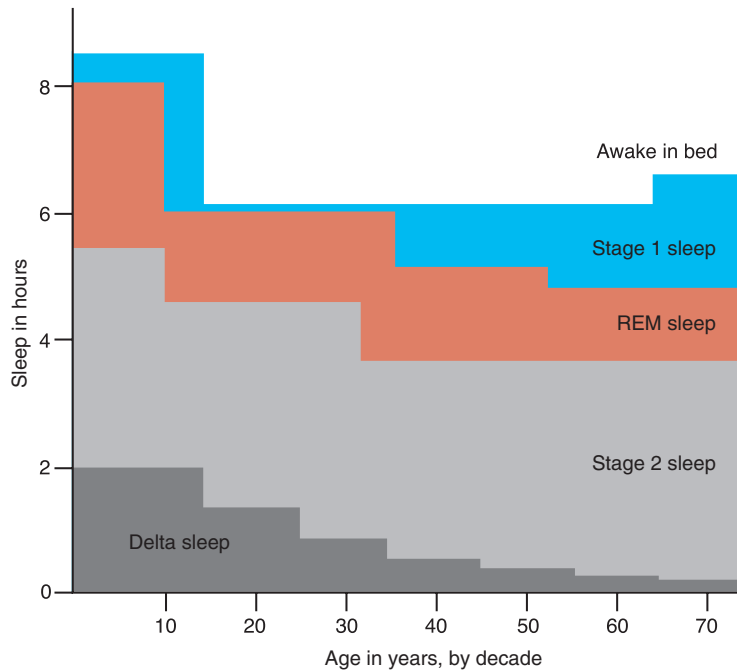


Figure 2.3 Sleep by decades. It shows the amount of sleep by stages over a lifetime and how these stages tend to change with age.

The number of hours of sleep that is considered to be optimal also varies with age. Infants and young children require more sleep time as compared to teenagers and adults (Table 2.2).

Neurotransmitters of wakefulness and sleep

The role of neurotransmitters in both the sleep and awake states is a complex topic. Because of their varying levels and interaction throughout the

Table 2.2 Optimal hours of sleep on the basis of age.

Age by years	Optimal hours
1–3	12–14
3–5	11–13
5–12	10–11
Teenagers	8.5–9.5
Adults	7–9

Source: Adapted from the National Sleep Foundation. Available at www.NSF.com, 2009.

24-hour period that impacts their function, many of the neurotransmitters may appear to have contradictory functions by impacting sleep as well as wakefulness.

The interaction of these various substances (neurohormones, neuro-modulators, and neurotransmitters) is responsible for maintaining wakefulness and initiating sleep. No one substance acts alone. Rather, a complex interaction of these is responsible to maintain the two states of wakefulness and sleep.

Neurotransmitters of wakefulness

The basic neurotransmitters pertaining to the awake state are the biogenic amines, also known as monoamines,²⁰ which have the role of either maintaining wakefulness or initiating sleep. These monoamines are further categorized into three subgroups: (1) catecholamines, in particular dopamine (DA), norepinephrine (NE), and epinephrine (adrenalin); (2) serotonin (5HT); and (3) histamine. The monoamines DA, NE, and 5HT along with another neurotransmitter, acetylcholine, have their origin in the brain stem, whereas histamine originates from the hypothalamus.

Acetylcholine

This neurotransmitter is predominately for vigilance and cortical activation. It is found at the neuromuscular junctions in the sympathetic, parasympathetic, and central nervous systems. Acetylcholine is considered to have excitatory properties, and it also plays a role in the precipitation of REM sleep, especially the phasic state. In addition, it is critical for memory function.²¹

Catecholamines

Catecholamines are found to be necessary for arousal and wakefulness, and each has unique properties:

Dopamine maintains wakefulness and can impact one's behavior. A reduction in dopamine is frequently associated with movement disorders.

Norepinephrine maintains EEG activity.

Epinephrine is unique because it is blood-borne and is only found to be at low levels in the brain. It is released from the adrenal medulla and is not known to cross the blood-brain barrier. Epinephrine is theorized to impact the sleep-wake cycle through autonomic and neuroendocrine regulation.

Serotonin (5HT)

This neurotransmitter is recognized for a wide variety of functions that are associated with mood, depression, pain, headache, and sleep. It is found to

be excitatory and can impact the awake state. However, its main role is to promote deep sleep.

Histamine

The main function of histamine is to maintain quiet wakefulness. The role of histamine was not known until it was recognized that antihistamine-containing medications could produce sleepiness. Further investigation led to the discovery of the role of histamine in the promotion of wakefulness and vigilance. It functions in a fashion similar to norepinephrine by promoting cortical activation during wakefulness.

Orexin/Hypocretin

Recently, the role of orexin (hypocretin) has also been uncovered as a neurochemical that maintains wakefulness.²² Each of these was discovered at about the same time by two different research groups, and hence the two different names for the same neurochemical. Its role is to maintain and stabilize wakefulness as well as vigilance. More important, a reduction in the presence of orexin/hypocretin is implicated in the clinical presentation of narcolepsy. Consequently, specific medications designed to influence the release of orexin/hypocretin are used for the management of narcolepsy by maintaining wakefulness and vigilance.

Cortisol

Cortisol is a hormone that is present to maintain wakefulness. This chemical substance is released by the adrenal glands and is oftentimes associated with stress. It also plays a role in maintaining alertness and may be increased in the early morning hours to promote wakefulness. However, because it will be increased with stress, it may also be associated with depression and insomnia.

Glutamate

Glutamate is a fairly significant excitatory neurotransmitter in the brain that is associated with normal brain function, and it has a basic role in the activity of the waking brain. It is present mostly during wakefulness when the largest amounts can be found.²³

Neurotransmitters and sleep

There are a number of neurotransmitters that promote sleep, including the following.

GABA

Gama-aminobutyric acid or γ -aminobutyric acid (GABA) is the main neurotransmitter of sleep, and it is released from the hypothalamus. With its greatest influence at the posterior hypothalamus, GABA inhibits the activating systems, hence promoting sleep.²⁰ Medications designed to

promote sleep, such as Ambien, Sonata, and Lunesta, do so by causing the release of GABA. Benzodiazepine-type medications are also known to increase GABA, which accounts for their sedating effects. GABA is synthesized from glutamate.

Adenosine

Adenosine is not a classic neurotransmitter. Associated with ongoing activity, it may accrue over time as a by-product of degradation from ATP. The concentration of adenosine increases with prolonged wakefulness and decreases during sleep,²⁴ and it is purported to be a key neurotransmitter in the homeostatic regulation of sleep. Adenosine seems to have an inhibitory affect in the central nervous system on acetylcholine and glutamate. In addition, it has the role of facilitating sleep along with GABA, which is exemplified by the fact that caffeine (methylxanthine) blocks adenosine receptors, thus explaining the role of caffeine as a stimulant.

Serotonin

Serotonin plays a role in the control of many different functions. It is produced from tryptophan in the pineal gland of the brain, where serotonin is further affected by norepinephrine, which in turn leads to the production of melatonin.²⁰ The role of serotonin in sleep is, therefore, directly linked to the release of melatonin. As such, the role of serotonin is related to its impact on other neurotransmitters in either an inhibitory or stimulating fashion, which accounts for its relationship with the sleep and awake states.

About 90% of serotonin is actually produced in the intestine, and the remaining 10% is found in the brain and the platelets.²⁵

Other sleep-inducing factors

Melatonin

Melatonin is a hormone that is released by the pineal gland in the brain and its major impact is on the circadian rhythm. As such, there is an association of light with the release of melatonin. Light that penetrates the eye terminates the release of melatonin. As darkness approaches, the stimulus to release melatonin increases, which promotes the sleep state.

The neurologic pathway related to the release of melatonin is not a direct one (Figure 2.4). The release of melatonin is initiated by the absence of light penetrating the eye, which then triggers neural activity that proceeds from the suprachiasmatic nucleus in the brain stem to the hypothalamus. The signal subsequently travels into the superior cervical nucleus of the spinal column and subsequently to the pineal gland.

Insulin

Insulin is known to have slow-wave (NREM Stages 3 and 4) sleep-inducing properties.²⁶ Recently, insulin receptors have been found in the brain,

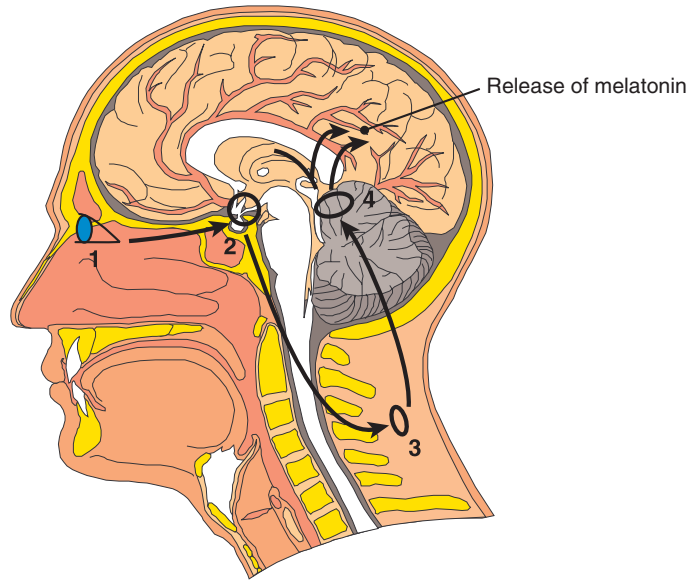


Figure 2.4 The route of neural pathway based on the amount of light entering the eye to get to the brain to impact the release of melatonin from the pineal gland to promote sleep.

which accounts for insulin's role in promoting sleep. One study has proposed a type of insulin dependence associated with the brain that has been termed diabetes type 3.²⁷ The findings are preliminary and appear to provide evidence of a relationship between sleep deprivation and Alzheimer's disease.

Muramyl peptides

These peptides originate in the gut, and they appear to have the ability to promote sleep by stimulating the production of interleukins that are known to promote slow-wave deep sleep.²⁶

ANATOMY AND FUNCTION OF THE AIRWAY

The anatomy of the airway from the dentist's perspective is mainly focused on the upper airway, especially the musculature that directly controls airway function. These muscle groups have been categorized by anatomical location,²⁸ and these same muscles are employed in the function of speaking and swallowing. In addition, the dentist should be familiar with the complex action of respiration (breathing).

Table 2.3 Muscles designed to elevate soft palate.

Muscle	Action
Levator veli palatini	Elevates the soft palate; pulls soft palate posteriorly to close the nasopharynx
Tensor veli palatini	Pulls soft palate laterally
Musculus uvulae	Elevates the uvula; pulls uvula laterally
Palatopharyngeus	Helps close the nasopharynx; elevates pharynx and larynx
Palatoglossus	Elevates tongue; narrows oropharyngeal isthmus during swallowing

Soft palate

Table 2.3 identifies the muscles designed to elevate and tense the soft palate. The palatopharyngeus and palatoglossus muscles are included with the soft palate because their origin is from the palatine aponeurosis, and the control of these muscles is the same as the musculus uvulae and levator veli palatini.

Oropharynx

The posterior portion of the airway and the tongue comprise the muscles of the oropharynx. The tongue muscles are further divided into two groups: extrinsic and intrinsic (Table 2.4).

Hyoid

The hyoid is an integral osseous structure that supports the suprahyoid and infrahyoid muscles. The position of the hyoid relative to the mandibular

Table 2.4 Muscles of the tongue.

Muscle	Action
Extrinsic	
Genioglossus	Protracts tongue; depresses tongue
Hyoglossus	Depresses tongue
Styloglossus	Retracts tongue; elevates tongue
Palatoglossus	Elevates tongue; narrows oropharyngeal isthmus during swallowing
Intrinsic	
Superior longitudinal	Shortens tongue; curls tongue apex upward
Inferior longitudinal	Shortens tongue; curls tongue apex downward
Transverse	Narrows tongue; lengthens tongue
Vertical	Broadens tongue; flattens tongue

Table 2.5 Infrahyoids and suprahyoids.

Muscle	Action
Infrahyoids	
Omothyroid	Depresses hyoid
Sternohyoid	Depresses hyoid
Sternothyroid	Depresses larynx
Thyrohyoid	Depresses hyoid
Suprahyoids	
Stylohyoid	Elevates hyoid; retracts hyoid
Mylohyoid	Elevates hyoid; elevates floor of mouth
Digastric	Elevates hyoid; aids in depression and retraction of mandible
Geniohyoid	Helps move hyoid and tongue anteriorly

plane is of importance as a reliable predictor of the risk for OSA when viewed on a cephalometric radiograph. The muscles associated with the positioning of the hyoid are divided into two groups: the infrahyoids and the suprahyoids (Table 2.5).

Pharynx

The pharynx is the portion of the airway that runs from the base of the tongue down to the larynx and the epiglottis (Table 2.6). Because the pharynx is unsupported, it is subject to narrowing or collapse with SRBD. The pharyngeal constrictor muscles function mainly during swallowing.

Other airway muscle considerations

Other muscles associated with mandibular function and the cervical spine play a role in positioning the mandible and head as well as maintaining the airway both in the awake and sleep states. The muscles include the masseter, temporalis, pterygoids (lateral and medial), and the anterior and posterior cervical groups.

Table 2.6 Muscles of the pharynx.

Muscle	Action
Superior constrictor	Constricts upper portion of pharynx
Middle constrictor	Constricts middle portion of pharynx
Inferior constrictor	Constricts lower portion of pharynx
Palatopharyngeus	Elevates pharynx; helps close nasopharynx
Salpingopharyngeus	Elevates upper and later portions of pharynx
Stylopharyngeus	Elevates pharynx; expands sides of pharynx (dilates pharynx)

The two main muscles of the neck that are considered as accessory to respiration are the sternocleidomastoid (SCM) and the scalene.²⁹ The SCM's primary purpose is to rotate, tilt, and flex the head. They also act as accessory muscles of respiration by elevating the sternum. The scalene group causes lateral flexion of the neck, and the anterior scalene specifically elevates the first rib as well as stabilizes the upper ribs with respiration.

The primary muscles of respiration during inspiration are the diaphragm and the external intercostals. During expiration, the abdominal muscles and the internal intercostals are active.

If the airway becomes compromised during inspiration, inspiratory pressures will elevate, which will result in an increased negative pressure in the airway that further compromises and possibly even collapses the airway. Many times, the muscles that are intended to support the airway as well as tongue position can no longer maintain the airway, and further collapse is inevitable. This airway collapse occurs because the musculature relaxes during sleep, thereby further complicating the compromised airway.

NERVE SUPPLY TO THE MUSCLES OF THE AIRWAY

The muscles that are involved with respiration are supplied by either cranial nerves or nerves that originate from the cervical spine (Table 2.7). Because of this neuromuscular relationship, the evaluation of patients with SRBD needs to include an assessment of the posture, cervical spine, and, when indicated, cranial nerves.

BASICS OF AIRWAY DYNAMICS

The Venturi effect and the Bernoulli's principle are two basic principles of airway dynamics. According to the Venturi effect, if the diameter of a tube (e.g., the airway) is decreased, then for a given volume of fluid to pass through the tube, the velocity has to increase. This concept applies to the airway relative to snoring. The Bernoulli's principle states that as fluid flows through a tube and with an increase in the flow of that substance, there is an increase in negative pressure at the periphery. The result is an increasing potential for airway collapse.

Another concept that can be associated with airway dynamics is that of the variable resistor. As the rate of flow increases, so does the resistance to that flow. However, the classic explanation that involves airway collapse related to flow is the Starling resistor concept. In this situation, a collapsible tube is positioned between two rigid structures. Relative to respiration, the collapsible tube is the airway between the nose and the trachea. When the airflow increases and the pressure outside the tube is greater than the pressure inside the tube, there is subsequent airway collapse. In the awake state,

Table 2.7 Nerve supply to the muscles of the upper airway.

Nerve	Muscles supplied
Trigeminal CN V	Tensor veli palatini Mylohyoid Anterior belly of digastric Masseter Medial/lateral pterygoids Temporalis
Facial CN VII	Stylohyoid Posterior belly of digastric
Glossopharyngeal CN IX	Stylopharyngeus
Pharyngeal plexus from vagus CN X	Musculus uvulae Palatoglossus
Cranial part of accessory CN XI	Palatopharyngeus Levator veli palatini Salpingopharyngeus Superior constrictor Middle constrictor
Spinal accessory CN XI	Sternocleidomastoid (SCM) Trapezius (along with C-3 and C-4 branches)
Pharyngeal plexus—external laryngeal of vagus & recurrent laryngeal of vagus	Inferior constrictor
Hypoglossal CN XII	Genioglossus Hyoglossus Styloglossus Superior longitudinal Inferior longitudinal Transverse Vertical
C-1 follows CN XII	Thyrohyoid Geniohyoid
Ansa cervicalis Motor component of cervical plexus (C-1 to C-4)	Omoxyoid Sternohyoid Sternothyroid
Ventral rami C-4 to C-6 C-5 to C-8 C-6 to C-8	Anterior scalenes Middle scalenes Posterior scalenes
Phrenic nerve from C-3 to C-5	Diaphragm

Source: Adapted from Norton NS. *Netter's Head and Neck Anatomy for Dentistry*. Philadelphia: Saunders/Elsevier. 2007.

the airway is not subject to these fluctuations in the pressure, and therefore the airway remains open. However, during sleep when the airway relaxes and negative pressure increases, the transmural forces that support the airway are not capable of resisting the negative pressure, and this leads to airway obstruction. Depending on the variation in pressure during respiration, the flow through the tube (i.e., airway) may be unimpeded, cause flutter, or become blocked. This correlates with normal breathing, snoring, and obstruction associated with apnea.³⁰

NORMAL RESPIRATION

Under normal circumstances, respiration is considered to be involuntary. It is primarily under the control of the diaphragm, which is innervated by the phrenic nerve that arises from the cervical nerves C3 to C5. It is primarily the muscle fibers that cause contraction of the diaphragm. During passive breathing, the diaphragm contracts and moves downward, causing an increase in the negative pressure within the lung and the alveoli. This negative pressure causes air to flow into the lungs to fill them. In addition, this action may be impacted by the intercostal muscles and the scalenes. Expiration during quiet breathing is passive, and there is no active muscle activity. The process is related to the elastic recoil of the lungs and the rib cage.

Forced inspiration and expiration are different. With forced inspiration, the scalene and the SCM muscles are active. They impact the first and second ribs as well as the sternum, and this causes elevation of the bony cage.

Forced expiration is primarily an action of the intercostal muscles that pull the thoracic cage inward and force air out of the lungs. They prevent the increased intrathoracic pressure from causing any bulging at the intercostal spaces.

CONCLUSION

Understanding sleep and the processes related to it is important because it helps in understanding the dynamics of sleep disorders, especially SRBD. The dentist does not have to understand the complex neurologic and neurochemical relationships that exist 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.

Humans spend nearly a fourth to a third of their lives sleeping. It is important to understand this process as much as the other physical conditions. The time that is spent sleeping has an important role in maintaining health, improving memory, enabling humans to learn, and maintaining alertness and vigilance while awake. Without quality sleep, the awake time, mood, and ability to function may be significantly impaired.

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Classification of sleep disorders

3

CONCEPTUAL OVERVIEW

Although this chapter will review the overall classification of sleep disorders, the focus will primarily be on the most common adult-related types of disorders. As with any other areas or disciplines of clinical dentistry, a classification system for sleep disorders will facilitate a comprehension of the various disorders, especially with regard to signs and symptoms, pathophysiology, etiology, and clinical management.

Along with insomnia, sleep-related breathing disorders (SRBD) constitute a significant portion of sleep disorders that may be encountered in a dental clinical practice. However, for a greater appreciation of the spectrum of sleep disorders, the practitioner is encouraged to at least be familiar with the more common sleep disorders.

CLASSIFICATION SYSTEMS

A classification system assists with the development of a differential diagnosis of any kind of health disorder.

There are four classification systems for sleep disorders that are most frequently used:

1. *Diagnostic Classification of Sleep and Arousal Disorders (DCSAD)*
2. *International Classification of Sleep Disorders, Second Edition (ICSD-2)*
3. *International Classification of Diseases (ICD)*
4. *Diagnostic and Statistical Manual of Mental Disorders (DSM)*

In general, the *DCSAD* and *ICSD-2* classification systems provide in-depth descriptive definitions that are employed for communication purposes in the clinical fields of medical sleep specialists and dentists, whereas the *ICD* versions and their numerical codes are more frequently used for medical records, billing purposes, and research focused on epidemiological studies.

DCSAD

The *Diagnostic Classification of Sleep and Arousal Disorders (DCSAD)* was the initial classification system for sleep disorders and was first published in 1979. The *DCSAD* was subsequently used as the foundation for the formation of the other classification systems.¹

ICSD/ICSD-2

The American Academy of Sleep Medicine along with the Japanese Society of Sleep Research, Latin American Sleep Society, and European Sleep Research Society published the *International Classification of Sleep Disorders (ICSD): Diagnostic and Coding Manual* in 1990,² which was later revised in 1997. These earlier versions of the *ICSD* were utilized mostly for clinical diagnostic purposes as well as enhancement of international communication relative to research in sleep disorders. Since the pathophysiology was not well understood at that time, the *ICSD* was based on the most common symptoms of dyssomnias (insomnia and excessive daytime sleepiness (EDS)) and parasomnias (abnormal physiological events) that would take place while sleeping.

The most recent edition, *International Classification of Sleep Disorders, Second Edition (ICSD-2)*, was published in 2005.³ This version provides a listing of the different sleep disorders as well as other diagnostic and epidemiologic information. There are 85 different sleep disorders in eight different major categories identified in the *ICSD-2* (Table 3.1).

ICD-9-CM/ICD-10

When the World Health Organization was formed in 1948, it published the sixth edition of the *International Classification of Diseases (ICD)* for the purposes of diagnostic coding as well as collating mortality statistics regarding all medical conditions. Prior to this edition, the classification and listing of causes of death had their origins in the 1850s.⁴ The *ICD* has since undergone ten revisions that include clinical modifications (*ICD-CM*).

Sleep disorders are classified in the ninth edition, *ICD-9-CM*, as either “the specific disorders of sleep of nonorganic origin” (coding 307.4) or “the sleep disturbances” (coding 780.5).

The *ICD-10* will eventually replace the *ICD-9-CM*.

Table 3.1 *International Classification of Sleep Disorders (ICSD-2).*

- I. Insomnias
 - Adjustment insomnia
 - Psychological insomnia
 - Paradoxical insomnia
 - Idiopathic insomnia
 - Insomnia due to mental disorder
 - Inadequate sleep hygiene
 - Insomnia due to drug or substance or alcohol use
 - Insomnia due to medical condition
 - Insomnia not due to substance or known physiological condition, unspecified (nonorganic insomnia)
 - Physiological (organic) condition, unspecified
 - II. Sleep-related breathing disorders
 - Primary central sleep apnea
 - Cheyne–Stokes breathing pattern
 - High-altitude periodic breathing
 - Central apnea due to medical condition, not Cheyne–Stokes
 - Central apnea due to drug or substance
 - Obstructive sleep apnea, adult
 - Sleep-related nonobstructive alveolar hypoventilation, idiopathic
 - Sleep-related hypoventilation/hypoxemia due to pulmonary parenchymal or vascular pathology
 - Sleep-related hypoventilation/hypoxemia due to lower airways obstruction
 - Sleep-related hypoventilation/hypoxemia due to neuromuscular and chest wall disorders
 - Other sleep-related breathing disorders
 - III. Hypersomnias
 - Narcolepsy with cataplexy
 - Narcolepsy without cataplexy
 - Narcolepsy due to medical condition without cataplexy
 - Narcolepsy due to medical condition with cataplexy
 - Narcolepsy, unspecified
 - Recurrent hypersomnia (including Kleine–Levin syndrome and menstrual-related hypersomnia)
 - Idiopathic hypersomnia with long sleep time
 - Idiopathic hypersomnia without long sleep time
 - Behaviorally induced insufficient sleep syndrome
 - Hypersomnia due to medical condition
 - Hypersomnia due to drug or substance (abuse) or alcohol use
 - Hypersomnia due to drug or substance (medications)
 - Hypersomnia not due to substance or known physiological condition
 - Physiological (organic) hypersomnia, unspecified
-

(Continued)

Table 3.1 (Continued)

- IV. Circadian rhythm sleep disorders
 - Delayed sleep phase type
 - Advanced sleep phase type
 - Irregular sleep–wake rhythm
 - Nonentrained type
 - Jet lag type
 - Shift work type
 - Circadian rhythm sleep disorder due to medical condition
 - Other circadian rhythm sleep disorder
 - Drug or substance or alcohol use
 - V. Parasomnias
 - Confusional arousals
 - Sleepwalking
 - Sleep terrors
 - REM sleep behavior disorder
 - Recurrent isolated sleep paralysis
 - Nightmare disorder
 - Sleep-related dissociative disorders
 - Sleep-related groaning
 - Exploding head syndrome
 - Sleep-related hallucinations
 - Sleep-related eating disorder
 - VI. Sleep-related movement disorders
 - Restless legs syndrome
 - Periodic limb movement disorder
 - Sleep-related leg cramps
 - Sleep-related bruxism
 - VII. Isolated symptoms, apparently normal variants, and unresolved issues
 - Long sleeper
 - Short sleeper
 - Snoring
 - Sleeptalking
 - Sleep starts (hypnic jerks)
 - Benign sleep myoclonus of infancy
 - Hypnagogic foot tremor and alternating leg muscle activation during sleep
 - Propriospinal myoclonus at sleep onset
 - Excessive fragmentary myoclonus
 - VIII. Other sleep disorders
 - Other physiological (organic) sleep disorder
 - Physiological (organic) sleep disorder, unspecified
 - Other sleep disorder not due to substance or physiological condition
 - Environmental sleep disorder
 - Other sleep disorder not due to substance or physiological condition, unspecified
-

Table 3.1 (Continued)

IX.	Sleep disorders associated with conditions classifiable elsewhere
	Fatal familial insomnia
	Fibromyalgia
	Sleep-related epilepsy
	Sleep-related headaches
	Sleep-related gastroesophageal reflux disease
	Sleep-related coronary artery ischemia
	Sleep-related abnormal swallowing, choking, and laryngospasm
X.	Other psychiatric/behavioral disorders frequently encountered in the differential diagnosis of sleep disorders
	Mood disorders
	Anxiety disorders
	Selected somatoform disorders (somatization disorder, hypochondriasis)
	Schizophrenia and other psychotic disorders
	Selected disorders usually first diagnosed in infancy, childhood, or adolescence (mental retardation, pervasive developmental disorders, attention deficit/hyperactivity disorder)
	Personality disorder

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

DSM

The American Psychiatric Association first published the *Diagnostic and Statistical Manual of Mental Disorders (DSM)* in 1952. The *DSM* has undergone several revisions since its initial publication. The *DSM Fourth Edition, Text Revision (DSM-IV-TR)* was published in 2000 and it incorporated textual descriptions for each of the sleep disorders. A 2005 publication of the classification reflects changes made to the *ICD-9-CM*.

DEFINITIONS OF COMMON SLEEP DISORDERS (BASED ON ICD-2)

Regarding frequency of sleep disorders, one report indicated that obstructive sleep apnea (OSA), narcolepsy, and restless legs syndrome (RLS) were the three most common disorders observed in a survey of sleep centers.⁵ Insomnia and other sleep disorders are also considered as frequently reported or described by patients seeking care.⁶

Since sleep disorders are common in the general population, it is prudent for the dental clinician to be familiar with the definitions of these disorders. This chapter presents the definitions of common sleep disorders on the basis of the *ICSD-2* classification.

Insomnia

Insomnia is defined by a repeated difficulty with sleep initiation, duration, consolidation, or quality that occurs despite adequate time and opportunity for sleep and results in some form of daytime impairment.³

The most commonly reported sleep disorder is insomnia.⁶ This disorder is founded on subjective reporting by the patient, and insomnia can typically include difficulty getting to sleep, awakening from sleep earlier than the desired wake-up time, difficulty in 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 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 polysomnogram (PSG) sleep study,⁸ but PSG is not the standard of care for the definitive diagnosis of insomnia (Table 3.2).

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.⁹

There are 10 types of insomnia (Table 3.1), 5 of which are further described here.

Table 3.2 ICSD-2 general criteria for insomnia.

- A. A complaint of difficulty initiating sleep, difficulty maintaining sleep, or waking up too early, or sleep that is chronically nonrestorative or poor in quality. In children, the sleep difficulty is often reported by the caretaker and may consist of observed bedtime resistance or inability to sleep independently.
- B. The sleep difficulty occurs despite adequate opportunity or circumstances for sleep.
- C. At least one of the following forms of daytime impairment related to the nighttime sleep difficulty is reported by the patient:
 - a. Fatigue or malaise
 - b. Attention, concentration, or memory impairment
 - c. Social or vocational dysfunction or poor school performance
 - d. Mood disturbance or irritability
 - e. Daytime sleepiness
 - f. Motivation, energy, or initiative reduction
 - g. Proneness for errors or accidents at work or while driving
 - h. Tension headaches, or gastrointestinal symptoms in response to sleep loss
 - i. Concerns or worries about sleep.

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Table 3.3 *ICSD-2* diagnostic criteria for psychophysiological insomnia.

-
- A. The patient's symptoms meet the criteria for insomnia.
 - B. The insomnia is present for at least 1 month.
 - C. The patient has evidence of conditioned sleep difficulty and/or heightened arousal in bed as indicated by one or more of the following:
 - a. Excessive focus on and heightened anxiety about sleep
 - b. Difficulty falling asleep in bed at the desired bedtime or during planned naps, but no difficulty falling asleep during other monotonous activities when not intending to fall asleep
 - c. Ability to sleep better away from home than at home
 - d. Mental arousal in bed characterized either by intrusive thoughts or a perceived inability to volitionally cease sleep-preventing mental activity
 - e. Heightened somatic tension in bed reflected by a perceived inability to relax the body sufficiently to allow the onset of sleep.
 - D. The sleep disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Psychophysiological insomnia

The essential feature of psychophysiological insomnia is heightened arousal and learned sleep-preventing associations that result in a complaint of insomnia and associated decreased functioning during wakefulness.³

This type of insomnia involves an overconcern or worry on the individual's part relative to his or her difficulty to adequately sleep.^{10,11} As such, it is a learned or behavioral insomnia since the individual is reacting to general psychologically stressful conditions that result in physical discomforts and a heightened level of arousal (Table 3.3).

Adjustment insomnia

The essential feature of adjustment insomnia is the presence of insomnia in association with an identifiable stressor.³

Associated with this type of insomnia is a specific stressful situation or factor that can be psychological, physiological, environmental, or physical.^{12,13} Usually, when the stressful situation resolves, so does the insomnia. Adaptation of the individual to the stress factor can also account for resolution of adjustment insomnia. As such, this type of insomnia is typically regarded as a short-term disorder, although an unaddressed stressor can cause it to develop into a more chronic condition (Table 3.4).

Table 3.4 ICSD-2 diagnostic criteria for adjustment insomnia.

-
- A. The patient's symptoms meet the criteria for insomnia.
 - B. The sleep disturbance is temporarily associated with an identifiable stressor that is psychological, psychosocial, interpersonal, environmental, or physical in nature.
 - C. The sleep disturbance is expected to resolve when the acute stressor resolves or when the individual adapts to the stressor.
 - D. The sleep disturbance lasts for less than 3 months.
 - E. The sleep disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Inadequate sleep hygiene

The essential feature of inadequate sleep hygiene is an insomnia associated with daily living activities that are inconsistent with the maintenance of good quality sleep and full daytime alertness.³

Inadequate sleep hygiene is usually associated with habits or daily activities that are not conducive to sleep or good sleep quality.^{14,15} Examples include (1) the intake of caffeine, alcohol, nicotine, or excess food too close to the desired initiation of sleep, (2) participating in physical exercise too close to the time of getting to sleep, and (3) a change in one's environment from travel or a change in one's work schedule, which can disrupt the body's circadian rhythms and associated sleep-wake cycle (Table 3.5).

Insomnia due to medical condition

The essential feature of insomnia due to a medical condition is insomnia that is caused by a coexisting medical disorder or other physiologic factor.³

Medical conditions or neurological disorders can disrupt sleep and cause insomnia.^{16,17} Examples include asthma, emphysema, bronchitis, headache, overactive thyroid, odontalgia, gastrointestinal disorders, stroke, RLS, and physical pain from fibromyalgia, myofascial pain, arthritis, temporomandibular disorders, and other medical issues.

From the dentist's perspective, the practitioner needs to discern whether a patient's chief symptoms of orofacial pain may or may not have an underlying sleep disorder as a contributing factor so that an appropriate diagnosis and management plan can be established (Table 3.6).

Table 3.5 ICSD-2 diagnostic criteria for inadequate sleep hygiene.

-
- A. The patient's symptoms meet the criteria for insomnia.
 - B. The sleep disturbance is present for at least 1 month.
 - C. Inadequate sleep hygiene practices are evident as indicated by the presence of at least one of the following:
 - a. Improper sleep scheduling consisting of frequent daytime napping, selecting highly variable bedtime or rising times, or spending excessive amounts of time in bed
 - b. Routine use of products containing alcohol, nicotine, or caffeine, especially in the period preceding bedtime
 - c. Engagement in mentally stimulating, physically activating, or emotionally upsetting activities too close to bedtime
 - d. Frequent use of the bed for activities other than sleep (e.g., television watching, reading, studying, snacking, thinking, planning)
 - e. Failure to maintain a comfortable sleeping environment.
 - D. The sleep disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Insomnia due to a drug or substance

The essential feature of insomnia due to drug or substance abuse is a suppression or disruption of sleep caused by consumption of a prescription medication, recreational drug, caffeine, alcohol, or food item or by exposure to an environmental toxin.³

The ingestion or discontinuation of a drug or substance can be associated with insomnia. Examples include alcohol, caffeine, over-the-counter antihistamine cold and allergy medications, prescription medications for asthma or beta-blockers for heart conditions, as well as recreational drugs (Table 3.7).

Table 3.6 ICSD-2 diagnostic criteria for insomnia due to medical condition.

-
- A. The patient's symptoms meet the criteria for insomnia.
 - B. The sleep disturbance is present for at least 1 month.
 - C. The patient has a coexisting medical or physiological condition known to disrupt sleep.
 - D. Insomnia is clearly associated with the medical or physiological condition. The insomnia began near the time of onset or with significant progression of the medical or physiological condition and waxes and wanes with fluctuations in the severity of this condition.
 - E. The sleep disturbance is not better explained by another sleep disorder, medical or neurological disorder, or mental disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Table 3.7 ICSD-2 diagnostic criteria for insomnia due to drug or substance.

-
- A. The patient's symptoms meet the criteria for insomnia.
 - B. The sleep disturbance is present for at least 1 month.
 - C. One of the following applies:
 - a. There is current ongoing dependence on or abuse of a drug or substance known to have sleep-disruptive properties either during periods of use or intoxication or during periods of withdrawal
 - b. There is current ongoing use of or exposure to a medication, food, or toxin known to have sleep-disruptive properties in susceptible individuals.
 - D. The insomnia is temporarily associated with the substance exposure, use or abuse, or acute withdrawal.
 - E. The sleep disturbance is not better explained by another sleep disorder, medical or neurological disorder, or mental disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Sleep-related breathing disorders

The disorders in this subgroup are characterized by disordered respiration during sleep. Central sleep apnea syndromes include those in which respiratory effort is diminished or absent in an intermittent or cyclical fashion due to central nervous system or cardiac dysfunction. The obstructive sleep apnea syndromes include those in which there is an obstruction in the airway resulting in continued breathing effort but inadequate ventilation.³

There are five major categories of SRBD: central sleep apnea (CSA), obstructive sleep apnea (OSA), sleep-related hypoventilation/hypoxemia, sleep-related hypoventilation/hypoxemia due to medical condition, and other SRBD (unspecified). Only CSA and OSA will be discussed.

Central sleep apnea syndromes

Characteristic of CSA is the absence of airflow and ventilatory effort for at least 10 seconds during sleep. 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.^{18,19} There may also be an airway collapse, but this is not necessarily a diagnostic event.²⁰ 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.²¹

There are five types of CSA syndromes (Table 3.1), four of which are further defined here.

Table 3.8 *ICSD-2* diagnostic criteria for primary central sleep apnea.

-
- A. The patient reports at least one of the following:
 - a. Excessive daytime sleepiness
 - b. Frequent arousals and awakenings during sleep or insomnia complaints
 - c. Awakening short of breath.
 - B. Polysomnography shows five or more central apneas per hour of sleep.
 - C. The disorder is not better explained by another current sleep disorder, medical or neurological disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Primary central sleep apnea

Primary central sleep apnea is of unknown etiology and is characterized on the PSG by recurrent cessation of respiration during sleep with the apnea having no associated ventilator effort.³

The absence of airflow and respiratory effort is characteristic of primary CSA, and five or more of these types of apneic events per hour are needed to be demonstrated on the PSG. Clinically, the patient will typically present with symptoms of insomnia and EDS or fatigue, and sometimes difficulty with breathing while sleeping, such as awakening with choking (Table 3.8).

Cheyne–Stokes breathing pattern

Cheyne–Stokes breathing pattern is characterized by recurrent apneas, hypopneas, or both apneas and hypopneas alternating with prolonged hyperpneas during which tidal volume gradually waxes and wanes in a crescendo–decrescendo pattern.³

Characteristics for CSA due to Cheyne–Stokes breathing pattern are repeated apneas that alternate with prolonged episodes of hyperpnea, that is deep and rapid respiratory efforts.^{22,23} Although Cheyne–Stokes breathing (Figure 3.1) can occur during sleep or waking hours, this abnormal breathing pattern is an indication of a more advanced disorder, such as congestive heart failure, when it does occur during waking hours (Table 3.9).

High-altitude periodic breathing

High-altitude periodic breathing is characterized by cycling periods of apnea and hyperpnea with the apnea being associated with no ventilator effort (a central apnea).³

Characteristic for high-altitude periodic breathing is a sleep disorder caused by an acute mountain sickness.²⁴ Typically, this will occur when sleeping at altitudes higher than 15,000 feet, especially when not adapted to that altitude (Table 3.10).

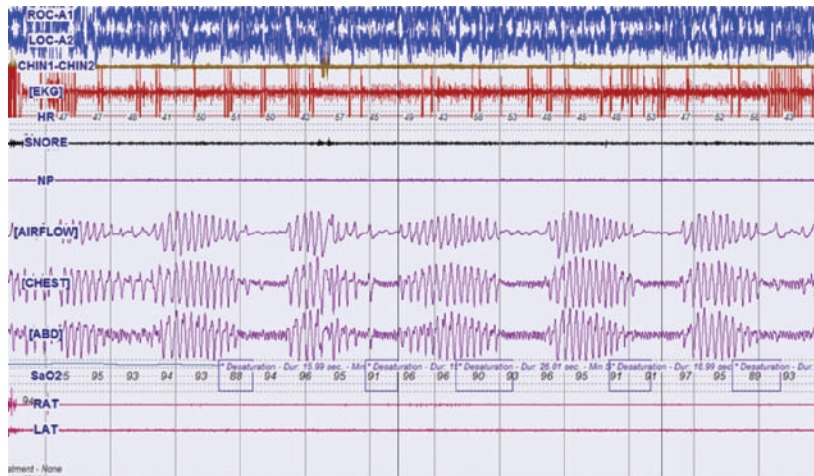


Figure 3.1 Cheyne–Stokes breathing pattern. Note characteristic crescendo and decrescendo pattern in breathing effort, paralleling the airflow. (Kakkar R and Hill G. Interpretation of the adult polysomnogram. *Otolaryngol Clin North Am.* 2007; 40(3):736. Reprinted with permission.)

Table 3.9 ICSD-2 diagnostic criteria for Cheyne–Stokes breathing pattern.

- A. Polysomnography shows at least ten central apneas and hypopneas per hour of sleep in which the hypopnea has a crescendo–decrescendo pattern of tidal volume accompanied by frequent arousals from sleep and derangement of sleep structure.

NOTE: Although symptoms are not mandatory to make this diagnosis, patients often report excessive daytime sleepiness, frequent arousals and awakenings during sleep, insomnia complaints, or awakening short of breath.

- B. The breathing disorder occurs in association with a serious medical illness, such as heart failure, stroke, or renal failure.
- C. The disorder is not better explained by another current sleep disorder, medication use, or substance use disorder.

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Table 3.10 ICSD-2 diagnostic criteria for high-altitude periodic breathing.

- A. Recent ascent to altitude of at least 4,000 meters.
- B. Polysomnography demonstrates recurrent central apneas, primarily during nonrapid eye movement (NREM) sleep at a frequency greater than 5 per hour. The cycle length should be 12 to 34 seconds.

NOTE: Because high-altitude periodic breathing is a normal adaptation to altitude, there are no specific criteria regarding the frequency of central apneas that should be considered normal or abnormal. Although no specific symptoms are required, recurrent awakening during the night and fatigue during the day may be present.

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Table 3.11 ICSD-2 diagnostic criteria for central sleep apnea due to drug or substance.

-
- A. The patient has been taking a long-acting opioid regularly for at least 2 months.
 - B. Polysomnography shows a central apnea index of 5 or more periodic breathing (ten or more central apneas and hypopneas per hour of sleep in which the hypopnea has a crescendo–decrecendo pattern of tidal volume, accompanied by frequent arousals from sleep and derangement of sleep structure).
 - C. The disorder is not better explained by another current sleep disorder or medical or neurological disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Central sleep apnea due to drug or substance

A small number of studies have documented that users of long-acting opioids may have central apneas during sleep.³

This type of CSA is caused by substance or drug use, usually the long-acting opioid pain medications, such as methadone and hydrocodone, with the former being the major contributor (Table 3.11).^{25,26}

Obstructive sleep apnea syndromes

Obstructive sleep apnea (OSA) is characterized by repetitive episodes of complete (apnea) or partial (hypopnea) upper airway obstruction occurring during sleep.³

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.^{27–29} Whereas apnea is complete cessation of airflow, hypopnea is characterized by a 70% reduction of airflow for ≥ 10 seconds or any reduction in airflow that is associated with either an arousal from sleep or a $\geq 3\%$ arterial oxygen desaturation.³⁰ Apneas and hypopneas as a result of these varying degrees and locations of upper airway obstructions are regarded as the most common SRBD.³¹

As measured by pulse oximetry, associated with these episodes are reduced blood oxygen saturation in spite of inspiratory efforts. Also associated is sleep fragmentation or disruption of the quality and duration of sleep along with the possible subjective reports of EDS and/or insomnia because of the repetitive arousals or awakenings that occur in an effort to

Table 3.12 ICSD-2 diagnostic criteria for obstructive sleep apnea, adult.

NOTE: A, B, and D or C and D satisfy the criteria.

- A. At least one of the following applies:
 - a. The patient complains of unintentional sleep episodes during wakefulness, daytime sleepiness, unrefreshing sleep, fatigue, or insomnia
 - b. The patient wakes with breath holding, gasping, or choking
 - c. The bed partner reports loud snoring, breathing interruptions, or both during the patient's sleep.
- B. Polysomnographic recording the following:
 - a. Five or more scoreable respiratory events (i.e., apneas, hypopneas, or respiratory effort-related arousals (RERAs)) per hour of sleep
 - b. Evidence of respiratory effort during all or a portion of each respiratory event (in the case of an RERA, this is best seen with the use of esophageal manometry).

OR

- C. Polysomnographic recording the following:
 - a. Fifteen or more scoreable respiratory events (i.e., apneas, hypopneas, or RERAs) per hour of sleep
 - b. Evidence of respiratory effort during all or a portion of each respiratory event (in the case of an RERA, this is best seen with the use of esophageal manometry).
- D. The disorder is not better explained by another current sleep disorder, medical or neurological disorder, medication use, or substance use disorder.

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

establish airway patency. In order for a diagnosis of OSA to be made, a sleep study must demonstrate a minimum of five or more apenic or hypopneic episodes per each hour of sleep or 30 episodes per 6 hours of sleep.³² Although obstruction of the upper airway may include multiple anatomical sites, closure of the upper airway is most commonly located in the retropalatal and retroglottal areas (Table 3.12).^{33–35}

Hypersomnias of central origin (not due to a circadian rhythm sleep disorder, SRBD, or other cause of disturbed nocturnal sleep)

This section includes 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.³

EDS is regarded as sleepiness that interferes with activities and quality of life during the waking hours. The individual is typically 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, or a sleep disorder.

The *ICSD-2* defines daytime sleepiness, or hypersomnia, as “the inability to stay awake and alert during the major waking episodes of the day, resulting in unintended lapses into drowsiness or sleep.”³ Specifically, hypersomnias of central origin are not caused by SRBD, circadian rhythm sleep disorder, or other causes of disturbed nocturnal sleep. The two more common ones are discussed here.

Narcolepsy with cataplexy

Narcolepsy with cataplexy is primarily characterized by excessive daytime sleepiness and cataplexy. Many of the symptoms of narcolepsy with cataplexy are due to an unusual proclivity to transition rapidly from wakefulness into rapid eye movement (REM) sleep and to experience dissociated REM sleep events (Table 3.13).³

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 (Table 3.14).³

The two most common types of narcolepsy are *narcolepsy with cataplexy* and *narcolepsy without cataplexy*.^{36,37} Cataplexy is regarded as a sudden but transient loss of muscle tone that is typically triggered by emotions. Some individuals who initially do not exhibit cataplexy will subsequently develop such episodes with their narcolepsy.³

The Multiple Sleep Latency Test (MSLT) is used to assess daytime sleepiness and diagnose narcolepsy.^{38,39}

Circadian rhythm sleep disorder

For optimal sleep, the desired sleep time should match the timing of the circadian rhythm of sleep and wake propensity. Therefore, a recurrent or 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-hour social and physical environments.³

Table 3.13 ICSD-2 diagnostic criteria for narcolepsy with cataplexy.

-
- A. The patient has a complaint of excessive daytime sleepiness occurring almost daily for at least 3 months.
 - B. A definite history of cataplexy, defined as sudden and transient episodes of loss of muscle tone triggered by emotions, is present.

NOTE: To be labeled as cataplexy, these episodes must be triggered by strong emotions—most reliably laughing or joking—and must be generally bilateral and brief (less than 2 minutes). Consciousness is preserved, at least at the beginning of the episode. Observed cataplexy with transient reversible loss of deep tendon reflexes is a very strong, but rare, diagnostic finding.

- C. The diagnosis of narcolepsy with cataplexy should, whenever possible, be confirmed by nocturnal polysomnography followed by a Multiple Sleep Latency Test (MSLT); the mean sleep latency on MSLT is less than or equal to 8 minutes and two or more sleep onset REM periods (SOREMPs) are observed following sufficient nocturnal sleep (minimum 6 hours) during the night prior to the test. Alternatively, hypocretin-1 levels in the cerebrospinal fluid (CSF) are less than or equal to 110 pg/mL or one-third of mean normal control values.

NOTE: The presence of one or more SOREMPs during the MSLT is a very specific finding, whereas a mean sleep latency of less than 8 minutes can be found in up to 30% of the normal population. Low CSF hypocretin-1 levels (less than or equal to 110 pg/mL or one-third of mean normal control values) are found in more than 90% of patients with narcolepsy with cataplexy and almost never in controls or in other patients with other pathologies.

- D. The hypersomnia is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Table 3.14 ICSD-2 diagnostic criteria for narcolepsy without cataplexy.

-
- A. The patient has a complaint of excessive daytime sleepiness occurring almost daily for at least 3 months.
 - B. Typical cataplexy is not present, although doubtful or atypical cataplexy-like episodes may be reported.
 - C. The diagnosis of narcolepsy without cataplexy must be confirmed by nocturnal polysomnography followed by an Multiple Sleep Latency Test (MSLT). In narcolepsy without cataplexy, the mean sleep latency on MSLT is less than or equal to 8 minutes and two or more SOREMPs are observed following sufficient nocturnal sleep (minimum 6 hours) during the night prior to the test.

NOTE: The presence of two or more SOREMPs during the MSLT is a very specific finding, whereas a mean sleep latency of less than 8 minutes can be found in up to 30% of the normal population.

- D. The hypersomnia is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Table 3.15 *ICSD-2* general criteria for circadian rhythm sleep disorders.

-
- A. There is a persistent or recurrent pattern of sleep disturbance due primarily to one of the following:
 - a. Alterations of the circadian timekeeping system
 - b. Misalignment between the endogenous circadian rhythm and exogenous factors that affect the timing or duration of sleep.
 - B. The circadian-related sleep disruption leads to insomnia, excessive daytime sleepiness, or both.
 - C. The sleep disturbance is associated with impairment of social, occupational, or other areas of functioning.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Circadian rhythm sleep disorders (CRSD) 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.⁴⁰ Another time indicator for the body is melatonin, which is low during the day since light suppresses the secretion of melatonin and increases as the body prepares for the onset of sleep.⁴¹

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.^{40,42} 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 (Table 3.15).³

There are nine recognized types of CRSD in the *ICSD-2*, but two of the more common are discussed here.

Jet lag type

Circadian rhythm sleep disorder, jet lag type, is a circadian rhythm sleep disorder in which there is a temporary mismatch between the timing of the sleep and wake cycle generated by the endogenous circadian clock and that of the sleep and wake pattern required by a change in time zone.³

Jet lag type (jet lag sleep disorder) occurs when there is a transient misalignment of the individual’s internal circadian sleep–wake cycle and the new time zone associated with the external environment. Typically, this type of CRSD worsens in relation to the number of crossed time zones.⁴³ Individuals with jet lag sleep disorder typically report a history of difficulty falling asleep at the desired time as well as EDS during the waking

Table 3.16 ICSD-2 diagnostic criteria for circadian rhythm disorder, jet lag type.

-
- A. There is a complaint of insomnia or excessive daytime sleepiness associated with transmeridian jet travel across at least two time zones.
 - B. There is associated impairment of daytime function, general malaise, or somatic symptoms such as gastrointestinal disturbance within 1 or 2 days after travel.
 - C. The sleep disturbance is not better explained by another current sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

hours. It is not uncommon to have disturbed sleep and impaired function during the waking hours associated with this disorder (Table 3.16).

Shift work type

Circadian rhythm sleep disorder, shift work type, is characterized by complaints of insomnia or excessive sleepiness that occur in relation to work hours that are scheduled during the usual sleep period.³

Shift work type (shift work sleep disorder) features insomnia or EDS that is associated with the scheduling of an individual's hours of work during the usual times for sleep for that individual. This type of time shift typically includes night shifts, early morning shifts, or rotating shifts. The adverse affect on sleep quality can result in EDS, insomnia, impaired function during the waking hours, and increased irritability.^{44,45} Specifically, these individuals will report a history of insomnia during their scheduled sleep time as well as EDS during their scheduled work time (Table 3.17).

Table 3.17 ICSD-2 diagnostic criteria for circadian rhythm disorder, shift work type.

-
- A. There is a complaint of insomnia or excessive daytime sleepiness associated with a recurring work schedule that overlaps the usual time for sleep.
 - B. The symptoms are associated with the shift work schedule over the course of at least 1 month.
 - C. Sleep log or actigraphy monitoring (with sleep diaries) for at least 7 days demonstrates disturbed circadian and sleep time misalignment.
 - D. The sleep disturbance is not better explained by another current sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Parasomnias

Parasomnias are undesirable physical events or experiences that occur during entry into sleep, within sleep, or during arousals from sleep.³

Parasomnias are undesirable and unintentional physical and/or subjective experiences that occur as the individual begins to enter into sleep, during sleep, or during arousals from sleep.^{3,46–49} 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 results in parasomnias.^{49,50}

Of the parasomnias, those considered to be disorders of arousal are the most common and can manifest in 4% of the adult population.⁵¹ Examples of such arousals can include sleepwalking, mumbling, shrieking, disorientation upon awakening, limb paralysis, and uncontrollable eating.⁵²

The more common of the parasomnias will be discussed here.

Confusional arousals

Confusional arousals consist of mental confusion or confusional behavior during or following arousals from sleep, typically from slow-wave sleep in the first part of the night, but also upon attempted awakening from sleep in the morning.³

Characteristic of confusional arousals is the display of confused or disoriented mental and behavior activities that can take place during and after awakening from sleep.³ Although this disorder is most common in younger children, it can occur with adults (Table 3.18).

Sleepwalking

Sleepwalking consists of a series of complex behaviors that are usually initiated during arousals from slow-wave sleep and culminate in walking around with an altered state of consciousness and impaired judgment.³

Table 3.18 ICSD-2 diagnostic criteria for confusional arousals.

- | | |
|----|--|
| A. | Recurrent mental confusion or confusional behavior occurs during an arousal or awakening from nocturnal sleep or a daytime nap. |
| B. | The disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder. |

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Table 3.19 ICSD-2 diagnostic criteria for sleepwalking.

-
- A. Ambulation occurs during sleep.
 - B. Persistence of sleep, an altered state of consciousness, or impaired judgment during ambulation is demonstrated by at least one of the following:
 - a. Difficulty in arousing the person
 - b. Mental confusion when awakened from an episode
 - c. Amnesia (complete or partial) for the episode
 - d. Routine behaviors that occur at inappropriate times
 - e. Inappropriate or nonsensical behaviors
 - f. Dangerous or potentially dangerous behaviors.
 - C. The disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Sleepwalking, also known as somnambulism, is characterized by walking behavior during an altered state of consciousness and impaired judgment.³ However, sleepwalking can also involve simply sitting up in one's bed. Inappropriate behavior, such as urinating in the middle of a room, can occur during sleepwalking, and even injuries can occur (e.g., falling down a set of stairs) which may cause trauma to the maxillo-mandibular region. Typically, these individuals do not remember sleepwalking occurrences (Table 3.19).

Sleep terrors

Sleep terrors consist of arousals from slow-wave sleep accompanied by a cry or piercing scream and autonomic nervous system and behavioral manifestations of intense fear.³

Sleep terrors are considered to be the most dramatic of the arousal disorders since it is common for the individual to exhibit screaming or crying along with extreme panic.³ There are usually autonomic system responses, such as tachycardia and increased muscle tone, along with intense fear. When these events occur, the individual typically sits up in bed and can even display significant motor activity that can include hitting walls and running around or even out of a bedroom,⁴⁹ again with the potential to cause physical injury such as trauma to the maxillo-mandibular region. The individual can be difficult to awaken, and upon awakening from an episode, the individual is usually confused and does not remember the episode (Table 3.20).

Recurrent isolated sleep paralysis

Recurrent isolated sleep paralysis is characterized by an inability to perform voluntary movements at sleep onset (hypnagogic or pre-dormital form) or on waking from sleep (hypnopompic or post-dormital form) in the absence of a diagnosis of narcolepsy.³

Table 3.20 ICSD-2 diagnostic criteria for sleep terrors.

-
- A. A sudden episode of terror occurs during sleep, usually initiated by a cry or loud scream that is accompanied by autonomic nervous system and behavioral manifestations of intense fear.
 - B. At least one of the following associated features is present:
 - a. Difficulty in arousing the person
 - b. Mental confusion when awakened from an episode
 - c. Amnesia (complete or partial) for the episode
 - d. Dangerous or potentially dangerous behaviors.
 - C. The disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Recurrent isolated sleep paralysis features an individual's inability to perform voluntary motor function of the limbs, trunk, and head at the time of onset of sleep or upon awakening. Typically, respiratory movements are not affected and cognition is not impaired. It is not uncommon for there to be associated hallucinations.

This type of disorder can have episodes with a duration of 1 to several minutes. Individuals have indicated such events to be extremely frightening (Table 3.21).

Nightmare disorder

Nightmare disorder is characterized by recurrent nightmares, which are disturbing mental experiences that generally occur during REM sleep and that often result in awakening.³

Nightmare disorder is characterized by dream patterns and themes that are increasingly frightening or distressing during the sleep period such that

Table 3.21 ICSD-2 diagnostic criteria for sleep paralysis.

-
- A. The patient complains of an inability to move the trunk and all limbs at sleep onset or on waking from sleep.
 - B. Each episode lasts seconds to a few minutes.
 - C. The disturbance is not better explained by another sleep disorder (particularly narcolepsy), a medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

NOTE: Hallucinatory experiences may be present but are not essential to the diagnosis. Polysomnography, if performed, reveals the event to occur at a dissociated state with elements of rapid eye movement (REM) sleep and wakefulness.

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Table 3.22 ICSD-2 diagnostic criteria for nightmare disorder.

-
- A. Recurrent episodes of awakenings from sleep with recall of intensely disturbing dream mentation, usually involving fear or anxiety, but also anger, sadness, disgust, and other dysphoric emotions.
 - B. Full alertness on awakening, with little confusion or disorientation; recall of sleep mentation is immediate and clear.
 - C. At least one of the following associated features is present:
 - a. Delayed return to sleep after the episodes
 - b. Occurrence of episodes in the latter half of the habitual sleep period.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

the individual can awaken with intense fear and anxiety. Usually this disorder is accompanied by an increase in heart and respiratory rates.⁵³ Individuals have indicated that it can be difficult to return to sleep after such an episode (Table 3.22).

Sleep-related eating disorder

Sleep related eating disorder (SRED) consists of recurrent episodes of involuntary eating and drinking during arousals from sleep with problematic consequences.³

Sleep-related eating disorder is characterized by repeated episodes of eating and/or drinking during arousals or partial arousals from sleep. This type of disorder may result in trauma to the maxillomandibular region, such as tongue biting or tooth fracture. Typically, this disorder involves an uncontrollable behavior of which the individual does not recall upon awakening (Table 3.23).

Sleep-related movement disorders

Sleep-related movement disorders are conditions that are primarily characterized by relatively simple, usually stereotyped, movements that disturb sleep or by other sleep-related monophasic movement disorders such as sleep-related leg cramps.³

Sleep-related movement disorders constitute characteristic body movements that cause sleep disturbance. The most common ones are discussed here.

Restless legs syndrome

Restless legs syndrome (RLS) 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 as a feeling that is simply difficult or impossible to describe.³

Table 3.23 ICSD-2 diagnostic criteria for sleep-related eating disorder.

-
- A. Recurrent episodes of involuntary eating and drinking occur during the main sleep period.
 - B. One or more of the following must be present with the recurrent episodes of involuntary eating and drinking:
 - a. Consumption of peculiar forms or combinations of food or inedible or toxic substances
 - b. Insomnia related to sleep disruption from repeated episodes of eating, with a complaint of nonrestorative sleep, daytime fatigue, or somnolence
 - c. Sleep-related injury
 - d. Dangerous behaviors performed while in pursuit of food or while cooking food
 - e. Morning anorexia
 - f. Adverse health consequences from recurring binge eating of high caloric foods.
 - C. The disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

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.⁵⁴ 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. The urge to move the legs and the paresthesias can be so unpleasant as to preclude the individual from initiating asleep.⁵⁵ It has also been reported that individuals can awaken from sleep because of the RLS episode.⁵⁶ Often the individual will relieve these sensations by getting up and walking.^{57,58} It is not uncommon to associate RLS in individuals who demonstrate reduced iron levels along with renal failure (Table 3.24).^{59,60,61}

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.³

Periodic limb movement disorder (PLMD) is characterized by repetitive limb movements that occur during sleep.⁶² PLMD can be associated with RLS, although PLMD can stand alone as episode independent of RLS. These periodic episodes of limb movement can result in sleep disturbances,³ although the individual is usually unaware of such partial

Table 3.24 ICSD-2 diagnostic criteria for restless legs syndrome for adults.

-
- A. The patient reports an urge to move the legs, usually accompanied or caused by uncomfortable and unpleasant sensations in the legs.
 - B. The urge to move or the unpleasant sensations begin or worsen during periods of rest or inactivity such as lying or sitting.
 - C. The urge to move or the unpleasant sensations are partially or totally relieved by movement, such as walking or stretching, at least as long as the activity continues.
 - D. The urge to move or the unpleasant sensations are worse, or only occur, in the evening or night.
 - E. The condition is not better explained by another current sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

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,⁶³ but it can also involve the upper limbs (Table 3.25).

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 the sleep period.³

Sleep-related leg cramps are characterized by sudden intense muscle contractions that occur during sleep. Typically, the muscles of the calves or 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 (Table 3.26).

Sleep-related bruxism

Sleep-related bruxism is an oral activity characterized by grinding or clenching of the teeth during sleep, usually associated with sleep arousals.³

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.^{64,65}

Table 3.25 *ICSD-2* diagnostic criteria for periodic limb movement disorder.

-
- A. Polysomnography demonstrates repetitive, highly stereotyped limb movements that are
 - a. 0.5–5 seconds in duration
 - b. Of amplitude greater than or equal to 25% of toe dorsiflexion during calibration
 - c. In a sequence of four or more movements
 - d. Separated by an interval of more than 5 seconds (from limb-movement onset to limb-movement onset) and less than 90 seconds (typically there is an interval of 20–40 seconds).
 - B. The PLMS index exceeds 5 per hour in children and 15 per hour in most adult cases.

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.

- C. There is clinical sleep disturbance or a complaint of daytime fatigue.

NOTE: If PLMS are present without clinical sleep disturbance, the PLMS can be noted as a polysomnographic finding, but criteria are not met for a diagnosis of PLMD.

- D. The PLMs are not better explained by another current sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder (e.g., PLMs at the termination of cyclically occurring apneas should not be counted as true PLMS or PLMD).
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

In dentistry, bruxism is regarded as a mandibular parafunctional activity, whereas in sleep medicine, SRB is considered to be a movement disorder. Because of this difference, SRB is included in the *ICSD-2* category of sleep-related movement disorders.

Table 3.26 *ICSD-2* diagnostic criteria for sleep-related leg cramps.

-
- A. A painful sensation in the leg or foot is associated with sudden muscle hardness or tightness indicating a strong muscle contraction.
 - B. The painful muscle contractions in the legs or feet occur during the sleep period, although they may arise from either wakefulness or sleep.
 - C. The pain is relieved by forceful stretching of the affected muscles, releasing the contraction.
 - D. The sleep-related leg cramps are not better explained by another current sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Table 3.27 ICSD-2 diagnostic criteria for sleep-related bruxism.

-
- A. The patient reports or is aware of tooth-grinding sounds or tooth clenching during sleep.
 - B. One or more of the following is present:
 - a. Abnormal wear of the teeth
 - b. Jaw muscle discomfort, fatigue, or pain and jaw lock upon awakening
 - c. Masseter muscle hypertrophy upon voluntary forceful clenching.
 - C. The jaw muscle activity is not better explained by another current sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

In addition to creating noise that may disturb the sleep of the bed partner, these involuntary mandibular movement episodes may cause arousals during the sleep period as well as abrasion wear of the teeth and restorations, changes of the periodontal tissues, hypersensitivity of the dentition, masticatory myalgia, and restriction of mandibular movement. Along with the studies demonstrating the relationship that emotional stress and psychosocial factors can have with SRBD,^{66,67} the microarousal effect of this activity on sleep architecture has been shown to be associated with the central and autonomic nervous system processes.⁶⁸ In addition, individuals with SRBD have been shown to have more frequent body movements when compared to individuals without SRBD (Table 3.27).⁶⁹

Isolated symptoms, apparently normal variants and unresolved issues

Sleep-related symptoms that either lie at the borderline between normal and abnormal sleep or that exist on the continuum of normal to abnormal events in sleep are included in this section.³

Snoring

Snoring is a respiratory sound generated in the upper airway during sleep that typically occurs during inspiration but may also occur in expiration; the snoring described here occurs without episodes of apnea or hypoventilation.³

The *Merriam-Webster Online Dictionary* defines snoring as “to breathe during sleep with a rough hoarse noise due to vibration of the soft palate.”⁷⁰ The resultant noise involves a vibration interaction of the airflow with the muscles and pharyngeal walls of the upper airway, particularly in the areas of the soft palate, uvula, and pharyngeal airway. Snoring is more often observed during inspiration, but it can also occur during expiration.

Snoring is very common with an occurrence in 24% of adult females and 40% of adult males.⁷¹ 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 consult. Not all individuals who snore have OSA, but those individuals with OSA generally demonstrate a snoring component during their sleep period.⁷² 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.^{2,73,74} Whereas individuals with OSA will report the classic descriptions of EDS or insomnia, individuals with primary or benign snoring will not have these experiences.^{73,75} 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 his or her primary care physician (Table 3.28).⁷⁶⁻⁸³

Other sleep disorders

Those sleep disorders that cannot be classified elsewhere in the *ICSD-2* are listed in this category. There are several reasons why a sleep disorder may be classified in this section: 1) the sleep disorder may overlap many other categories, 2) there may be insufficient data collected to firmly establish another diagnosis, 3) it seems likely that new sleep disorders will be discovered during the lifetime of the *ICSD-2*. Such disorders would be classified here until a permanent diagnosis is established in future editions.³

Environmental sleep disorder

Environmental sleep disorder is a sleep disturbance due to a disturbing environmental factor that causes a complaint of either insomnia or daytime fatigue and somnolence.³

Table 3.28 *ICSD-2* diagnostic criteria for snoring.

- A. Audible snoring noises are reported by an observer.
- B. The patient has no complaints of insomnia, excessive daytime sleepiness, or sleep disruption that are attributable to snoring or airflow limitation.

NOTE: Polysomnography is not required for the diagnosis of snoring, but, when performed, demonstrates audio signal peaks via microphone that are confirmed by the attendant technical staff to be snoring. These noises are not associated with airflow limitation, arousals from sleep, oxygen desaturations, or cardiac arrhythmias.

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Table 3.29 ICSD-2 diagnostic criteria for environmental sleep disorder.

-
- A. The patient complains of insomnia, daytime fatigue, or a parasomnia. In cases in which daytime fatigue is present, the daytime fatigue may occur as a result of the accompanying insomnia or as a result of poor quality of nocturnal sleep.
 - B. The complaint is temporarily associated with the introduction of a physically measurable stimulus or environmental circumstances that disturb sleep.
 - C. It is the physical properties, rather than the psychological meaning of the environmental factor, that account for the sleep complaint.
 - D. The sleep disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.
-

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Sleep can be disturbed to varying degrees by different environmental factors, including sound, light, temperature, bed partner's snoring and/or OSA, bed motion, and even the psychological factor of needing to maintain an alertness level that may be needed when caring for someone in the household such as an infant.

The prevalence of this disorder is not known, but the practitioner can query the patient about the more common environmental factors that may interfere with sleep. Sometimes it may be necessary to ask the patient to maintain a sleep diary so that patterns can be discerned. The bed partner can also be helpful in providing resourceful information.

An environmentally controlled sleep study may be necessary for a definitive evaluation. If the environmental issues can be resolved, a reduction or even elimination of the sleep disorder usually follows. Otherwise, there may be symptoms of long-term sleep disturbance, affecting the quality of life of the individual (Table 3.29).⁸⁴

Appendix A: Sleep disorders associated with conditions classifiable elsewhere

Numerous medical disorders affect sleep or are affected by sleep. This appendix is not designed to list them all. Rather, it lists a small number of medical disorders that may be of particular importance to sleep diagnosticians.³

Fibromyalgia

According to the 1990 American College of Rheumatology consensus criteria, fibromyalgia is characterized by widespread pain of at least 3 months' duration and muscle tenderness, as determined by palpation.³

One of the greatest challenges for the dentist is to evaluate and diagnose the orofacial pain complaint. A pain that has an etiology localized and related to the tissues of the oral cavity is generally easier to manage than when the pain does not have an obvious source such as a musculoskeletal pain, which can be more systemic with central nervous system involvement.

Fibromyalgia is a syndrome described by multiple tender point sites and long-standing musculoskeletal pain that is usually diffuse. The criteria for fibromyalgia 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.⁸⁵ The prevalence of fibromyalgia is the second most common rheumatological disorder after osteoarthritis.

Many individuals with the fibromyalgia syndrome also experience sleep disturbance that can result in feeling tired, unrefreshed sleep, reduced cognitive function, and early awakening from sleep.^{86–88} It has been found that most subjects with fibromyalgia experienced microarousals⁸⁷ and an electroencephalographic (EEG) alpha-delta or alpha-NREM anomaly that interrupts the deep Stage 4 NREM restorative level of sleep.^{89,90} This EEG anomaly, though, may not be specific to fibromyalgia as it has also been found in individuals who did not have fibromyalgia complaints.⁹¹

Despite these investigative differences with EEG activity and fibromyalgia, the field of sleep medicine does acknowledge the relationship and affect of fibromyalgia syndrome on sleep (Table 3.30).

Sleep-related headaches

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.³

Table 3.30 ICSD-2 diagnostic criteria for fibromyalgia.

- | |
|---|
| <p>A. A history of widespread pain is present for at least 3 months. The pain affects the left and right sides of the body, above and below the waist, and the axial skeletal regions (cervical, chest, and upper and lower back).</p> <p>B. Tenderness, as determined by digital palpation with approximately 4 kg of pressure, is present in at least 11 of 18 specific anatomic sites that are located in the following bilateral areas: subocciput, anterior lower cervical at intertransverse spaces at C5–C7, upper borders of mid-trapezius, supraspinatus, second costochondral junctions of the anterior ribcage, lateral epicondyles, gluteals, greater trochanters, and medial fat pad of the knees.</p> |
|---|

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

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.⁹² 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.⁹³

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.⁹² Relative to cluster headache, melatonin secretion may be impaired in those individuals.⁹⁴

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.⁹⁵⁻⁹⁸ Studies demonstrated a significant increase in SRBD with cluster headache subjects.⁹⁹

For the practitioner involved with orofacial pain populations, it is important to differentiate the sleep-related headache patients from those patients whose headaches may be of different origin. Even though neuroimaging may rule in or out any structural or infection etiology for headache, there are no pathognomonic PSG findings for each of the different types of headaches (Table 3.31).

While there are no empirically established algorithms to guide clinical practice, there are now at least a few empirically supported tenets. The review suggests: (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.⁹³

Sleep-related gastroesophageal reflux

Sleep-related gastroesophageal reflux is characterized by regurgitation of stomach contents into the esophagus during sleep.³

Table 3.31 ICSD-2 diagnostic criterion for sleep-related headaches.

A. The patient complains of headache during sleep or upon awakening from sleep.

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

Table 3.32 ICSD-2 diagnostic criteria for sleep-related gastroesophageal reflux.

Either A and B, or C.

- A. The patient complains of recurrent awakenings from sleep with shortness of breath or heartburn.
- B. At least one of the following:
 - a. A sour bitter taste in the mouth upon awakening from sleep
 - b. Sleep-related coughing or choking
 - c. Awakening from sleep with heartburn.
- C. Polysomnography and esophageal pH monitoring demonstrate gastroesophageal reflux during sleep with associated arousal.
- D. The disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

This sleep disorder may result in a pain that is usually located substernal, but it may also manifest in the area of the throat. Gastroesophageal reflux disease (GERD), also more commonly known by patients as heart burn or acid indigestion, may present the same symptoms as with this sleep disorder. The dentist needs to be aware of this sleep disorder because of the patient who may present with a symptom of throat pain (Table 3.32).

Sleep-related coronary artery ischemia

Sleep-related coronary artery ischemia is characterized by ischemia of the myocardium that occurs at night, presumably during sleep.³

Typically, this particular sleep disorder will present itself with the sensation of pressure or pain in the chest area. However, because the patient may present with a symptom of pain radiating to the mandibular area, the practitioner should be familiar with this particular sleep disorder (Table 3.33).

Table 3.33 ICSD-2 diagnostic criterion for coronary artery ischemia.

- A. Sleep-related electrocardiographic features of cardiac ischemia are present (ST segment elevation or ST segment depression).

NOTE: Identifiable sleep disorders such as OSA may be present and may be temporally associated with the ST segment depression.

Source: International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. American Academy of Sleep Medicine. 2006.

CONCLUSION

There are numerous sleep disorders that patients may have. For the practicing dentist, it is beneficial if one is knowledgeable of symptoms that may indicate the risk for a sleep disorder.

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

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4

Pediatrics and sleep-related breathing disorders

CONCEPTUAL OVERVIEW

Sleep disorders are not exclusive to the adult population. Children and adolescents also may suffer from sleep disorders. However, sleep disorders are not as easily recognized in this group as compared to adults and therefore are frequently undiagnosed. The sleep problems are often manifested as behavioral issues, emotional concerns, or personal conflicts that are out of proportion to the actual event, such as family conflicts or stress.

The more common sleep disorders in children and adolescents involve sleep-related breathing disorders (SRBD), difficulty initiating or maintaining sleep (i.e., insomnia), periodic limb movement disorder (PLMD), or restless legs syndrome (RLS). There are other sleep disorders that may impact this age group, but the majority of them fall within these categories. SRBD is one of the major interests for the dentist.

Awareness of SRBD in the pediatric population dates back to the 1800s. A medical manuscript in 1884 indicated that mouth breathing had ill effects on children as well as adults.¹

Another publication in 1889 noted that children presented with deafness appeared “backward and even stupid,” had headaches, and would mouth breathe.² Also, in 1892 a medical document linked sleep and daytime performance in children with sleep-related upper airway obstruction.³ The author stated that the “child is very stupid looking,” that an “influence on mental development is striking,” that the child found it “impossible to fix the attention for long at a time,” and that “the expression is dull, heavy, and apathetic.” Also recognized in that document were symptoms of headaches and listlessness. Others in the early half of the 1900s also found a

correlation between sleep, airway obstruction, and daytime functioning. The current scientific literature is abundant with evidence that confirms these earlier anecdotal findings.

An increasing awareness in the last 20 years regarding the importance of recognizing pediatric sleep issues is demonstrated by the increase in the number of articles and textbooks that are devoted to the topic. During that period, there has been a 1,226% increase in the number of published articles related to this area.⁴

FREQUENCY OF SLEEP DISORDERS IN THE PEDIATRIC AND ADOLESCENT POPULATION

Between 20 and 50% of the pediatric and adolescent population may have a sleep disorder.⁵⁻⁸ This frequency is significant and often results in parental concern because of the clinical impact on the child's behavioral, physical, and mental health. Unfortunately, sleep disorders in this age group are not recognized and are therefore undiagnosed.

Many times the presentation of insomnia is secondary to other situations that can impact sleep, and, as such, it may actually be the presenting symptom of SRBD. The presence of insomnia, oftentimes described as individuals being poor sleepers or having insufficient sleep, may be 12–33% in this younger population.^{7,9,10}

SRBD frequency in children, especially with regard to snoring or obstructive sleep apnea (OSA), has been studied the most. OSA is estimated to occur in 1–3% of children,¹¹ and snoring is believed to occur in 3–12% of this population, with occasional snoring being present about 20% of the time and habitual snoring being present about 10% of the time.¹²

More recently, the presence of RLS and PLMD in children has been investigated. Although the frequency is not specifically known, one study reported that about one-third of 138 adults who reported symptoms of RLS also indicated that these same symptoms were present before age 10.¹³

The key finding related to RLS in the younger age group is the association between RLS and inattentiveness, hyperactivity, and attention-deficit hyperactivity disorder (ADHD).¹⁴

Unlike adults, these sleep disorders may present themselves differently in this younger population. Accordingly, the sleep disorder may not be considered in conjunction with other complaints that a child may have or with signs that are evident, and thus may be overlooked. Interestingly, the utilization of health care by children with a sleep disorder is 2.6 times as compared to their matched controls.¹⁵

There are many health-related conditions that may be associated with a sleep disorder in a child or adolescent, but these findings may go undetected because sleep problems in this age group are oftentimes not a primary consideration (Table 4.1).

Table 4.1 Health-related conditions associated with sleep-related breathing disorders in children and adolescents.

Depression	Diabetes type 2	Allergy
Enuresis	Increased triglycerides	Headaches
Asthma	Tired/irritable ADD/ADHD	Obesity

In addition, there are congenital conditions that indicate that a child may be at a higher risk for a sleep disorder and specifically SRBD, especially OSA (Table 4.2).

Children that present for dental care may have clinical findings that would suggest that they are at risk for SRBD.

RECOGNITION OF SLEEP DISORDERS IN CHILDREN AND ADOLESCENTS

The recognition of a sleep disorder in children and adolescents requires the use of proper questioning techniques as well as the identification of related signs of these disorders. If a sleep disorder is suspected, then a simple questionnaire can be used to determine if the individual is at risk for a sleep disorder, but the questionnaire itself cannot be utilized as a diagnostic tool. On suspicion, a more formal referral to the child's physician or a sleep specialist is indicated.

A number of these questionnaires have been developed over the years. The Pediatric Daytime Sleepiness Scale is composed of eight questions that are most applicable to middle-school children, and it assesses the relationship between daytime sleepiness and school-related outcomes, mainly educational achievement.¹⁶ The Pediatric Sleep-Disordered Breathing OSA-18 questionnaire evaluates quality-of-life issues related to OSA, and it looks at the symptoms as well as sleep disturbances and daytime functioning.¹⁷ Another instrument, the Pediatric Sleep Questionnaire, has been designed as a generalized-type questionnaire that covers a wide range of issues related to sleep disruption. Table 4.3 represents a generalized pediatric sleep questionnaire.

Table 4.2 More common congenital conditions that predispose the child to sleep-related breathing disorders.

Down syndrome	Pierre Robin syndrome
Prader-Willi syndrome	Achondroplasia
Asperger syndrome	Chiari malformation

Source: Adapted from Sheldon SH, Ferber R, Kryger MH. Principles and Practice of Pediatric Sleep Medicine. Elsevier/Saunders. 2005; 23.

existence of sleep problems as well. The most common recommendation then would be to give instructions for good sleep practices, also known as sleep hygiene.

Sleep-related breathing disorders

SRBD consists mainly of snoring and OSA. In a child or even the adolescent patient, the presentation of snoring may appear more like heavy breathing. For an apnea or any cessation in breathing during sleep to be identified, a parent or someone else would need to observe this first hand. Therefore, the presence of apnea, like snoring, often goes undetected because most children sleep alone or in a separate room.

Another type of SRBD is termed upper airway resistance syndrome (UARS), although it is not officially recognized in the *International Classification of Sleep Disorders, Second Edition (ICSD-2)*. UARS may appear to be OSA; however, the key differences are that the respiratory events of UARS are related to an arousal and there are no distinct apneas or hypopneas.²⁰ In addition, the level of blood oxygen, also known as oxygen saturation, does not decrease with UARS.

The recognition of SRBD in a child can also be related to simply mouth breathing alone.²¹ This type of breathing pattern during sleep appears to have an almost identical affect on the quality of sleep, and thus may result in very similar symptoms as a definitive SRBD.

The presentation of symptoms or signs associated with SRBD in children can be recognized on the basis of either the daytime or nighttime findings (Table 4.4).

One study outlined the presentation of symptoms associated with SRBD that were divided into three age groups: preschool, preadolescent, and adolescent (Table 4.5).²²

Because SRBD has not been a typical or usual consideration when evaluating pediatric patients, the American Academy of Pediatrics (AAP)

Table 4.4 Symptoms and findings at nighttime and daytime in children with sleep-related breathing disorders.

Nighttime	Daytime
Snoring	Neurocognitive impairment
Bruxism	ADHD and ADD
Awakenings	Hyperactivity
Mouth breathing	Behavioral issues (irritable)
Nightmares	Tired/poor school performance

Table 4.5 Clinical manifestations of sleep-related breathing disorders in three age groups—up to the age of 18; based on the review of 189 charts.

Associated problem	Preschool (n = 41)	Preadolescent (n = 91)	Adolescent (n = 51)
Daytime fatigue	30%	50%	71.1%
Excessive daytime sleepiness (EDS)	38.7%	59.2%	80.4%
Sleep-onset insomnia	40%	21%	48.1%
Nocturnal sleep disruption	85.3%	69.5%	70.6%
Nightmare	12.5%	19.7%	21.3%
Sleep walking	9.4%	24%	12.8%
Enuresis	40.7%	31.9%	20.5%
Sleep bruxism	50%	49.3%	23.9%
ADHD	13.8%	29.4%	40.9%
Morning headaches	9.7%	12%	19.1%
Delayed sleep phase syndrome	0%	4.1%	30.6%
Mean AHI	16.4 (±16.8)	10.3 (±13.3)	16.2 (±22.9)
Mean RDI	16.6 (±15.7)	11.1 (±12.2)	16.3 (±21.8)

Source: Adapted from Kim J, Won C, Guilleminault C. The clinical manifestation of sleep-disordered breathing in children and adolescent. *Sleep*. 2007; 30(Abstract Supplement):86.

developed a guideline for the diagnosis and management of SRBD in this age group of patients.²³ The guidelines recommend that

- all children be screened for snoring;
- in the presence of a cardiorespiratory health condition, the child should have a more extensive evaluation;
- diagnostic evaluation be conducted to determine if the risk for OSA is present, most often resulting in an overnight sleep study.

This type of guideline can easily be implemented in the dental setting by anyone who treats pediatric patients.

Treatment for OSA in children is most often a tonsillectomy and adenoidectomy when those anatomical structures are enlarged because of their potential to be interfering with the airway. This is the first line of treatment and often the most successful. Following this surgical intervention, in many instances the symptoms are reversed and no further treatment is indicated.

CLINICAL FINDINGS

Clinically, at the time of an oral evaluation, there are some signs that a patient may be at risk for a sleep disorder and specifically SRBD. Prior to the clinical evaluation, the history is a critical component in the recognition process.



Figure 4.1 An example of an adenoidal face. (Meyer B and Marks MD. Stigmata of Respiratory Tract Allergies. Kalamazoo, MI: Upjohn Co. 1977. Used with permission from Pfizer Inc.)

There are facial features that indicate a risk for SRBD. The most common of these are the following:

- *Adenoidal faces*: This is a condition where the face is rounded with an often blank stare (Figure 4.1).
- *Allergic shiners*: These are the dark circles that are often found under the eyes. They are related to a reduction or absence in nasal breathing with an increased amount of mouth breathing (Figure 4.2).



Figure 4.2 An example of allergic shiners. (Meyer B and Marks MD. Stigmata of Respiratory Tract Allergies. Kalamazoo, MI: Upjohn Co. 1977. Used with permission from Pfizer Inc.)



Figure 4.3 An example of poor or inadequate (open) lip seal. (Meyer B and Marks MD. *Stigmata of Respiratory Tract Allergies*. Kalamazoo, MI: Upjohn Co. 1977. Used with permission from Pfizer Inc.)

- *Poor or inadequate lip seal*: In this situation, the lips are found to be apart with the appearance of difficulty in the ability to maintain a lip seal (Figure 4.3).
- *Small nares*: The opening to the nasal airway is small and appears constricted (Figure 4.4).
- *Nasal crease*: This is a horizontal line that goes across the nose above the tip of the nose. Oftentimes this may be associated with the allergic salute, which is a gesture associated with a repetitive action of wiping the nose due to the feeling of a constant drainage. This clinical feature may be associated with an allergy (Figure 4.5).



Figure 4.4 Small nares.



Figure 4.5 An example of nasal crease; note the arrow pointing to the line across the nose. (Meyer B and Marks MD. *Stigmata of Respiratory Tract Allergies*. Kalamazoo, MI: Upjohn Co. 1977. Used with permission from Pfizer Inc.)

On clinical evaluation of the intraoral structures, there are a number of findings that indicate the increased potential for being at risk for SRBD. The ones most commonly encountered are the following:

- Bruxism or worn teeth (Figure 4.6)
- Crossbite and/or high palate (Figure 4.7)
- Scalloped tongue (Figure 4.8)
- Swollen or enlarged uvula (Figure 4.9)
- Enlarged tonsils (Figure 4.10)
- Deep or collapsed bite (Figure 4.11)

Neck size may also be a factor, but this has not been as clearly defined as it has for adults.



(a)



(b)

Figure 4.6 Worn posterior teeth due to severe bruxism over many years as seen (a) on a study model and (b) clinically on the molars.



(a)



(b)

Figure 4.7 High palatal vault often accompanied by a posterior crossbite: (a) high or deep palate and (b) posterior crossbite oftentimes seen with a high palate.



Figure 4.8 Scalloping (crenations) along the lateral border of the tongue.



Figure 4.9 Swollen or enlarged uvula.



Figure 4.10 Swollen or enlarged tonsils.

SLEEP STUDIES FOR CHILDREN

Sleep studies for children are the most effective means by which a determination of OSA can be made. In this age group, the data values that relate to the adult population do not apply here. Although a standardized set of values do not exist, there are guidelines by the American Thoracic Society (ATS) regarding what degree of apnea or hypopnea should be present to make a diagnosis of OSA (Table 4.6).²⁴

The ATS guidelines outline the severity of sleep apnea by apnea index, and they also report that an apnea-hypopnea index (AHI) greater than 5

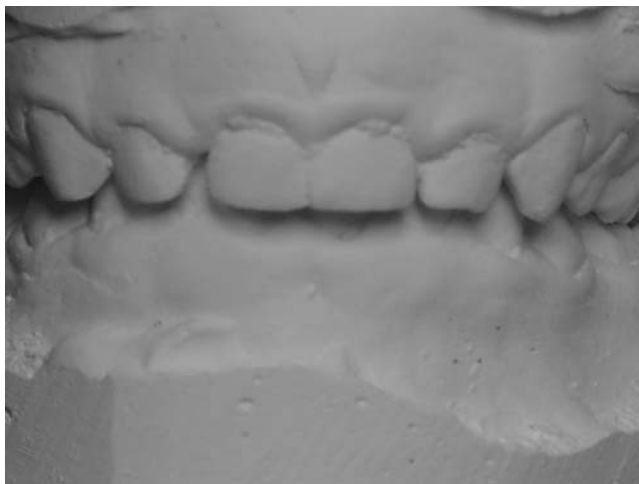


Figure 4.11 Deep or collapsed bite as seen in the primary dentition.

Table 4.6 Polysomnographic (sleep study) values for sleep apnea in the pediatric population.

Diagnosis	Apnea index (AI) events per hour	Oxygen saturation
Mild	1–4	86–91%
Moderate	5–10	76–85%
Severe	>10	≤75%

Source: Adapted from American Thoracic Society. Standards and indications for cardiopulmonary sleep studies in children. *Am J Respir Crit Care Med.* 1996; 153:866–878.

with a drop in the blood oxygen level below 92% also indicates that OSA is present. The most likely reason for these values to be lower than those for the adult population is that OSA is a progressive disease, and the symptoms that affect the child can worsen over time if they are not addressed.

CONSEQUENCES OF SLEEP DISRUPTION

Sleep disruption does not need to imply that the sleep is disrupted at some period between initiating sleep and waking up. It can also be related to an inadequate number of hours of sleep, and a reduction in total sleep time has the potential to lead to a variety of health-related consequences. One study found that among children that were 9–12 years of age who did not get 9 hours of sleep a night, the incidence for the onset of obesity was increased.²⁵ In a study of children who were 7 years of age, if they slept less than 9 hours per night, they were three times more likely to be overweight or obese when compared to similar children who slept more than 9 hours.²⁶

The reverse is also true. Children who are obese or overweight tend to be at a higher risk for SRBD, particularly OSA. However, sleep issues associated with obesity may be overlooked, and the focus may turn to diet and exercise in lieu of the number of hours of sleep or if snoring is present.

A recent study resulted in an AAP policy statement regarding obese children and the need to screen for elevated lipid levels.²⁷ Outlined in the study was the associated risk that exists for diabetes type 2, cardiovascular disease, and hypertension. In addition, the policy statement emphasized the need for dietary considerations as well as the need for exercise.

Growth can be impacted by poor sleep quality, especially by the reduction or absence in nonrapid eye movement sleep and particularly deep sleep or delta sleep, also known as restorative sleep.²⁸ This impaired sleep quality may lead to a decreased level of growth hormone that can be associated with SRBD, and it can also be attributed to an increase in the size of the tonsils and adenoids. The compromised airway from the enlarged

tonsils and adenoids may lead to sleep disruption directly associated with breathing disorders during sleep.

In addition, the enlarged tonsils can impact the ability of the child to eat and swallow and thus result in a decreased appetite. It is often found that children with airway problems are deficient in their growth as compared to their matched controls. One study found that growth failure was at least twice the expected norm in children who presented for surgical removal of the tonsils and adenoids or who had sleep symptoms.²⁹

When children, and particularly adolescents, have difficulty initiating sleep, also known as sleep onset insomnia, the potential for having an increased risk for sleep disruption and related symptoms increases. Most of the time, this situation is associated with a sleep disorder common to adolescents known as delayed sleep phase syndrome,³⁰ which is a condition classified as a circadian rhythm disorder. In this situation, the individual attempts to go to bed and to sleep at a set specific time; however, because of a natural alteration in their internal clock, they are not truly ready to initiate sleep. The subsequent outcome is that they lie awake in bed despite the attempts to fall asleep. Consequently, they fall asleep a few hours later than was desired, which can cause difficulty in waking up at a set time the next morning. This may result in mood swings, irritability, symptoms similar to attention deficit disorder (ADD) and attention-deficit hyperactivity disorder (ADHD), depression, daytime hypersomnolence or excessive daytime sleepiness (EDS), and, where applicable, an increased risk for a motor vehicle accident.

Sleep disruption and the lack of sufficient sleep may also lead to inattention that can result in problems with learning and cognition.³¹ This may be observed as behavioral or poor school performance, and it may result in an increased risk for accidents or injury.

OTHER SLEEP DISORDERS OF INTEREST

Insomnia

Insomnia is classically defined as the inability to initiate sleep or maintain sleep. Symptoms or signs that a child may be at risk for insomnia include depression, anxiety, mood changes, painful conditions, and headaches. Insomnia may actually be diagnosed as one of these other medical conditions as opposed to the actual sleep disorder that is involved. Oftentimes these conditions are recognized as a consequence of medication(s) that has been prescribed as opposed to the actual condition for which the medication is indicated. In addition, there may be times when insomnia is thought to be simply a “normal” consequence of the individual’s age, especially in the teenage years.

If a child or adolescent has difficulty with their sleep and this appears to be related to insomnia, then attention should be directed toward the sleep

Table 4.7 Recommended hours of sleep for children and adolescents.

Age range	Ideal hours of sleep
Infants	
0–3 months	15–17
3–18 months	13–15
Toddlers	
1–3 years	12–14
3–5 years	11–13
5–12 years	10–11
Adolescents (early teens)	8.5–9.25
Teens (mainly high school)	8.5–9.5

Source: Adapted from National Sleep Foundation. Available at www.nsf.org. 2009.

disorder. When a child simply gets a reduced number of hours of sleep, then his or her general health can suffer. The lack of adequate sleep in terms of the number of hours for children and adolescents needs to be addressed by the parents. However, for the parents to facilitate this, they must know what the proper number of hours of sleep should be for the appropriate age group. Studies have looked at this situation, and, in general, the number of hours of sleep varies with age, and it is believed to be more than that required for an adult (Table 4.7).

A National Sleep Foundation's research report demonstrated that adolescents clearly do not get an adequate number of hours of sleep as recommended.³² Only 20% of teenagers get the necessary 9 hours of sleep, and they get fewer and fewer hours as they progress from sixth grade to the senior year in high school. In addition, parents are not aware of the number of hours of sleep that teenagers are actually getting. In a study from the 2006 Sleep in America poll, parents felt that adolescents were getting adequate sleep 90% of the time while 44% of those same adolescents felt that they were getting adequate sleep.³³

Restless legs syndrome and periodic limb movement disorder

The presentation of RLS and PLMD need to be evaluated or considered. The best way to determine if this is present is by history and by recognition of clinical findings that may be present. Oftentimes the recognition of the risk for RLS and PLMD is based on the possible outcome as opposed to the specific symptoms.

RLS is most often present later in the day or at the time when the individual first attempts to initiate sleep. The most commonly occurring symptoms are the inability to sit or lay still, frequent movement of the legs, and a feeling of an achy or crawling sensation in the legs.³⁴ Many times the

presentation of these symptoms is passed off as leg pain or may be referred to as growing pains.

In conjunction with RLS, patients may have SRBD. The symptoms from the breathing disorder are the first ones to be recognized and may include the feeling of being tired, inattentiveness or ADD/ADHD-type symptoms, irritability or mood swings, and even poor school performance. The initiating factor in the presentation of these symptoms is the sleep disruption that leads to arousals during sleep. This fragmented sleep pattern results in a reduction of the quality of sleep and hence the symptoms begin to appear.

Clinically, it is possible that the presentation of evidence that suggests a tooth grinding or bruxism habit may also be related. Sleep bruxism and its relationship to RLS in adults may be present in 9–11% of the adult population.³⁵ Even though this has not been well defined in the pediatric population, the general relationship may apply.

PLMD most often occurs during sleep, and it is a condition where the legs and possibly the arms move or jerk involuntarily during sleep. The recognition of this is most often dependent on a bed partner. It is possible that the individual themselves might feel that their sleep is restless, thus indicating its presence.

Both RLS and PLMD originate in the central nervous system and are associated with the dopamine centers in the brain. Because of their close association with sleep bruxism, the occurrence of these two conditions may also be precipitated by some foods, beverages, or medications. Most commonly recognized are cigarettes, caffeine, amphetamines, and mainly selective serotonin reuptake inhibitor antidepressants. In addition, a lowered ferritin level is a possibility as a precipitating factor.

Narcolepsy

Narcolepsy presents as brief attacks of uncontrollable sleepiness or EDS. Oftentimes this sleep disorder may go unrecognized and may even be thought to be symptoms associated with SRBD. The age of onset is typically between 15 and 30 years, and the disorder may not be diagnosed until the third or fourth decade of life. There may be a family history of others with it as well.

The presence of narcolepsy is 3–16 per 10,000, and it usually presents in the second decade of life.³⁶ One of the classic symptoms is cataplexy, also known as a sudden muscle weakness, particularly of the legs and trunk, and it occurs in association with some emotional stimulus such as laughter, fear, or rage.

Many times pharmaceutical stimulants similar to those used for managing ADD or ADHD can be used to treat the narcolepsy. In addition, there are other more specific medications that can be used to treat this particular sleep disorder and they should be prescribed by a physician, such as a sleep specialist, who is well-versed in their use and who will provide adequate follow-up care.

TREATMENT STRATEGIES

The treatment of pediatric sleep disorders, especially with the management of an SRBD, is frequently a multidisciplinary approach involving the pediatrician or family physician, an otolaryngologist, the general dentist, a pediatric dentist, an orthodontist, and at times a speech therapist. Complete evaluation and diagnosis of sleep disorders need to be referred to the appropriate medical specialist, and oftentimes the role of the dentist is relegated to the recognition and initial education of the parents and the patient.

The actual treatment often may not take place as early in the developmental process as it should because of the lack of initial identification of the problem along with the need for one of the practitioners to coordinate the care that is needed. In every case, the first step in this process is the recognition of a sleep disorder, especially SRBD. Any dentist who sees children is well-positioned to recognize a developing situation and propose the necessary treatment.

First step is recognition

The recognition of a SRBD involves the detection of a variety of signs that the possible disorder exists. Many times these are subtle in nature, but, more often than not, they involve the growth and development of the child. The potential for early recognition of SRBD and the appropriate treatment of the upper airway, once there is a recognized problem, may be a means of preventing the development of OSA as an adult.²¹

Because SRBD is the condition with which the dentist will be involved, the recognition of related symptoms as well as clinical findings is essential. In addition, various pediatric questionnaires should be utilized whenever necessary along with an ongoing review of the patient's health history.

Subsequently, the development of a treatment plan as well as making the necessary referrals are in order.

Treatment options

The treatment specifically as it relates to SRBD should be initiated as soon as a problem is recognized, which typically involves a number of other health care providers that include a variety of medical specialties.

Tonsillectomy and adenoidectomy

The surgical removal of the tonsils and adenoids has and continues to be the first line of treatment. This intervention has been recognized as the primary treatment for children who snore and may be at risk for OSA.^{23,37} One study found that the success rate for this procedure, as measured by sleep study results, demonstrated that the AHI was improved by

95% and that the normalization of the sleep study occurred 82.9% of the time.³⁸

The removal of the tonsils and adenoids also has an impact on other factors related to SRBD. It has been shown that behavioral problems associated with SRBD were improved after this procedure.³⁹ Another study demonstrated that behavioral issues such as hyperactivity, ADHD, and EDS were improved 1 year after the surgery was performed.⁴⁰ It is interesting to note that children with ADHD were three times as likely to snore, a common finding associated with enlarged tonsils and adenoids, when compared to non-ADHD children.⁴¹

Snoring by itself should not be overlooked. Even though behavioral symptoms may not be present, the recognition of snoring has been found to be a strong risk factor for the presentation of hyperactivity over a 4-year period.⁴² This finding is significant since the recognition of ADHD has increased significantly between 1970 and 2000 during which time the reported prevalence has gone from 1.7% to a range of 5–10%.⁴³

The indications for tonsillectomy and adenoidectomy have been documented.⁴⁴ These include hyperplasia of these structures, OSA, failure to thrive, and abnormal growth of the craniofacial structures. Additional considerations identified include suspicion of other pathology, dysphagia, speech changes, and perhaps even halitosis.

Dental and orthodontic treatment

Dental and orthodontic treatment is intended to enhance growth and development as well as to improve the airway. There may be instances, though, when this will only prove beneficial after the tonsils and adenoids have been removed.

One of the primary means by which the airway may be improved is with palatal expansion. Studies have demonstrated that palatal expansion improved snoring and daytime hypersomnolence or EDS.^{45,46} Another study looked at children who had a mean AHI of 12.2. After 4 months, all the children had an AHI that was less than 1.0 and had an improvement in their nasal airway. The mean cross-section expansion was 4.32 mm and a variation of less than 1 mm. A more current investigation looked at adenoidectomy and palatal expansion in combination.⁴⁷ These two procedures were found to be mutually beneficial in the resolution of the symptoms as well as the OSA. Also of importance in this study is the recognition of the team treatment from various disciplines. The article also points out the need for an orthodontic treatment plan to focus on the airway and not just the dental and occlusal conditions. If the patient has an already existing compromised airway, then the use of techniques such as extraction and headgear should be avoided because of their impact on anteroposterior development. Instead, orthodontics should be considered as a treatment directed toward improvement of the airway on a long-term basis. Furthermore, it is

indicated that when a pediatric patient has OSA or is at risk, then a facial analysis may be warranted.

In order for an awareness of the significance of airway compromise to be recognized, questions related to the airway must be performed on a regular basis. One study reviewed the impact of snoring and sleep disturbance in the orthodontic setting.⁴⁸ It found that 17% of the patients were habitual snorers, and the patients who were mouth breathers were three times as likely to snore. In addition, if they slept with their heads tipped back, they were more likely to have headaches and were more likely to have frequent colds and cough.

Other considerations

In addition to the aforementioned treatment considerations, it may be necessary to involve other health care providers. These other providers may include speech therapy and more importantly myofunctional tongue therapy to address the tongue posture, tongue thrusting, any speech-related issues, and the swallowing pattern. Physical therapy may also be necessary to address postural considerations as well as cervical dysfunction. In both these situations, the patient would benefit the most from a home exercise program that is designed to reinforce and maintain the intended outcomes of these therapies.

Another association that should be considered is the nasal airway and allergy. It is important for the patient to be able to adequately breathe through the nose, especially during sleep, because of the affects that allergy may have on the nasal structures. Lack of adequate nasal breathing leads to habitual mouth breathing, and this may progress to snoring and OSA. Addressing the nasal airway has been shown to improve the sleep in patients with OSA.⁴⁹ If the nasal airway is compromised, then allergy, structural conditions, and even asthma and reflux need to be considered. This would best be done by a referral to an allergist, otolaryngologist, or the primary care physician.

CONCLUSION

Sleep disorders and particularly SRBD are not uncommon to the pediatric population. Many times the presentation of the patient is with symptoms that do not directly indicate that a sleep disorder may be present, and subsequently the sleep disorder is unrecognized. Because dentists are involved with the head, neck, and oral cavity, there may be signs that a patient is at risk, but the subtle signs or symptoms go undetected.

The dentist who treats children needs to include a clinical examination and questioning for signs or symptoms that may indicate a sleep disorder and thus make the necessary referral to the physician. This can only be of benefit to the child in the long run and it may improve their quality of life.

The dentist who is involved with any type of treatment that involves the airway or growth and development, particularly orthodontics, needs to be especially vigilant of the airway and the implications of the proposed treatment on the airway. The possibility of preventing the development of OSA as the child reaches adulthood is intriguing.

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5

Medical and dental conditions related to sleep-related breathing disorders

CONCEPTUAL OVERVIEW

Research during the past decade has confirmed that sleep disorders adversely affect various physiological systems, including cardiovascular, metabolic, psychobiologic, endocrine, nervous, and immune.

Almost from the onset of the recognition of sleep-related breathing disorders (SRBD) in patients, there has been an awareness that this particular sleep disorder could increase the risk for health-related consequences. Although cardiovascular disease has been found to be one of the more significant consequences of SRBD, the risk for other health problems has also been demonstrated.

The most widely recognized and often referred to study regarding the health-related risks with SRBD is the Sleep Heart Health Study.¹ Additional studies have also been published that support the association between SRBD and the risk for many common medical conditions.

HEALTH CONSEQUENCES ASSOCIATED WITH SRBD

An increasing prevalence of metabolic syndrome is linked with SRBD. Metabolic syndrome is the term for a group of risk factors that are associated with specific health issues, including but not limited to cardiovascular disease, diabetes, and stroke. This syndrome, which is also known as Syndrome X, insulin resistance, and obesity syndrome, may be associated with SRBD. In one study, the presence of metabolic syndrome was found to be almost 40% greater in SRBD subjects than in control subjects.² There

is ongoing research and discussion in the medical field relative to further clarifying this syndrome.

The criterion by which metabolic syndrome is diagnosed is predicated on an individual having a minimum of three of the five following metabolic risk factors:³ (1) increased girth of the waist or having a gut with an increased body mass index (BMI), (2) increased blood levels of triglycerides, (3) decreased blood levels of high-density lipoprotein (HDL) cholesterol, (4) increased blood pressure, and (5) impaired fasting glucose.

The prevalence of diabetes and obesity in the United States is increasing, and the link of these risk factors to metabolic syndrome lends to the estimation of almost 50 million adults with this syndrome.⁴ In addition, obesity is associated with other health problems such as increased insulin resistance, increased levels of cholesterol and triglycerides, and a variety of other health consequences, primarily related to cardiovascular disease.

The most widely acknowledged association with SRBD and even just sleep loss or restriction relates to the presence of obesity,⁵⁻⁷ although SRBD does not have to be present for obesity to occur. Sleep restriction or a decrease in adequate hours of sleep may also lead to the obesity.^{8,9} The sleep loss and obesity association is not exclusive to adults. Obesity in children and adolescents is an increasing concern and appears to also be related to a lack of adequate number of hours of sleep.^{10,11}

Not all patients with SRBD are obese. Even the nonobese are at risk for similar health problems, mostly associated with short sleep duration.¹² An increase in abdominal girth or central (visceral) size is the one common finding that most often indicates SRBD as increasing the risk for health-related consequences.⁹

Hypertension

Since the early 1990s, studies have recognized that an individual with SRBD is at an increased risk for elevated blood pressure or hypertension. One study looked at the incidence of hypertension in obstructive sleep apnea (OSA) patients independent of obesity,¹³ and it demonstrated the following: (1) the incidence of elevated blood pressure existed in 20–70% of OSA patients, (2) significant OSA was found in 30–40% of hypertensive patients, and (3) nocturnal SRBD was more closely related to elevated blood pressure.

Arousals from sleep are also a factor in the elevation of blood pressure.¹⁴ These repeated arousals are associated with an increase in blood pressure during the day, and they appear to be related to an increase in sympathetic activity or tone that occurred during sleep and resulted in a rise in daytime blood pressure.¹⁵ The Sleep Heart Health Study further confirmed an association between SRBD and hypertension.¹

Snoring alone can also lead to an elevation in blood pressure. A study demonstrated that snoring without any significant apnea or hypopnea can

result in hypertension, but the risk is lower.¹⁶ Another study showed that SRBD and the risk for hypertension were greater in younger males than in males over the age of 60.¹⁷ Given the fact that snoring over time may lead to OSA, this should not be taken lightly.

Cardiovascular disease

The development of cardiovascular disease takes on a variety of related conditions, all of which have been found to be prevalent in the presence of SRBD. Studies may vary on the degree of risk, but it is generally agreed upon that SRBD is a significant factor in the presentation of cardiovascular disease.

Arrhythmias

Various arrhythmias have been documented during episodes of OSA, and they are resolved with continuous positive airway pressure therapy.¹⁸ In addition, bradycardia and tachycardia have been implicated as associated with OSA. Hypoxia's main outcome is bradycardia. Arrhythmia appears to be associated with oxygen desaturation, is common in patients with coronary heart disease, and appears to resolve with the management of the apnea.

Atrial fibrillation

There is an association between sleep apnea and the presence of atrial fibrillation.¹⁹ The odds ratio for the association of atrial fibrillation with OSA was found to be 2.19. All the patients studied were similar, especially as it related to the presence of diabetes, hypertension, and congestive heart failure (CHF).

Atherosclerosis, endothelial dysfunction, and coronary heart disease

OSA and the presence of coronary artery disease is significant. A study found that in a population of patients who had symptomatic angina diagnosed with angiography, 30.5% had OSA.²⁰ This group had a higher apnea-hypopnea index and more of a tendency to be overweight or obese.

The potential for atherosclerosis is also greater among OSA patients.²¹ Although these patients may be free of signs and symptoms of cardiovascular disease, atherosclerosis and coronary heart disease were found to be significantly higher as the degree of OSA worsened. The significance of this is critical if one considers that only 12.5% of a control group had signs of coronary heart disease while mild OSA patients demonstrated a 42% incidence and moderate to severe patients had an 80% incidence of disease in

Table 5.1 Risks for coronary heart disease relative to hours of sleep.

Risk factor	Hours of sleep
5	1.5–2
6	1–1.5
7–8	1
9+	1.5 or more

Source: Adapted from Ayas NT. The adverse health effects of sleep restriction. *Sleep Rev.* 2003; (May/June):16.

multiple vessels.²² Sleeping less than 7 hours a night or more than 8 hours may increase the risk for coronary heart disease (Table 5.1).

Endothelial dysfunction can also be associated with OSA. There can be subsequent relaxation in the vascular structures as well as atherosclerotic changes and cardiovascular disease, and the endothelial injury at a tissue level leads to atherogenesis.²³ The progression of atherosclerosis may be related to apnea events that alter inflammatory mediators and metabolic factors, which can result in hypertension.²⁴ These related events lead to atherosclerosis and cardiovascular disease.

A recent study has demonstrated that snoring alone may put patients at risk for atherosclerosis of the carotid artery.²⁵ The proposed mechanism involves vibrations in the pharyngeal airway that are in close proximity to the artery, and these vibrations may cause endothelial damage that results in inflammation and thereby promotes changes that may lead to atherosclerosis. The prevalence of the atherosclerosis worsened in direct proportion to the degree of snoring, and the prevalence ranged from 20% in mild snorers to 64% in heavy snorers.

Congestive heart failure

The association of cardiovascular disease and sleep apnea is well known. CHF has been found to be prevalent in patients mainly with central sleep apnea (CSA).²⁶ The recurring respiratory events are associated with sleep disruption, arousals, hypoxia, and hypercapnia. It appears that a high percentage of patients with heart failure when screened for SRBD tested positive.²⁷ In addition, there is oftentimes a relation between Cheyne–Stokes respiration and CHF that is present in CSA patients.

Interestingly, the use of an oral appliance (OA) can beneficially impact CHF.^{28,29} In these studies, the focus in determining the effect of OAs was based on the measurement of brain natriuretic peptide (BNP), which has been found to be elevated in OSA. This increase is linked to left ventricular pressure and volume levels. In patients with CHF, the BNP levels rise and are associated with sudden death, and it may predict morbidity and mortality.

Cerebrovascular function and stroke

Stroke is the third leading cause of death and long-term disability.³⁰ The incidence of cerebrovascular disease and stroke is related to SRBD, and it is independent of other known risk factors for stroke.³¹ The risk for stroke associated with OSA is also independent of hypertension, but the existence of hypertension, especially diurnal hypertension, further increases the risk. The mechanism is related to a decrease in cerebral perfusion and increased coagulation. There is an increased incidence of sudden death during sleep.³² In addition, after a stroke, there is an increase in the prevalence of OSA, which affects the ability of the patient to rehabilitate and recover following the stroke.

Triglycerides and cholesterol

HDL, or the good cholesterol, functions as both an antioxidant and antiatherogenic. HDL dysfunction has been found to be present in OSA patients.³³ This association is related to oxidative stress, which is present in patients with coronary heart disease. Lower levels of HDL have also been linked to issues with short-term memory deficits. Difficulty with memory has also been linked to OSA as one of the common symptoms.

Diabetes type 2

2 diabetes is linked to SRBD such as snoring and OSA. In fact, snoring alone has been shown to be an independent risk factor for this type of diabetes.³⁴ The impact of snoring and OSA, by virtue of upper airway obstruction, may lead to oxygen desaturation that in turn may cause a rise in the level of cortisol and catecholamines. The outcome of this cascade may be an increase in insulin resistance, which is considered to be a precursor to diabetes. The Sleep Heart Health Study demonstrated that hypoxia during sleep resulted in glucose intolerance which was independent of age, sex, BMI, or the size of the waist.¹ Other studies have also shown that SRBD, irrespective of obesity, may serve as a contributing factor to diabetes through its association with glucose and insulin metabolism.^{35–37}

Sleep disruption has also been shown to be common among those with type 2 diabetes.^{38,39} Three main factors were found that are related to sleep disruption: obesity, pain, and an increase in the need to use the bathroom during the night. One-third of those who have type 2 diabetes experience sleep problems. In addition, the severity of the diabetes is directly related to the sleep disruption.

Sleep restrictions and excessive sleep are other issues that may impact the risk for diabetes (Table 5.2).⁴⁰ Getting between 7 and 8 hours of sleep a night is optimum. Less than 6–7 hours and over 8–9 hours may lead to an increased risk for diabetes.^{1,41} After adjusting for BMI, where the tendency

Table 5.2 Risks for symptomatic diabetes relative to hours of sleep.

Risk factor	Hours of sleep
5	1.5–2
6	1–1.5
7–8	1 or <
9+	1.5 or more

Source: Adapted from Ayas NT. The adverse health effects of sleep restriction. *Sleep Rev.* 2003; (May/June):16.

for being overweight or obese was corrected, the increased risk for diabetes became modest (Table 5.2).

Gastroesophageal reflux disease

Gastroesophageal reflux disease (GERD) (Table 5.3) is estimated in the United States to be experienced daily by 7% of the adult population and weekly by 70%.⁴² Until more recently, though, there has been a slow acknowledgement of sleep-related GERD as well as its impact on daily function.⁴³

Many people who have SRBD also may complain or may have been diagnosed with GERD.⁴⁴ The prevalence of GERD is much higher in patients with OSA as compared to the general population.⁴⁵ This is a condition that is associated with the relaxation of the phreno-esophageal sphincter where the esophagus enters the stomach, and it is oftentimes associated with obesity and diabetes.

GERD may be more prevalent in SRBD because of the following:

1. Negative pressure in the airway is associated with narrowing of the airway during inspiration, which also impacts the esophagus. This negative inspiratory (intrathoracic) pressure generated during apnea is not selective to the airway, and thus the esophagus is also affected.⁴⁶

Table 5.3 Common symptoms of gastroesophageal reflux disease.

Nocturnal awakenings	Noncardiac chest pain
Laryngopharyngitis	Bronchial asthma
Chronic bronchitis	Pulmonary aspiration
Chronic cough	Acidic damage to teeth

Source: Adapted from Demeter P and Pap A. The relationship between gastroesophageal reflux disease and obstructive sleep apnea. *J Gastroenterol.* 2004; 39(9):815–820.

During expiration, the negative pressure is released, and the potential for reflux to occur is greater.

2. Lying in the prone position where the effects of gravity are negated increases the risk for reflux. For this reason, many people with GERD sleep in an elevated position.

In like manner, GERD, particularly when present at night along with OSA, was found to be a cause of sleep disturbance.⁴⁷ The presence of GERD-type symptoms is also associated with awakenings from sleep. It has been estimated that the symptoms of GERD can cause awakenings in 58.6% of these patients.⁴⁸ Other symptoms associated with these awakenings are difficulty in initiating sleep and experiencing nightmares. It was also demonstrated that patients with GERD had more symptoms of excessive daytime sleepiness (EDS) as well.⁴⁹

Asthma

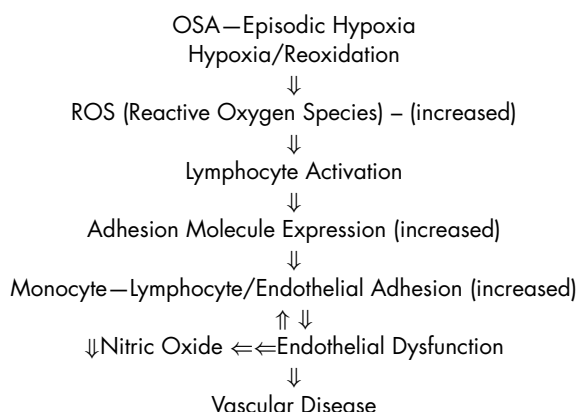
The presence of asthma, along with other respiratory conditions, may be associated with SRBD. In the general population, the prevalence of asthma is 5%. The coexistence of asthma and OSA is not well documented, although from a clinical perspective, many people who have SRBD also have asthma.

The one significant association that exists between asthma and SRBD is between asthma and GERD, the common denominator being the presence of inflammation. This same inflammatory condition has also been implicated in the progression of OSA. Nocturnal GERD associated with OSA can lead to the presentation of asthma.⁵⁰ The presence of airway obstruction and obesity are also common findings. The role of reflux in the precipitation of asthma has been linked to an elevation in BMI. As the BMI increases and obesity is more prevalent, the incidence of asthma also increases.

Alzheimer's disease

The link between OSA and Alzheimer's disease is becoming more apparent. The common finding at this time is the presence of a genotypic marker known as apolipoprotein (APOE4), which is a risk factor for Alzheimer's and cardiovascular disease.⁵¹ For those who carry this marker, there appears to be some complex interaction with brain pathology, cardiovascular disease, and OSA. One study demonstrated the relationship that exists between this genotype and OSA.⁵²

OSA and the association of hypoxia in the elderly may be linked to mental deterioration that is associated with dementia.⁵³ In the future, more research will be needed to confirm this association, but the proper management of SRBD may be a significant factor. It is also plausible that the proper management of hypertension, elevated cholesterol, or diabetes, all three of which are three commonly occurring conditions in SRBD, can impact brain function.⁵⁴

Table 5.4 Oxidative stress: pathway to cardiovascular disease and atherosclerosis.

Source: Adapted from Lavie L. Obstructive sleep apnoea syndrome—oxidative stress disorder. *Sleep Med Rev.* 2003; 7(1):35–51.

Oxidative stress

Until the early part of the 2000s, the association of elevated blood pressure, cardiovascular disease, and other health issues as a consequence of SRBD, particularly snoring and OSA, were thought to be related to the presence of respiratory events during sleep. The significance of hypoxia associated with the drop in oxygen saturation was also a consideration as was the impact on the sympathetic nervous system.

The hypoxia that may occur during SRBD can lead to an increase in the prevalence of oxidative stress that in turn will lead to an increase in free radical production. This complicated group of events is directly linked to the presentation of cardiovascular disease, hypertension, and endothelial cell damage. There is an associated increase in what has been termed reactive oxygen species that is related to inflammation, erythropoietin (a protein that promotes red blood cell production), and adhesion molecules.⁵⁵ All these findings are associated with the presentation of the aforementioned cardiovascular consequences. In addition, there is a diminished level of nitric oxide, which is an agent that causes vascular relaxation or dilation and is itself a free radical. Levels of nitric oxide are impacted by hypoxia, particularly when the hypoxia is chronic.

A simplified flow diagram shows the impact of OSA and how hypoxia can lead to endothelial dysfunction and contribute to the presentation of cardiovascular disease. What can be seen is the presentation of inflammatory conditions that are implicated in both OSA and cardiovascular disease (Table 5.4).

In addition, the presentation of atherosclerosis may be an outcome of the modification of LDL (the bad cholesterol that contributes to the clogging of

arteries) by oxidation that results in injury to endothelial cells as well as the underlying smooth muscle cells.

One animal study found that residual sleepiness, which may be an issue with some OSA patients despite adequate management of the apnea, may be related to long-term intermittent hypoxia associated with oxidative stress that results in neuronal injury of wake-promoting areas of the brain.⁵⁶ It is not clear as to what extent this gradual development may progress and to what degree the injury occurs. Additional study is needed to resolve this issue. However, the underlying thought may be that the sooner the SRBD is recognized and treated, the better off the outcome may be. To date, the treatment utilizes medication to manage the residual sleepiness.

PAIN AND SLEEP

Pain and painful conditions can be worsened by the lack of sleep or by sleep disorders. In like manner, pain can lead to poor quality of sleep, a loss of sleep, and a reduction in an adequate number of hours of sleep that only continues to perpetuate the pain cycle. Thus, the improvement in sleep can in and of itself lead to pain reduction. This realization, despite the recognition of the relationship of these two conditions, has not had significant attention until recently.⁵⁷

Studies have demonstrated that chronic pain can be present in 11–29% of the adult population,⁵⁸ and that 50–90% of these individuals can indicate that their sleep is adversely affected by their pain.^{59–62}

There are a number of painful conditions that the dentist will encounter. As such, it is imperative that the loss or 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.

SLEEP BRUXISM

Many times the management of bruxism is predicated on tooth wear or co-existing conditions such as headache, myofascial pain (MFP), or temporomandibular disorders (TMD). It is important for the dentist to understand that bruxism is a sleep disorder as described in the *ICSD-2* classification of sleep disorders as well as in other resources.^{63,64}

Sleep bruxism, also referred to as rhythmic masticatory muscle activity (RMMA), has been determined to be part of a sleep arousal response that is associated with the central nervous system and specifically the dopamine system.⁶⁵ Other factors have been considered such as psychological factors, stress, and personality traits, although they are now believed to play only a

Table 5.5 Comparison of awake and sleep time bruxism.

Awake time bruxers	Sleep time bruxers
Tooth clenching	Tooth grinding
Tooth tapping	Jaw muscle contractions
	Phasic (rhythmic)
Jaw bracing (no tooth contact)	Tonic (sustained)

Source: Adapted from Roehrs JD. Sleep talking and noisy grinding. *J Clin Sleep Med.* 2006; 2(4):477–478.

minor role. In addition, other factors such as occlusion and other anatomic structures of the orofacial region that have in the past been considered to be factors in bruxism are now viewed as playing only a limited role.⁶⁶

Sleep bruxism predominately occurs during nonrapid eye movement (NREM) stage 2 and rapid eye movement (REM) sleep.⁶⁷ There is also a relationship involving autonomic nervous system activity that precipitates tachycardia in association with the bruxing event. One study reported (1) that 4 seconds prior to the bruxing event, there was an increase in electroencephalographic (EEG) activity; (2) that one heartbeat prior to masticatory muscle depressor activity, there was a tachycardia; and (3) that within 800 milliseconds, there was masticatory muscle elevator activity.⁶⁸ All these reported events appear to be related to microarousals.

Another study demonstrated that with sleep bruxism, RMMA is present in about 60% of patients when asleep.⁶⁹ This is a slow movement of the orofacial structures that can become extreme in nature and lead to sleep bruxism. In addition, the resulting sleep bruxism may be more prevalent when the patient sleeps on their back.

Sleep bruxism was originally classified as a parafunctional activity that occurred during sleep. In the revised *ICSD-2*, sleep bruxism is viewed as a movement disorder and its classification is within the same category as restless legs syndrome (RLS) and periodic limb movement disorder (PLMD). Many times during a sleep study, the presence of RLS or PLMD activities is also found in sleep bruxers. Snoring and OSA have an increased risk in patients with sleep bruxism. It has been determined that the odds ratio for snoring, OSA, and EDS is 1.8, 1.4, and 1.3, respectively.⁷⁰

Sleep bruxism may be classified as either a comparison of awake time versus sleep time or as primary versus secondary (Tables 5.5, 5.6, and 5.7).

OROFACIAL PAIN

Of particular interest to the dentist are those patients that present with an orofacial pain condition. As with pain in general, these conditions are frequently associated with some type of sleep disruption. Similar to other

Table 5.6 Differentiation of primary and secondary bruxism.

Primary (idiopathic)	Secondary (iatrogenic)
No known medical or dental causes	Associated with a medical or psychiatric condition
Exacerbated by psychosocial factors at times	Iatrogenic: following drug intake or withdrawal

Source: Adapted from Kato T, Thie NMR, Montplaisir JY, et al. Bruxism and orofacial movements during sleep. In: Attanasio R and Bailey DR, eds. Sleep Disorders: Dentistry's Role. Dental Clinics of North America, 45. 2001; 657–684.

types of painful conditions, it may be difficult to determine which came first, the pain or the sleep problem.

Regardless of the type of orofacial pain complaint, addressing the sleep issues is most often helpful in managing the pain problem.

Table 5.7 Classification of secondary (iatrogenic) bruxism.

Movement disorders
Oromandibular dyskinesia
Oral tardive dyskinesia
Huntington's disease
Neurologic or psychiatric disorders
Dementia, depression, mental retardation
Coma
Cerebellar hemorrhage
Sleep-related disorders
RLS and PLMD
Apnea/snoring
REM sleep behavior disorder
Chemical substances
Alcohol
Nicotine (smoking)
Caffeine
Cocaine
Medications
Amphetamines
Antipsychotic drugs
Antidepressant drugs (especially selective serotonin reuptake inhibitors)
Calcium blockers
Antiarrhythmic drugs

Source: Adapted from Lavigne, GJ, Manzani C, and Kato T. Sleep bruxism. In: Kryger MH, Roth T, and Dement WC, eds. P and Practice of Sleep Medicine. 2005; 946–959.

Myofascial pain

Muscle pain is a common finding among patients with poor sleep. However, not all patients with poor sleep will have muscle pain or muscle tenderness when palpated. Those with preexisting muscle pain are more likely to have insomnia as opposed to SRBD. Sleep disturbance is a common finding in MFP patients.⁷¹ However, patients who snore or have OSA may also have muscular pain.

In some instances, muscle pain will not be restricted to just the orofacial structures, and it will encompass the entire body. These patients may be diagnosed with fibromyalgia (FM), a condition that is closely linked to sleep disruption as well as poor sleep quality and nonrestorative sleep.⁷² One study demonstrated that greater than 50% of subjects diagnosed with FM also experienced chronic fatigue.⁷³ 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 is important to recognize.

Temporomandibular disorders

Many times the presence of TMD is associated with muscle pain and/or sleep bruxism. The occurrence of TMD in conjunction with poor sleep may be associated with MFP and/or bruxism that occurs during sleep and/or waking hours.

In 2001 a National Heart, Lung, and Blood Institute workshop reported that patients with TMD were also at risk for cardiovascular and sleep-related consequences.⁷⁴ The findings of this workshop concluded that patients with TMD were similarly at risk for and predisposed to cardiovascular disease, including related conditions such as hypertension, heart failure, and stroke. The study also concluded that additional research is needed to actually understand the relationship that appears to exist amongst these conditions. However, there is current evidence to suggest support of these findings. Of interest is the evidence that suggests this relationship may be related to interactions in the central nervous system. These interactions may impact the presentation of TMD as well as alterations in sleep architecture.

Trigeminal 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.⁷⁵

Glossopharyngeal neuralgia

Glossopharyngeal neuralgia is similar to trigeminal neuralgia in that the attacks are paroxysmal, unilateral, and can be sharp to electric-like. They typically involve the ear, throat, tongue, or tonsillar area. Also the pain is oftentimes caused by function or touch. The difference from trigeminal neuralgia, though, is that the pain may awaken the patient from sleep.

Temporal arteritis

Also known as giant cell arteritis, this is a pain that often feels like a headache with throbbing around the area of the temporal artery.⁷⁶ The patient may have pain in the masticatory muscles associated with chewing. The pain will frequently be worse at night and may be exaggerated by resting the head on a pillow.⁷⁷ Because of the serious nature of this type of pain, immediate attention to the treatment should be initiated once it is diagnosed.

Atypical (idiopathic) toothache or atypical odontalgia

This type of orofacial pain 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.

Relative to sleep, 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.

SLEEP LOSS AND PAIN

Of interest is the finding that sleep loss, specifically 4 hours and REM-type sleep, is associated with hyperalgesia the following day.⁷⁸ 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.

One study demonstrated that sleep is analgesic in nature.⁷⁹ In patients with osteoarthritis, improvement of sleep latency and sleep efficiency was analgesic when compared to control subjects.

HEADACHE DISORDERS

Headache and sleep disorders are the most prevalent conditions seen in clinical practice. 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.⁸⁰

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.⁸¹

In cluster headache, the presence of OSA is 8.4 times that of the normal population.⁸² When the patient is over 40 years of age and has an increased 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 (Table 5.8). Of the patients who had headache, 53% were diagnosed with OSA.

CONCLUSION

Sleep disorders may contribute to the presence of a large number of chronic medical disorders.⁸³ The resolution and management of the medical condition, pain, headache complaint, and orofacial pain problems may best be achieved with the comanagement of a sleep disorder when it is present. Therefore, uncovering the presence of a sleep disorder or simply improving the patient's sleep may be significant in the resolution of the original concern.

Being an astute clinician now translates into looking for any contributing factors that may impact the situation as well as the resolution of that condition. All too often, the sleep issue is not addressed, and this makes it more difficult, more frustrating for the patient and clinician alike, and less cost effective when the ultimate goal is to efficiently and effectively manage the patient as a whole person.

Once a sleep disorder is uncovered, the next step is the appropriate treatment with the clinician who is most capable of managing the patient.

Table 5.8 Types of headaches—relationship to sleep.

Type of headache	Relevant findings
Migraine headache	May occur at night with stages 3 and 4 NREM sleep Occurs in 54% of narcoleptics May be provoked by sleep Common to sleep for relief (related to serotonin) Responds best to proper sleep hygiene
Cluster headache	Occurs predominately at night between 9 and 10 p.m. Linked to REM sleep Linked to sleeping late
Chronic paroxysmal hemicrania (CPH)	May be greater at night May wake the patient Considered variant of cluster Linked to REM sleep Responds to indomethacin (indocin)
Tension-type headache (headaches on awakening)	Becomes less with increased activity—disappears when awake and active Successful treatment of sleep apnea decreases the headaches Occurs with increased bruxism, alcohol, sinus inflammation
Hypnic headache	Prevalent in older people REM sleep-related disorder Appears with dreams Diffuse location Duration of 1–2 hours No migraine symptoms Responds to aspirin Prophylactic therapy: lithium, caeffine, indocin

Source: Adapted from Kryger MH, Roth T, and Dement WC, eds. *Principles and Practice of Sleep Medicine*. Philadelphia: Elsevier/Saunders. 2005; 879–881.

However, there are times when simply making the patient aware of the co-existence of a sleep problem lends a great deal to the patient's ability to self-manage their sleep and thus improve their quality of life.

In instances where the painful condition is associated with SRBD and OA therapy is determined to be a treatment option, the appropriate selection of an OA that will also address the airway may be the best choice.

The resolution of the sleep problem, particularly OSA, may have a major impact on the overall pain as well.

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Section 2

Assessment of the Sleep-Related Breathing Disorder Patient

Sleep assessment/studies



CONCEPTUAL OVERVIEW

The *International Classification of Sleep Disorders, Second Edition (ICSD-2)* has classified 96 sleep disorders.¹ When a patient presents with chief concerns that are potentially related to sleep disorders, it may be necessary to augment the history-taking and clinical examination with subjective and/or objective measurement instruments in order to fully assess and diagnose the patient. These same measurement instruments have been beneficial when conducting research of sleep disorders.

Prior to when actual physiological information could be assessed via instrumentation, behavioral-based criteria were employed for descriptions of sleep.² The advent of laboratory-based polysomnographic studies allowed for the inclusion of measurable data regarding the physiological changes occurring during wake/sleep stages and disorders.³

The purpose of this chapter is to allow for the reader to understand the availability and application of different subjective-based self-assessment and objective-based assessments for sleep disorders.

SUBJECTIVE SELF-ASSESSMENT

Relying on a patient to subjectively report his or her sleepiness without the use of a standardized set of questions is inconsistent and highly variable, especially with the significant potential to either underestimate or overestimate their sleepiness. At times it can be better to obtain a more consistent report from an individual who has the opportunity to observe that patient

during sleep. However, this latter method is also flawed with its inherent inconsistencies since the patient's bed partner is generally not awake during the patient's entire sleep period to provide observable history that is consistent and accurate.

In an effort to quantify self-assessment of sleepiness, question-based instruments to subjectively estimate one's own sleepiness are composed of a standardized set of statements or questions. 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

Of the available subjective-based self-assessments, the most frequently used instrument is the Epworth Sleepiness Scale (ESS), which was developed in 1991. The ESS has been demonstrated to identify degrees of sleepiness,⁴ and the results are considered to be within acceptable limits for test-retest reliability.⁵ The eight questions of the ESS query the individual about his or her subjective reporting of sleepiness relative to their expectation of dozing in eight different situations (Table 6.1).

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.^{6,7} While higher scores correlate with sleep disorders, it has also been shown that scores also improve relative to the efficacy of management of sleep-related breathing disorders (SRBD).⁸

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 pharmaceuticals. Thus, the ESS is best when it is employed to be an adjunct to the patient history and clinical examination.

Stanford Sleepiness Scale

Although not employed as commonly as the ESS, another self-assessment instrument is the Stanford Sleepiness Scale (SSS), which was developed in 1972.⁹

Through a questionnaire that utilizes a 7-point scale, the SSS provides a subjective measurement that relates to the individual's perception of sleepiness at a specific time (Table 6.2). It is regarded as a momentary evaluation and is therefore useful in assessing the degrees of perceived sleepiness throughout the period of a day. This ability to repeatedly be administered and reflect the current state of sleepiness is an advantage of the SSS over the ESS.

Table 6.1 Epworth Sleepiness Scale.

How likely are you to doze off or fall asleep in the following situations, in contrast to just feeling tired? This refers to your usual way of life in recent times. Even if you have not done some of these things recently, try to work out how they would have affected you. Use the following scale to choose *the most appropriate number* for each situation:

- 0 = would never doze
- 1 = slight chance of dozing
- 2 = moderate chance of dozing
- 3 = high chance of dozing

Situation	Chance of dozing
Sitting and reading	_____
Watching television	_____
Sitting, inactive in a public place (e.g., theatre or meeting)	_____
As a passenger in a car for an hour without a break	_____
Lying down to rest in the afternoon when circumstances permit	_____
Sitting and talking to someone	_____
Sitting quietly after lunch without alcohol	_____
In a car, while stopped for a few minutes in traffic	_____
	TOTAL _____

Source: Adapted from Johns MW. A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. *Sleep*. 1991; 14:540–545.

As with the ESS, the inherent brevity of the SSS adds to the simplicity of administering it, although it is best when used to complement the patient history and clinical examination.

OBJECTIVE-BASED ASSESSMENT

To definitively diagnose a sleep disorder, a complete collection of physiologic data from the wake/sleep stages needs to be obtained and interpreted. 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 (Table 6.3).^{10,11} A level I study is regarded as the most accepted study for the assessment and treatment of obstructive sleep apnea (OSA). The four levels are differentiated per the number of simultaneously recorded physiological signals as well as whether or not the sleep study was attended by a sleep technologist.

Table 6.2 Stanford Sleepiness Scale.

Circle one number which best describes your level of alertness or sleepiness right now.

Scale	Characteristics
1	Feeling active and vital; wide awake
2	Functioning at a high level, but not at peak; able to concentrate
3	Relaxed; awake; not a full alertness; responsive
4	A little foggy, not at peak; let down
5	Fogginess; beginning to lose interest in remaining awake; slowed down
6	Sleepiness; prefer to be lying down; fighting sleep; woozy
7	Almost in reverie; sleep onset soon; lost struggle to remain awake
8	Asleep

Source: Adapted from Hoddes E, Zarcone V, Smythe H, et al. Quantification of sleepiness: a new approach. *Psychophysiology*. 1973; 10:431–436.

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.¹² The

Table 6.3 Levels of sleep studies.

Type I	A standard attended in-laboratory polysomnography (PSG) with a minimum of seven parameters measured (electroencephalogram—EEG; electrooculogram—EOG; chin electromyogram—EMG; electrocardiogram—ECG; airflow; respiratory effort; oxygen saturation).
Type II	A comprehensive portable PSG that usually measures the same parameters as Type I, but it can be unattended.
Type III	Also referred to as a cardiorespiratory sleep study. A modified portable sleep apnea testing with a minimum of four parameters measured (heart rate or ECG, oxygen saturation, and at least two channels of respiratory movement or respiratory movement and airflow) and it is unattended.
Type IV	A continuous or dual bioparameter recording with a minimum of one parameter measured (oxygen saturation, flow, or chest movement) and it is unattended.

Source: Adapted from Kushida CA, Littner MR, Morgenthaler TM, et al. Practice parameters for the indications for polysomnography and related procedures: an update for 2005. *Sleep*. 2005; 28(4):499–519.

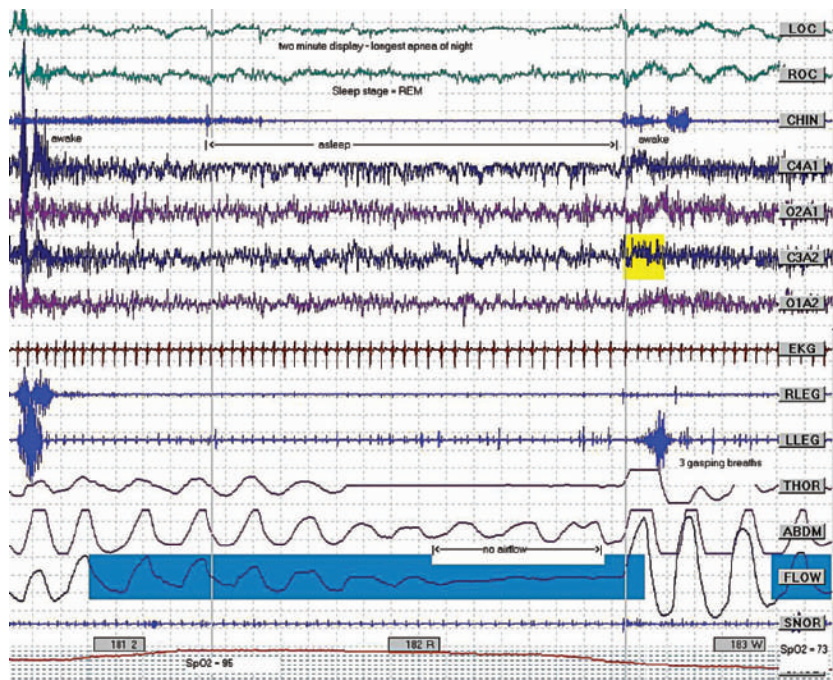


Figure 6.1 Attended polysomnograph demonstrating sleep apnea.

study is usually conducted in a sleep laboratory/center type of facility with trained staff to ensure proper placement of the necessary sensors as well as recognizing and addressing any displacement of sensors as needed during the study.¹³

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 apneic events (Figure 6.1), hypopneic events (Figure 6.2), bruxism (Figure 6.3), snoring (Figure 6.4), and Cheyne–Stokes breathing (Figure 6.5).

A summary of the entire PSG data can be reflected in a graphic form called a hypnogram (Figure 6.6), 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,¹⁴ 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,¹⁵ including SRBD.¹⁶ Effectiveness of treatment methods can also be achieved with a PSG.

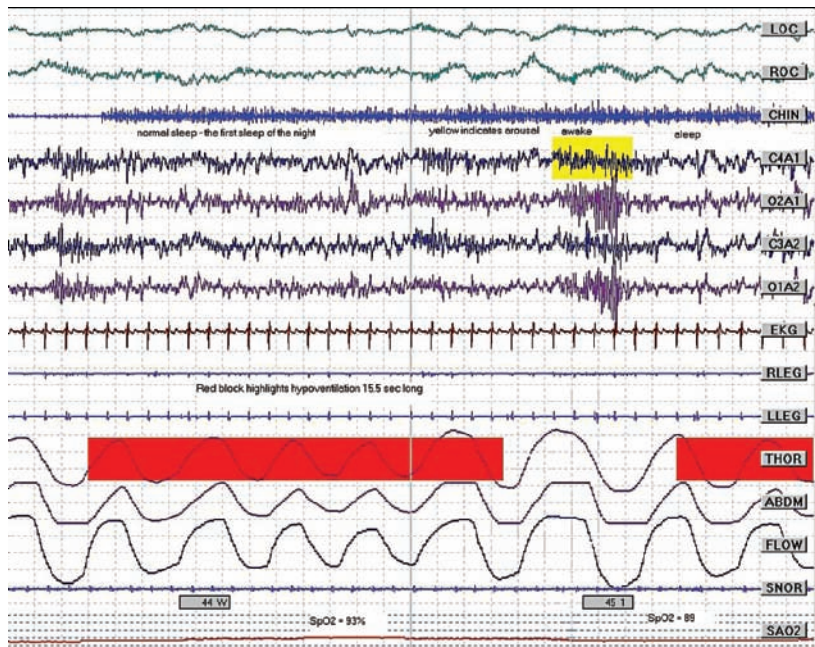


Figure 6.2 Attended polysomnograph demonstrating hypopnea.

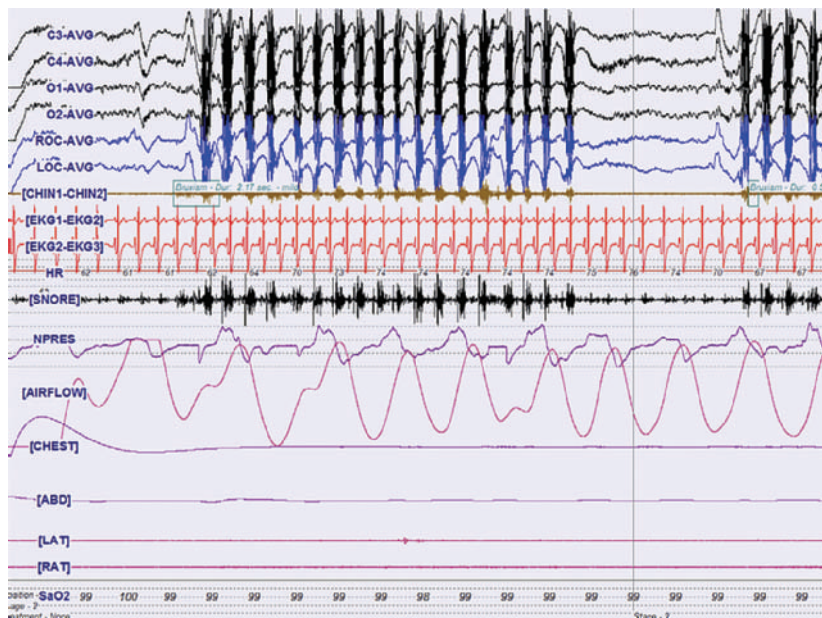


Figure 6.3 Bruxism in 29-year-old female. Prominent grinding sounds were heard on video replay. (Kakkar R and Hill G. Interpretation of the adult polysomnogram. *Otolaryngol Clin North Am.* 2007; 40(3):732. Reprinted with permission.)

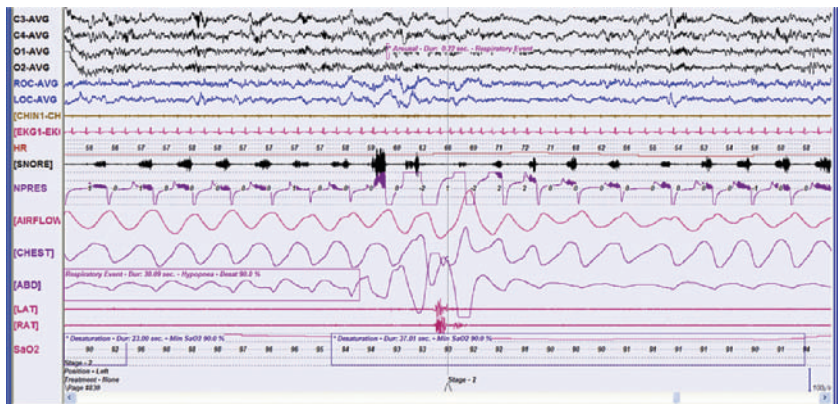


Figure 6.4 Snoring during a hypopnea. (Kakkar R and Hill G. Interpretation of the adult polysomnogram. *Otolaryngol Clin North Am.* 2007; 40(3):733. Reprinted with permission.)

Because the PSG is labor intensive and its complexity necessitates specialized facilities, equipment, and staff, most communities in the United States either do not have the availability to provide such testing or cannot meet the demand in a timely fashion.^{17–20} This issue can be of particular concern since third-party payers require a PSG-based diagnosis for SRBD. For example, OSA on an annual basis necessitates a minimum of 2,310 PSG studies per 100,000 population to meet the current demand, yet

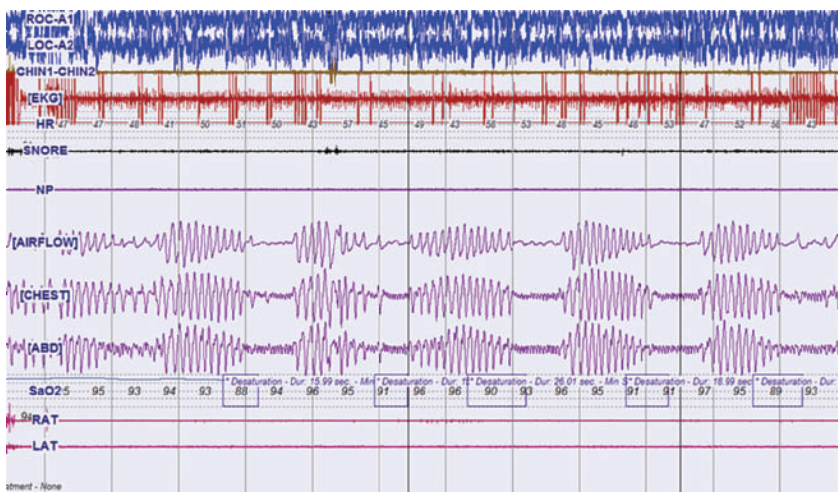


Figure 6.5 Cheyne–Stokes breathing. Note characteristic crescendo and decrescendo pattern in breathing effort, paralleling the airflow. (Kakkar R and Hill G. Interpretation of the adult polysomnogram. *Otolaryngol Clin North Am.* 2007; 40(3):736. Reprinted with permission.)

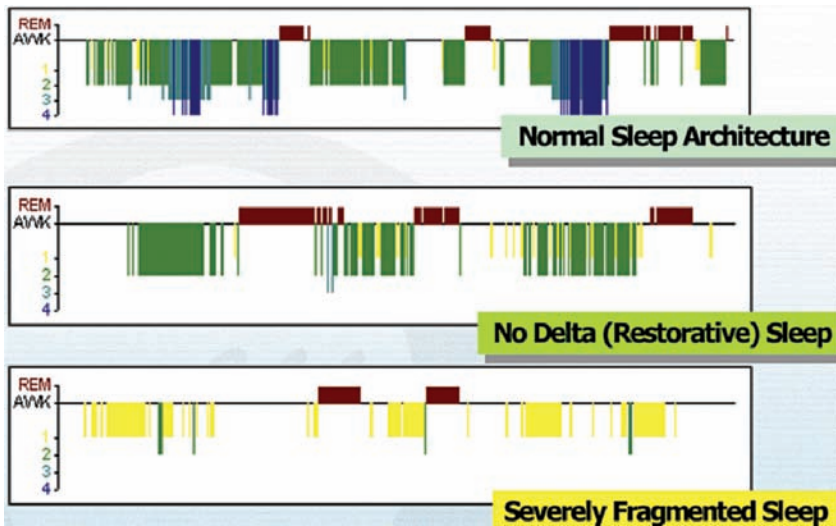


Figure 6.6 Hypnogram. (Kakkar R and Hill G. Interpretation of the adult polysomnogram. *Otolaryngol Clin North Am.* 2007; 40(3):727. Reprinted with permission.)

only 427 PSG studies per 100,000 population are conducted annually on the average.²¹

Multiple Sleep Latency Test

A Multiple Sleep Latency Test (MSLT) is commonly used to measure daytime sleepiness.^{8,22–24} The MSLT consists of a series of four to five 20-minute daytime naps during 2-hour intervals at a sleep laboratory/center, and the test often lasts 7–8 hours. In addition, the test is to begin 1.5–3 hours 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,^{25–27} including the instruction to the patient, “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 minutes is considered normal and less than 5 minutes is regarded as generally indicating the presence of a sleep disorder. Individuals who demonstrate a quick onset of rapid eye movement sleep are also more likely to have a sleep disorder.

Table 6.4 Indications for the use of the Multiple Sleep Latency Test (MSLT).

1	The MSLT is indicated as part of the clinical evaluation for patients with suspected narcolepsy to confirm the diagnosis.
2	The MSLT may be indicated as part of the evaluation of patients with suspected idiopathic hypersomnia to help differentiate hypersomnia from narcolepsy.
3	The MSLT is not routinely indicated in the initial evaluation and diagnosis of obstructive sleep apnea syndrome or in assessment of change following treatment with nasal continuous positive airway pressure (CPAP).
4	The MSLT is not routinely indicated evaluation of sleepiness in medical and neurological disorders (other than narcolepsy), insomnia, or circadian rhythm disorders.
5	Repeat MSLT testing may be indicated in the following situations: (1) when the initial test is affected by extraneous circumstances or when appropriate study conditions were not present during initial testing, (2) when ambiguous or uninterpreted findings are present, and (3) when the patient is suspected to have narcolepsy but earlier MSLT evaluation(s) did not provide polygraphic confirmation.

Source: Adapted from Standards of Practice Committee of the American Academy of Sleep Medicine. Practice parameters for clinical use of the multiple sleep latency test and the maintenance of wakefulness test. *Sleep*. 2005; 28(1):113–121.

Often the MSLT is the instrument of choice to definitively diagnose narcolepsy or idiopathic hypersomnia. The MSLT can also be used to document outcomes of treatment.^{28–30} Residual sleepiness can also be demonstrated for those individuals who do not report sleepiness after undergoing treatment.³¹

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.^{27,32} Table 6.4 identifies specific indications for the use of the MSLT from the AASM report.

Maintenance of Wakefulness Test

A Maintenance of Wakefulness Test (MWT) is commonly used to measure the alertness of an individual during the waking hours.³³ 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

Table 6.5 Indications for the use of the Maintenance of Wakefulness Test (MWT).

- | | |
|---|---|
| 1 | The MWT 40-minute protocol may be used to assess an individual's ability to remain awake when his or her inability to remain awake constitutes a public or personal safety issue. |
| 2 | The MWT may be indicated in patients with excessive sleepiness to assess response to treatment. |

Source: Adapted from Portable Monitoring Task Force of the American Academy of Sleep Medicine. Clinical guidelines for the use of unattended portable monitors in the diagnosis of obstructive sleep apnea in adult patients. *J Clin Sleep Med.* 2007; 3(7):737–747.

of four to five 20-minute daytime naps during 2-hour intervals at a sleep laboratory/center.³³ Although there are also similar standardized conditions under which the MWT is performed,^{25–27} 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.”^{27,34}

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

Practice parameters for the clinical application of the MSLT have been published by the AASM.^{27,32} Table 6.5 identifies specific indications for the use of the MWT from the AASM report.

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 excessive daytime sleepiness and thereby affording the ability to the Aviation Medical Examiners to reissue an airman medical certificate for commercial airline pilots.³⁵

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.^{36–38}

PORTABLE MONITORING DEVICES

The in-laboratory PSG is regarded as the current “gold standard” procedure for the evaluation of sleep and the diagnosis of SRBD, including but not limited to OSA, central sleep apnea, and Cheyne–Stokes breathing.¹² However, because of the associated high costs and issues of availability and accessibility of the PSG, there has been an increased interest in the use of portable monitoring (PM) devices as an alternative for the objective

Table 6.6 Indications for portable monitoring (PM).

- 1 PM for the diagnosis of obstructive sleep apnea (OSA) should be performed only in conjunction with a comprehensive sleep evaluation. Clinical sleep evaluations using PM must be supervised by a practitioner with board certification in sleep medicine or an individual who fulfills the eligibility criteria for the sleep medicine certification examination. In the absence of a comprehensive sleep evaluation, there is no indication for the use of PM.
- 2 Provided that the recommendations of #1 have been satisfied, PM may be used as an alternative to polysomnography (PSG) for the diagnosis of OSA in patients with a high pretest probability of moderate to severe OSA. PM should not be used in the patient groups with comorbid medical conditions (e.g., severe pulmonary disease, neuromuscular disease, or congestive heart failure), other sleep disorders (e.g., central sleep apnea, periodic limb movement disorder, insomnia, parasomnias, circadian rhythm disorders, or narcolepsy), or for general screening of asymptomatic patients.
- 3 PM may be indicated for the diagnosis of OSA in patients for whom in-laboratory PSG is not possible by virtue of immobility, safety, or critical illness.
- 4 PM may be indicated to monitor the response to non-CPAP treatments for OSA, including oral appliances, upper airway surgery, and weight loss.

Source: Adapted from Portable Monitoring Task Force of the American Academy of Sleep Medicine. Clinical guidelines for the use of unattended portable monitors in the diagnosis of obstructive sleep apnea in adult patients. *J Clin Sleep Med.* 2007; 3(7):737–747.

assessment of sleep as well as for measuring effectiveness of treatment for SRBD. Some studies state that PM devices can collect the same data as in-laboratory PSG.^{39,40}

In 1994, the AASM published its first practice parameters for the use of PM in assessing OSA.⁴¹ A review of the literature and evidence regarding ambulatory testing with PM devices was performed by the AASM,⁴² and a subsequent practice parameters report regarding the use of PM devices relative to OSA was published in 2003.⁴³ The most recent AASM guidelines for the clinical use of PM devices in assessing individuals suspected of having OSA were published in 2007.¹² Table 6.6 identifies specific indications for the use of PM devices from the 2007 AASM report. Specific indication #4 will be of interest to the dentist treating OSA patients with oral appliance (OA) therapy since PM devices may provide valuable follow-up objective documentation to supplement the patient's subjective documentation relative to the effectiveness of OA therapy:

This recommendation is based on Task Force consensus. PM may be used to monitor the efficacy of therapies other than CPAP when the diagnosis of OSA has already been made, either through PM or in-laboratory PSG.¹²

Up to this point, unless an OSA patient has undergone an in-laboratory PSG subsequent to the use of an OA, the only acceptable documentation for therapeutic effectiveness has been the subjective responses of the patient and/or the patient's bed partner.

CONCLUSION

Because of the multitude of categories of sleep disorders and the serious and life-threatening conditions of some of these disorders, it is typical for the evaluation process to include subjective and/or objective measurement instruments in order to obtain a complete assessment of the patient. There are various diagnostic modalities and instruments available. A subjective-based questionnaire-type of instrument may be useful for the practitioner to make appropriate clinical predictions. There are also in-laboratory sleep studies that may provide more comprehensive physiological assessments to assist in determining the diagnosis and appropriate management plan.

The dentist has an opportunity to be an integral component in the screening and management process for SRBD. In order to do so, it is prudent for the dentist to be familiar with the various diagnostic instruments and modalities available for sleep assessment.

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Evaluation by the dentist



CONCEPTUAL OVERVIEW

The dentist is called on today, more than ever, to be cognizant of related health care issues of their patients and not just of their dental and oral health status. This understanding and subsequent formal training in dental education began several decades ago with the recognition of hypertension when the blood pressure was taken at an initial visit or at a periodic visit for reevaluation, such as a dental hygiene visit. When the blood pressure was elevated, the patient was advised to contact their physician and have this evaluated more thoroughly. This heightened awareness led to the recognition of many people who were at risk for hypertension and who otherwise would have been undetected.

More recently, the association between periodontal disease and cardiovascular disease has been identified, and more aggressive steps are being taken clinically to resolve the periodontal condition in order to reduce the risk for cardiovascular disease. More than any other health care provider, oral cancer screening is another action that the dentist implements during the initial and follow-up care visits. Other examples are related to the recognition of oral conditions associated with systemic illnesses such as diabetes, leukemia, and many of the autoimmune diseases (e.g., Sjogren's syndrome).

Sleep disorders, and particularly obstructive sleep apnea (OSA), are no exception. Not only are sleep disorders prevalent in the general population, but they also have a potential for significant impact on an individual's health as well as on society. Sleep disorders may impair one's quality of life

and daily performance relative to schooling, driving or operating any other machinery, the workplace, and relationships.

The role of the dentist in the recognition of patients at risk for OSA and other sleep-related breathing disorders (SRBD), such as snoring, is now well established. The dentist is just as likely to identify a patient who is at risk for OSA as is the physician.¹ However, a study found that dentists had a general deficiency in their ability to recognize a patient at risk for OSA, and they also knew very little about the use of oral appliance (OA) therapy for the management of SRBD.² Also, only an estimated 16% of the dentists were taught anything about SRBD in dental school, and about 40% knew very little about OA therapy for the management of OSA. The study demonstrated the need for more education related to OSA and the use of an OA as an option for the management of the patient diagnosed with OSA.

WHAT THE DENTIST SEES THAT INDICATES THE RISK FOR SRBD

The dentist as well as the dental hygienist sees patients regularly who have signs of SRBD. However, unless the practitioner is knowledgeable of and recognizes the potential for these findings to suggest that there is a risk for SRBD, the sleep disorder may go undetected. Many of the conditions that may be identified by both the dentist and the dental hygienist that may indicate a risk for SRBD and health-related issues are commonly observed findings. Unfortunately, these findings often may be evaluated on their own merit as being stand-alone, and thus they may not be considered as potentially being related to some other health issue.

Once any of these conditions are recognized, then it becomes imperative to do the following: (1) determine if the risk for snoring or OSA is present, (2) inform the patient of the findings, and (3) consult with them regarding the appropriate measures needed for a complete diagnosis and management plan.

Many intra- and extraoral conditions have an association with risk for SRBD that warrant in-depth consideration (Table 7.1).

ASKING THE PROPER QUESTIONS

The addition of a few questions to the existing health history questionnaire is an important element of the data collection phase. These questions may not only uncover an individual who is at risk for snoring or having OSA, but they may also assist in the identification of someone who has been previously diagnosed with SRBD.

Table 7.1 Conditions that indicate the risk for a sleep-related breathing disorder: sleep apnea and snoring.

Observed condition	What this may indicate
Wear on the teeth	Indicative of sleep bruxism
Scalloped borders (crenations) of the tongue	Found to correlate with an increased risk for sleep apnea ¹²
Enlarged tongue	Increased potential for upper airway obstruction
Coated tongue	Possible gastroesophageal reflux disease
Enlarged, swollen, or elongated uvula	Increased potential for snoring or sleep apnea
Large tonsils	Higher incidence of airway obstruction
Narrow airway	Greater risk for snoring or sleep apnea
Gingival recession and/or abfraction	Greater potential for sleep bruxism (grinding or clenching)
Tongue obstructs view of airway (Mallampati score)	The greater the obstruction, the higher the potential for snoring and sleep apnea
Chronic mouth breather (poor lip seal)	Blocked nasal airway; more likely to snore

The basic questions that the dentist might include in the initial patient history form are the following:

- Do you or have you been told you snore when sleeping?
- Are you tired upon awakening from sleep or during the day?
- Do you fall asleep or are you drowsy in inappropriate situations such as in meetings, at movies, at church, or in social situations?
- Are you drowsy when driving?
- Do you have headaches in the morning?

If the response to any of these questions is positive, then additional questioning for a more comprehensive understanding of any potential sleep disorders may be necessary.

To further recognize a patient who may be at risk for OSA, the use of a common questionnaire known as the Epworth Sleepiness Scale (ESS) is utilized. The ESS identifies patients who are experiencing symptoms related to daytime sleepiness, which suggests the risk for OSA (Figure 7.1).³ This eight-item survey can be easily completed by the patient, and the scored results assist the practitioner in considering the appropriate course of action that may be advisable, which, most often, is a referral for a sleep study (polysomnogram) or to the patient's physician for further evaluation.

Epworth Sleepiness Scale

Situation	Chance of Dozing (0–3)			
Sitting and reading	0	1	2	3
Watching television	0	1	2	3
Sitting inactive (meeting, movie, church)	0	1	2	3
As a passenger in a car – for an hour – no break	0	1	2	3
Lying down to rest in the afternoon	0	1	2	3
Sitting and talking to someone	0	1	2	3
Sitting quietly after lunch (had no alcohol)	0	1	2	3
Stopped at a light or in traffic	0	1	2	3
	Total Score			
	0 = would never doze	2 = moderate chance of dozing		
	1 = slight chance of dozing	3 = high chance of dozing		

Figure 7.1 Epworth Sleepiness Scale—modified and adapted from original version. (Johns MW. A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. *Sleep* 1991; 14:540–545.)

Interpretation of the ESS score is a common means of communication within the sleep medicine field regarding the risk for OSA. As the total score approaches 9, the risk for OSA increases.⁴ As the total score becomes greater than 9, then the risk factors are considered to be even more significant. An elevated score, though, is not always definitive for OSA and is also not indicative of its severity. The results from the ESS also need to be considered in light of other clinical and patient history findings.

The second portion of the ESS evaluates the patient's behavior during sleep and more specifically some of the well-recognized characteristics associated with OSA. Snoring and its severity are assessed along with conditions associated with snoring that may suggest an increased risk for OSA such as waking up gasping for air or experiencing a choking sensation during sleep. If snoring is the only recognized condition along with the ESS total score being less than 9, then the risk for OSA may be less, but this is not always the case.

CLINICAL SCREENING FOR SRBD

Once it has been determined that a patient is at risk for SRBD, it may be advisable to perform a sleep disorder screening examination. In most instances, a significant amount of clinical information regarding the patient's dental and medical status and history has already been collected. The screening evaluation will supplement the existing record with documentation that is designed to identify relevant conditions that support the possible risk for SRBD, in particular for OSA.

Table 7.2 reflects the progression of steps that might be considered to assess the patient who is at risk for SRBD.

Table 7.2 Steps for assessment of the patient at risk for a sleep-related breathing disorder and sleep apnea.

Step 1:	Recognition of existing risk factors (Table 7.1)
Step 2:	Positive response(s) to the health history questions
Step 3:	Completion of the Epworth Sleepiness Scale
Step 4:	Discussion with the patient regarding the positive responses from above
Step 5:	Reappointment for clinical screening evaluation
	Consultation to discuss findings
	Make recommendations for management plan
	Management options
	<ul style="list-style-type: none"> ● Refer to patient's physician for further evaluation ● Refer for a sleep study

Source: Treatment Sequencing. Handout for the UCLA School of Dentistry Dental Sleep Medicine Mini-Residency; 2009.

There are a number of components that should make up an SRBD screening evaluation, including SRBD history, review of medical history, review of current medications, temporomandibular disorders (TMD) assessment, oral airway evaluation, nasal airway evaluation, and subjective airway testing.

SRBD history

The SRBD history is designed to obtain patient's history-related findings that are specific to SRBD, such as the following patient symptoms or previously diagnosed conditions:

- Snoring
- Sleep apnea
- Low energy
- Daytime sleepiness/tired
- Difficult to concentrate
- Previous or current use of positive airway pressure therapy
- Previous surgery for SRBD
- Mood swings/irritable
- Feel depressed
- Headaches
- Bruxism (grinding and/or clenching)

Review of medical history

The patient's medical history may be indicative of an underlying sleep issue. A number of preexisting medical conditions may suggest an increased risk for SRBD, particularly OSA, such as the following:

- Hypertension
- Cardiovascular disease

- Headaches
- Respiratory conditions (especially asthma)
- Diabetes
- Gastroesophageal acid reflux disease
- Hypothyroidism
- Allergy

Review of current medications

The patient's current medications need to be reviewed. There may be prescription medicines that are being used for the management of a medical condition, yet the condition may be related to a sleep disorder. In addition, many medications may have an impact on the patient's sleep.

Medications and sleep

Almost all medications that are taken can impact sleep in some manner. Table 7.3 outlines some of the more common medications that are frequently encountered in a dental practice and which may impact sleep.

Not all patients have similar responses to medications, and they may not experience an adverse effect on their sleep. Also, patients may be taking medications for a particular health issue, and this may also be an indicator that a sleep disorder is present but may have been overlooked or not considered. In addition, there are many medications that are used to promote and improve sleep.

Medications by class associated with sleepiness

As reported in clinical trials and case reports

- Antihistamines
- Anti-Parkinson agents
- Skeletal muscle relaxers
- Opiate agonists
- Alcohol

Natural or alternative medications

- Ginseng
- St. John's Wort
- Valerium
- Dehydroepiandrosterone (DHEA)
- Ephedra
- Vitamin C

Medications associated with insomnia

- Amphetamines
- Caffeine
- Nicotine

Table 7.3 Effect of common medications on sleep.

Medication	Effect on sleep
Aspirin and ibuprofen in healthy subjects	Disrupts sleep architecture Increases sleep latency Increases nonrapid eye movement (NREM) stage 2 sleep Increases slow-wave sleep Decreases sleep efficiency (Note: When pain is present, these medications may improve sleep)
Opioids	Increases NREM stage 2 sleep Decreases slow-wave restorative sleep Worsens SRBD or may induce it (respiratory depression)
Methadone	Known to precipitate central sleep apnea
Tricyclic antidepressants	Increase total sleep time Increase NREM stage 2 (a stage when bruxism increases) Decrease arousals Increase rapid eye movement (REM) latency Decrease REM
Trazodone	Increases total sleep time Decreases sleep latency (Note: good long-term sleep aid)
Benzodiazepines	Decreases sleep latency Increases NREM stages 1 and 2 Increases total sleep time Decreases slow-wave restorative sleep Decreases REM Increases sedation
Antidepressants (selective serotonin reuptake inhibitor)	Increase wakefulness Decrease total sleep time Slightly increase NREM stage 1 Decrease REM May induce insomnia May cause sleep bruxism

Sources: Adapted from (1) Lee-Chiong T. Sleep: A Comprehensive Handbook. Hoboken, NJ: John Wiley & Sons. 2006. (2) Kryger MH, Roth T, and Dement WC. Principles and Practice of Sleep Medicine. Philadelphia: Elsevier/Saunders. 2005. (3) Pagel JF. Medications effects on sleep. In: Attanasio R and Bailey DR, eds. Sleep Disorders: Dentistry's Role (Dental Clinics of North America, 45:4). Philadelphia: W.B. Saunders. 2001;855–865.

- Corticosteroids
- Theophylline

Medications for the treatment of insomnia

- Sonata (zaleplon)
- Ambien (zolpidem)

- Lunesta (eszopiclone)
- Dalmane (flurazepam)
- Restoril (temazepam)
- ProSom (estazolam)
- Halcion (triazolam)—increases NREM stage 2 and interferes with slow-wave sleep
- Rozerem (ramelteon)—acts on melatonin receptors (M1 and M2)

Medications that impact respiratory drive

May have an effect on OSA and chronic obstructive pulmonary disease

- Benzodiazepines
- Barbiturates
- Narcotics
- Topamax

Antihypertensives' effects on sleep

- Beta agonists (Propranolol)
 - Increase wakefulness
 - Increase NREM stage 1
 - Decreased REM
- ACE inhibitors: Lotensin, Vasotec, Monopril, Zestril, Accupril, Altace
 - Increased insomnia
- Diuretics (HCTZ)
 - Drowsiness
- Calcium agonists
 - No sleep study data

Medications that increase slow-wave sleep

- Gabatril (tiagabine)
- Gabapentin (Neurontin)
- Pregabalin (Lyrica)
- Trazadone (Desyrel)
- Mirtazepine (Remeron)
- Valdoxan (Agomelatine)—a new antidepressant that is in the third phase of clinical trials; also increases slow-wave sleep

For further details, it is recommended that each specific medication be evaluated with the use of current literature dedicated to this topic.

When evaluating a patient who has a sleep disorder, medication use needs to be taken into consideration as a factor. One study demonstrated that the use of an antidepressant or antihypertensive increases the risk for OSA.⁵ The use of these two agents at the same time increases the risk for OSA significantly.

Temporomandibular disorders assessment

It is important to be aware of a patient's status relative to past or existing TMD, which may involve the temporomandibular joint (TMJ) and/or the masticatory muscles. Although the TMD evaluation is often included as part of the initial new patient examination for every patient in a dental practice, a number of patients that present with a TMD condition may also have an underlying sleep disorder, and this may affect the overall management plan of the patient.

If a TMD condition is present, it is important to document its existence so that it can be further assessed should an OA be fabricated for OSA and/or snoring at some point in the future. For example, if OA therapy is being considered for management of an intracapsular disorder, such as a recent onset of a disc displacement with reduction, and there is also an OSA condition, then an OA design can be considered that may address both issues.

Temporomandibular joint

In addition to recording any findings regarding sounds and tenderness to palpation of the TMJs, there should be documentation regarding the patient's mandibular range of motion.

A screening assessment of the TMJs may include the following components:

- Previous treatment, including OA therapy
- Joint tenderness (capsule, retrodiscal)
- Joint sounds (clicking, crepitus, popping)
- Range of motion (opening, protrusion, lateral excursions)

Masticatory and cervical muscles

Palpation of the muscles of the head and neck should be performed to determine if there is any local tenderness or referred pain patterns. An awareness of these masticatory and cervical muscles is essential in determining the source of pain. The muscles that were found to be tender should be recorded for future reference.

Oral airway evaluation

The following components should comprise the oral airway evaluation:

- Uvula
 - Normal
 - Enlarged/swollen
 - Elongated
 - Surgically removed

- Soft palate
 - Normal
 - Enlarged/swollen
 - Slopes downward into the oropharynx
- Gag reflex
 - Normal
 - Diminished
 - Absent
 - Exaggerated
- Tonsils grade (0, I, II, III, IV)

Dentition and supporting structures

It is important that the patient's current dental health status be recorded, which includes the teeth as well as the supporting structures. The occlusion and maxillomandibular relationship are major factors because of the concern that exists for potential changes in these areas that may be associated with the use of an OA.

Components of documentation for the dental and supporting structures evaluation include the following:

- Classification of occlusion (I, II, III; Div 1, Div 2)
- Deep bite
- Crossbite
- Maxillary incisors (retroclined, normal)
- Wear facets on the teeth (mild, moderate, severe)
- Periodontal status (no disease, gingivitis, recession, halitosis, abfraction, teeth mobility)
- Hard palate (narrow, high)
- Lip seal (strained/forced, no lip seal, lips dry/chapped)

Importance of lip seal

Assessment of the patient's ability to maintain a lip seal and identification of any indicators for mouth breathing are critical components of the oral airway evaluation. Lack of a lip seal and the resulting mouth breathing pattern or habit is also indicative of an individual who may have the following: (1) difficulty breathing comfortably through the nose, (2) allergies, or (3) nasal airway obstruction. Both mouth breathing and limited nose breathing may contribute to an increase in inspiratory pressure as well as to snoring and OSA because of airway compromise (Figure 7.2).

It is helpful to recognize someone who may be a mouth breather. When an individual is sitting comfortably in a relaxed position, the lips should be comfortably together without any appearance of being strained. If the lips are not in contact and are apart, this is usually indicative of a chronic mouth breathing pattern, often referred to as an obligate mouth breather. When this same individual attempts to close the lips, it will appear strained. In



Figure 7.2 Mouth breathing appearance: (a) lips apart at rest and (b) forced or strained lip seal.

addition, the chin may appear tight or wrinkled, oftentimes a sign of increased mentalis muscle activity (Figure 7.3).

Tongue assessment

Evaluation of the tongue includes observation for scalloping, size, or coated surface. In addition, the Mallampati score assesses tongue position relative to the soft palate as well as visualization of the oropharynx as indicators of the risk for OSA. The assessment's scoring has been revised from



Figure 7.3 Lips closed at a relaxed position without strain—note the lack of wrinkling in the area of the chin.

when it was initially developed and used by anesthesiologists to assess the difficulty of intubation.⁶

To determine a score, the mouth is held open with the tongue at a rest position as compared to the version utilized by the anesthesiologist where the tongue is protruded. In both cases, the position is graded from I to IV (Figure 7.4).

As the degree of obstruction of the oropharyngeal airway and the soft palate increases, the risk for OSA also increases. It has recently been demonstrated that as the score progresses from I to IV, the potential severity of OSA also worsens.⁷ The study also found that for each 1-point increase in the Mallampati score, the odds of having OSA were more than twice as likely, and the apnea–hypopnea index (AHI) may increase more than 5 events per hour (Table 7.4).

Evaluation of the tongue should include the following components:

- Large
- Coated
- Scalloped
- Fissured
- Tongue-tied (lingual frenum restricts movement)
- Mallampati score: o I o II o III o IV

Uvula assessment

The appearance of the uvula may also indicate the risk for OSA and/or snoring. The uvula may appear enlarged, swollen, elongated, and even bruised (Figure 7.5). Negative intrapharyngeal pressure is associated with a narrowed or obstructed airway, and these clinical findings may be a result of the mechanical trauma associated with the snoring and obstructive breathing events.

Soft palate assessment

Observation of the soft palate is another necessary component of the evaluation by the dentist because of the clinical significance of the slope or length of the soft palate. The more that the soft palate slopes down into the oropharyngeal space, the greater is the potential impact for airway obstruction. In addition, the more the soft palate slopes downward, the higher the Mallampati score.

As with the uvula, the soft palate may also appear swollen from the mechanical trauma associated with snoring and/or OSA.

Gag reflex

In patients who snore or have sleep apnea, the gag reflex may be impacted by neurological alterations in this response, resulting in a less pronounced

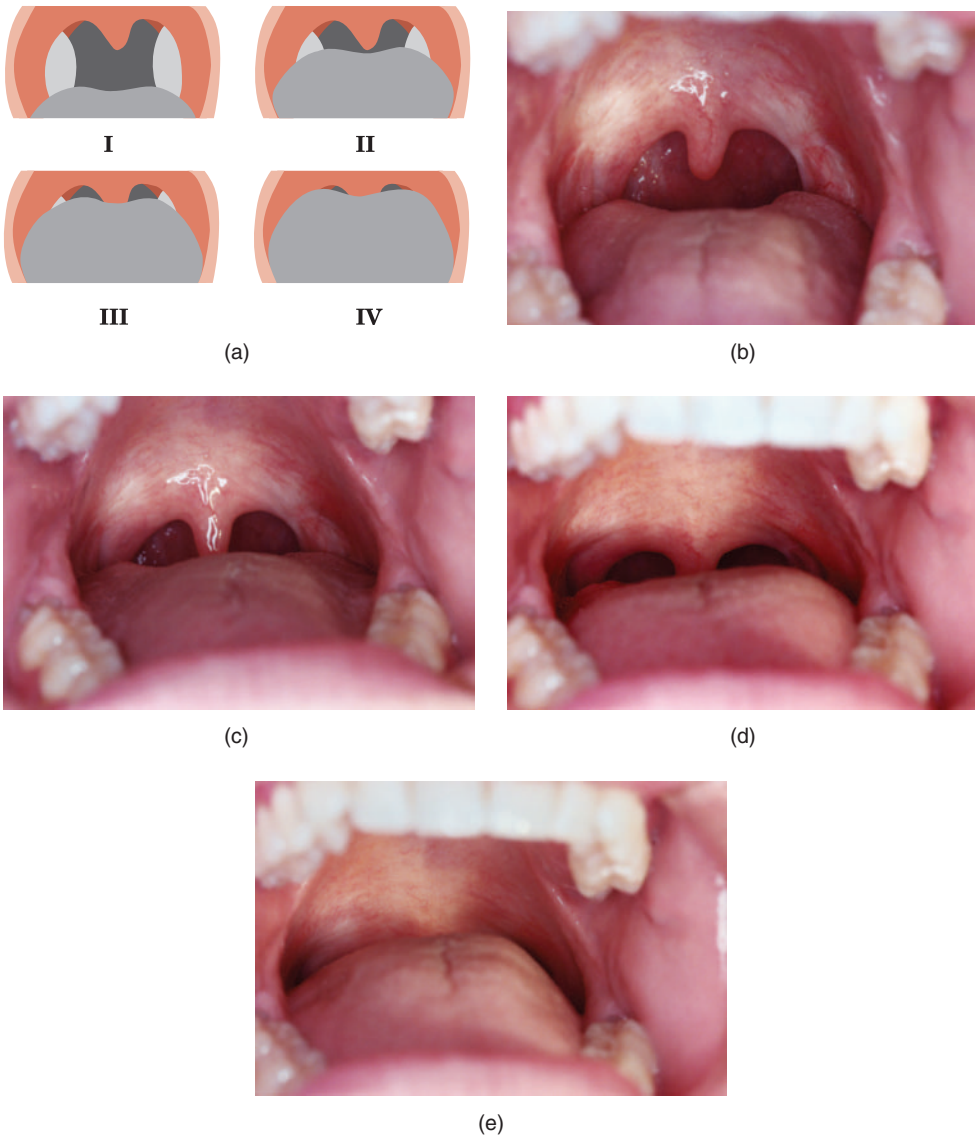


Figure 7.4 Illustrations of the Mallampati score: (a) diagrams of the four designations (I—a clear view of oropharynx, uvula, and soft palate; II—a limited view of the oropharynx with a view of most of the uvula and soft palate; III—unable to view the oropharynx, the uvula, and only a portion of the soft palate; IV—view of oropharynx, uvula, and soft palate totally obstructed by the tongue), (b) example of the Mallampati I, (c) example of Mallampati II, (d) example of Mallampati III, and (e) example of Mallampati IV.

Table 7.4 Example of effect of Mallampati score on OSA and AHI.

Mallampati score	Odds ratio for OSA	Possible AHI
I	1	5
II	2.5	10 or more
III	5	15 or more
IV	7.5	20 or more

Source: Adapted from Nuckton TJ, Glidden DV, Browner WS, et al. Physical examination: Mallampati score as an independent predictor of obstructive sleep apnea. *Sleep*. 2006; 9(7):903–908.

or even absent reflex. Even though an altered gag reflex may not always be present, it is advisable to screen for this clinical finding. If this is suspected during the course of a routine oral examination, then the possibility of snoring and/or OSA should be considered.

Tonsils assessment

The enlargement of the tonsils may contribute to airway obstruction as well as an increased tendency for mouth breathing. This enlargement may also compromise the airway and contribute to snoring and OSA. This is particularly true in children and adolescents. In adults, this may also be the case but to a lesser degree.

The standard grading system for the tonsils rates them on a scale from 0 to IV, with 0 indicating that the tonsils are absent and grade IV indicates they are grossly enlarged (Figure 7.6).⁸

Typically as one goes through puberty, the size of the tonsils will decrease to a grade I or 0. In some situations this will not occur, and this is when they may impact the airway. Thus, the evaluation of the tonsils should be a routine part of the oral airway evaluation.

**Figure 7.5** The uvula: (a) normal size and (b) enlarged/swollen.



Figure 7.6 Typical grading of the tonsils: 0, removed or not visible if present; I, barely visible; II, enlarged with limited obstruction; III, enlarged with significant obstruction; and IV, grossly enlarged and obstructive (“kissing tonsils”).

Nasal airway evaluation

Nose breathing is the preferred mode of respiration despite the fact that many patients are habitual mouth breathers. Chronic mouth breathing is often associated with nasal airway obstruction. It is within the scope of the dentist to perform a nasal airway screening to assess the status of the nasal airway.

To help determine if the patient perceives nasal airway problems during the last month, the use of a scale called the Nasal Obstruction Symptom Evaluation (NOSE) Instrument may be utilized (Figure 7.7).⁹

In addition to the instrument itself, a visual analog scale is used to assess the difficulty on average for nose breathing. The results may be helpful in

Nasal Airway / Breathing Assessment
 Recently how much have the following conditions been a concern or problem (place a mark on the line that best describes your situation)

	<u>Minimal</u>	<u>Mild</u>	<u>Moderate</u>	<u>Severe</u>
Nasal congestion stiffness obstruction	-----			
Difficult to nose breathe when sleeping	-----			
Difficult to nose breathe with exercise or exertion	-----			
Difficult to nose breathe in general	-----			

Figure 7.7 The nasal airway/breathing assessment. (From Stewart MG, Witsell DL, Smith TL, et al. Development and validation of the Nasal Obstruction Symptom Evaluation (NOSE) scale. *Otolaryngol Head Neck Surg.* 2004; 130:157–163.)

determining if further evaluation is needed or if a referral to an otolaryngologist may be indicated. The assessment may also be useful in evaluating if treatment designed to improve the nasal airway has been successful.

The nasal airway is important because it performs three main functions, basically acting as the carburetor of the body. Air passing through the nose is warmed and humidified to an 80% level, both of which contribute to the absorption of oxygen by the lungs.¹⁰ In addition, air passing through the nose is also filtered.

Nasal airway anatomy

The anatomy of the nasal airway starts at the outer portion of the nose with the alar rim or external nasal valve and the columella that separates the two nostrils (Figure 7.8).

Evaluation of the inner portion of the nose reveals structures that may act to restrict nasal airflow. To observe the inner anatomy of the nose, it is helpful to use a nasal speculum (Figure 7.9). This instrument allows for an improved visualization of the inferior turbinates, the nasal septum, and the nasal valve.

In order to adequately see inside the nose, a bright light source is necessary. This can be obtained with a bright flashlight or with a nasal illuminator (Figure 7.10). The internal aspect of the nose can then be better visualized to assess some critical structures.

The perceived nose is actually two separate components. The portion that is more anterior is the externally visible portion of the nose, and the

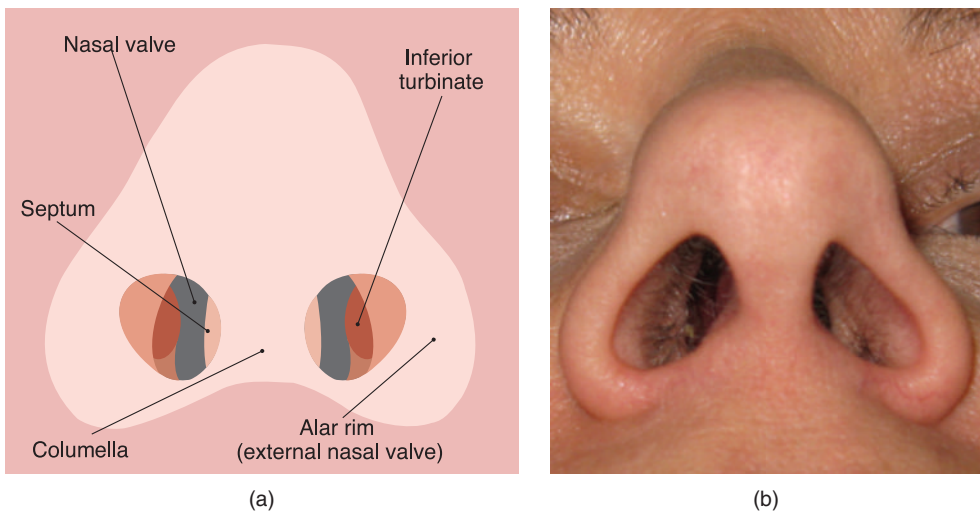


Figure 7.8 The nose. It demonstrates the outside area of the nose—the part surrounding the nose is termed the alar rim and the mid-section that divides the two nostrils is termed the columella. (a) Diagram of nose and (b) clinical picture of nose.

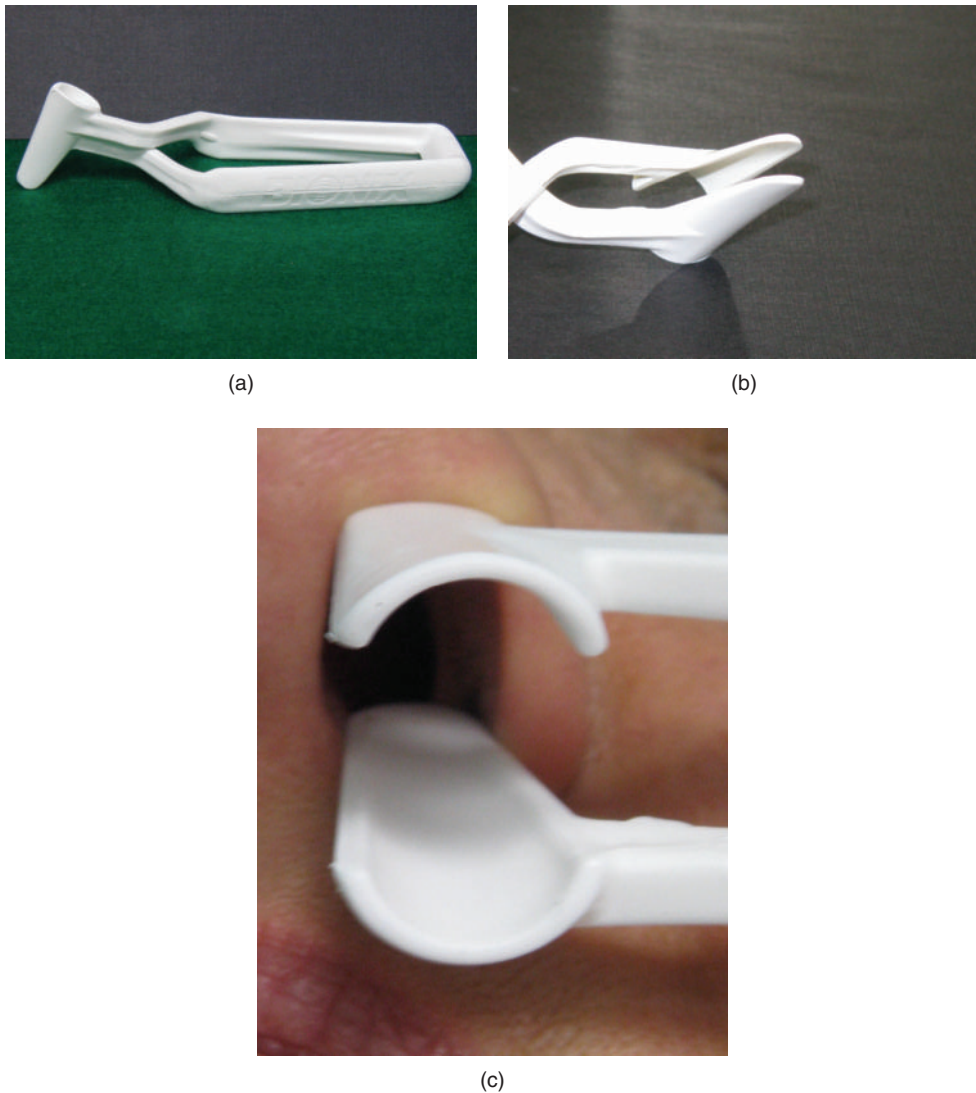


Figure 7.9 Nasal speculum: (a) disposable speculum, (b) speculum with the beaks opened, and (c) speculum in use.

part behind this that connects to the oropharynx is the nasopharynx. These two areas are separated by the posterior choanae.

The use of a nasal speculum will allow for observation of the following structures (Figure 7.11):

- The turbinates, especially the inferior turbinates, are present at the lateral aspect of the nasal airway.

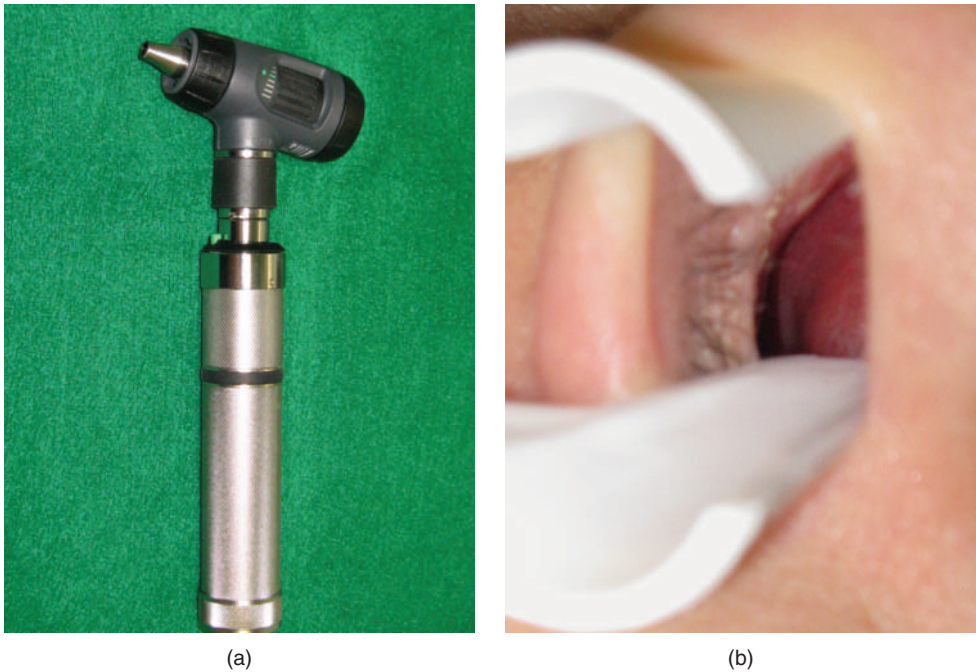


Figure 7.10 Illuminator used for visualization into the nose as part of the nasal airway evaluation: (a) otoscope with adaptor for visualization and (b) visualization of the inside of the nose with light source in use.

- The nasal septum is at the midline of the nose and separates it into two compartments. Deviation of the septum may be observed.
- The nasal valve can be assessed to determine its width and degree of opening. This is not an anatomic structure, but rather an area whose boundary is the nasal septum and the inferior turbinates.

It is the nasal valve that regulates airflow through the nose. This valve is subject to many different conditions that can affect it such as allergy, inflammation of the inferior turbinates, and nasal septum deviation. However, airflow can also be impacted by the presence of other pathology in the nose, such as polyps.

Even a small change in the opening of the nasal valve may result in significant improvement, and this observation is referred to as Poiseuille's Law. Regarding the nasal airway, the inspiratory pressure required to draw air through the nose is impacted by the fourth power of the radius. Thus, a small change or improvement in the opening of the nasal valve significantly decreases the pressure required to inspire air. An increase in nasal airway obstruction leads to an increase in inspiratory pressure, which results in airway collapse and an increased risk for OSA.¹¹

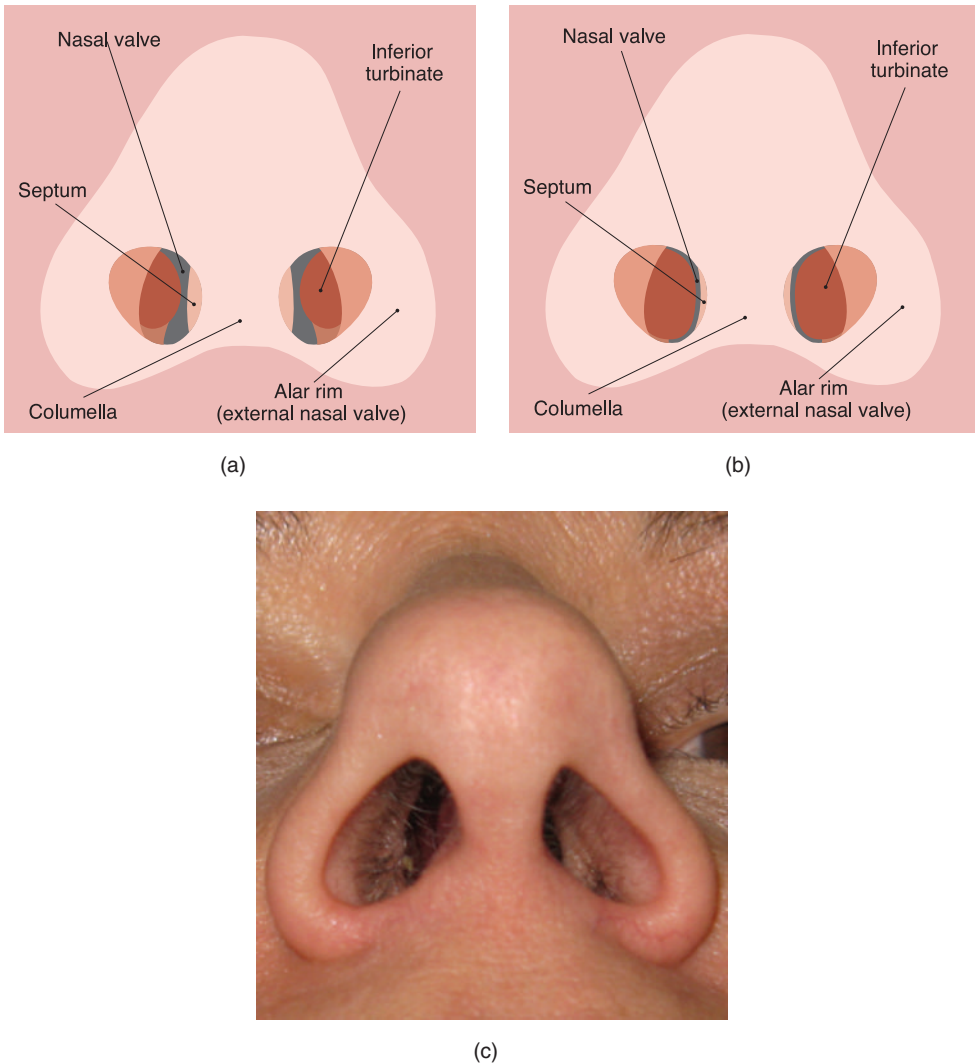


Figure 7.11 View inside the nose: (a) diagram of nose, (b) nose with enlarged inferior turbinates and narrowed nasal valves, and (c) clinical picture of the nasal airway.

Components of the nasal airway evaluation should include the following:

- Nasal airway (open, obstructed, stuffy, septal deviation)
- Inferior turbinates, both right and left (normal, enlarged)
- Columella (normal width, wide, compression improves breathing)
- Nasal valve, both right and left (open/normal, narrow, blocked)
- Effect of nasal dilation (Cottle Test) (improved breathing; no effect)



Figure 7.12 Means of testing mandibular position to determine if there is subjective improvement in the airway and if the snoring is reduced or eliminated: (a) using cotton rolls between the posterior teeth and (b) using the George Gauge.

Subjective airway testing

As a component of the overall SRBD evaluation, it is important to determine if the patient experiences an improvement in their breathing and/or a reduction in the ability to snore if the mandible is repositioned. This testing may be assessed by using a prop, such as a bite stick, or by placing half of a cotton roll between the maxillary and mandibular posterior teeth to open the vertical between 2 and 5 mm, and subsequently having the patient move the mandible to an edge-to-edge position (Figure 7.12).

With this repositioning exercise, the patient needs to maintain a lip seal as well. With the mandible repositioned, have the patient practice breathing mainly through the nose. Determine at this point if they perceive an improvement in their breathing.

To assess if the mandibular repositioning has affected the ability to snore, ask the patient to make a snoring sound prior to the repositioning exercise. Subsequently with the mandible opened and advanced, have the patient attempt to snore. With the mandible opened and advanced, the ability to snore is often decreased and/or eliminated.

If the airway does not feel as if it has improved or if the snoring was not significantly impacted, then additional attempts with this same exercise at varying degrees of opening and/or advancement may be attempted. The results of this type of testing can be documented on the screening evaluation form.

MANAGEMENT PLAN

Once all the data from the various components of the evaluations have been completed, a plan of action needs to be presented to the patient. This

management plan may take on a number of options depending on the scope of the treatment and the degree to which the practitioner wishes to be involved. The overall outcome most often will involve either the patient going on for further testing such as a sleep study or getting an OA. Regardless, various options should be explored as part of the consultation:

- Schedule
 - Consultation with the dentist
 - Schedule a more detailed evaluation
- Refer patient for a sleep study or to the physician
- Patient had a sleep study—obtain a copy for review
- Patient tried positive airway pressure therapy and/or had surgical intervention—consider OA therapy
- Schedule for OA therapy
- Need additional records
 - Panoramic radiograph
 - Cephalometric radiograph
 - Cone beam imaging
 - TMJ tomograms
- Refer for
 - Physical therapy
 - Myofunctional therapy
 - Otolaryngology evaluation
- Recommend
 - Commercial nasal dilator (e.g., Breathe Rite[®] strips)
 - Commercial sinus rinse (e.g., Neil Med[®] Sinus Rinses)

CONCLUSION

The evaluation of the patient presenting for dental care also should be viewed as an opportunity to screen for health-related issues as well, and SRBD is no exception. A particular case in point is the screening of the periodontally involved patient who is at risk for cardiovascular disease. There are similar findings that may indicate a risk for SRBD.

Once the possibility of SRBD is recognized, then additional steps can be taken to further evaluate the patient. The outcome may lead to the ability to provide a service, such as a management plan and even treatment (e.g., OA therapy) that can ultimately improve the patient's quality of sleep and hence their quality of life.

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8 Imaging for sleep-related breathing disorders

CONCEPTUAL OVERVIEW

Imaging of the airway and related structures is used for a variety of reasons. For the most part, imaging has been utilized in an effort to measure anatomical structures of the airway, to understand the dynamics of the airway with and without therapeutic interventions, and to better understand the pathophysiology of sleep-related breathing disorders (SRBD).

In the dental management of SRBD, the use of imaging to predict whether an individual may be at risk for obstructive sleep apnea (OSA) or snoring is limited at this time. As techniques are developed and refined, the possible use of imaging may be of some benefit. Currently, imaging as it relates to the diagnosis of upper airway compromise as well as to assess the dynamics of airway collapse is mainly for research purposes.

IMAGING IN THE DENTAL OFFICE FOR SRBD

The use of imaging associated with the treatment of SRBD in dentistry may be considered in three circumstances:

1. To predict the presence or risk in an individual for SRBD
2. To assess dental and related structures pertaining to the treatment of SRBD, primarily with an oral appliance (OA)
3. To determine if mandibular repositioning will improve the airway

There are a number of imaging options for the dentist to consider related to these three circumstances (Table 8.1).

Table 8.1 Imaging options related to sleep-related breathing disorders and mandibular repositioning.

Condition	Imaging option
Predict SRBD	Cephalometric X-ray Cone beam CT Pharyngometry
Assess condition of dental and related structures	Panoramic X-ray Cephalometric X-ray Cone beam CT Cone beam CT
Determine if mandibular repositioning will improve the airway	Pharyngometry

The three most common imaging options that can be utilized are computed tomography (CT), magnetic resonance imaging (MRI), and nasal-pharyngoscopy. These modalities are used on a limited basis and typically are not employed in the treatment of SRBD.

Computed tomography and magnetic resonance imaging

CT and MRI scans may be useful if other pathology is being investigated as a possible factor in patients with sleep disorders. These imaging modalities have been shown to be resourceful when a secondary medical or neurological cause for a sleep disorder was suspected.^{1,2}

CT is frequently employed by the otolaryngologist to evaluate the nasal airway and the sinuses, particularly for discerning the presence of any airway compromise from craniofacial structures.

CT has also been utilized to do research regarding the airway. A study using dynamic CT determined the impact of an OA on the airway, in particular examining the effect of the OA as it advanced the mandible, also known as anterior or mandibular repositioning, from its habitual maxillomandibular relationship.³ The results demonstrated that the OA appeared to have a greater impact on the lateral aspect of the airway in the retropalatal and retroglottal areas.

MRI has been used extensively to study the dynamics of the upper airway in a variety of circumstances (Figure 8.1).^{4,5} This particular imaging modality is not practical, nor it is indicated in everyday clinical use on a routine basis. It needs to be utilized in specific circumstances mostly related to research endeavors. It would be infrequent for the dentist to order this imaging modality for clinical purposes because of the associated expense to the patient, the difficulty for patients to initiate and maintain sleep in the noisy scanner, and the potential exclusion of some patients with metallic implants or pacemakers.

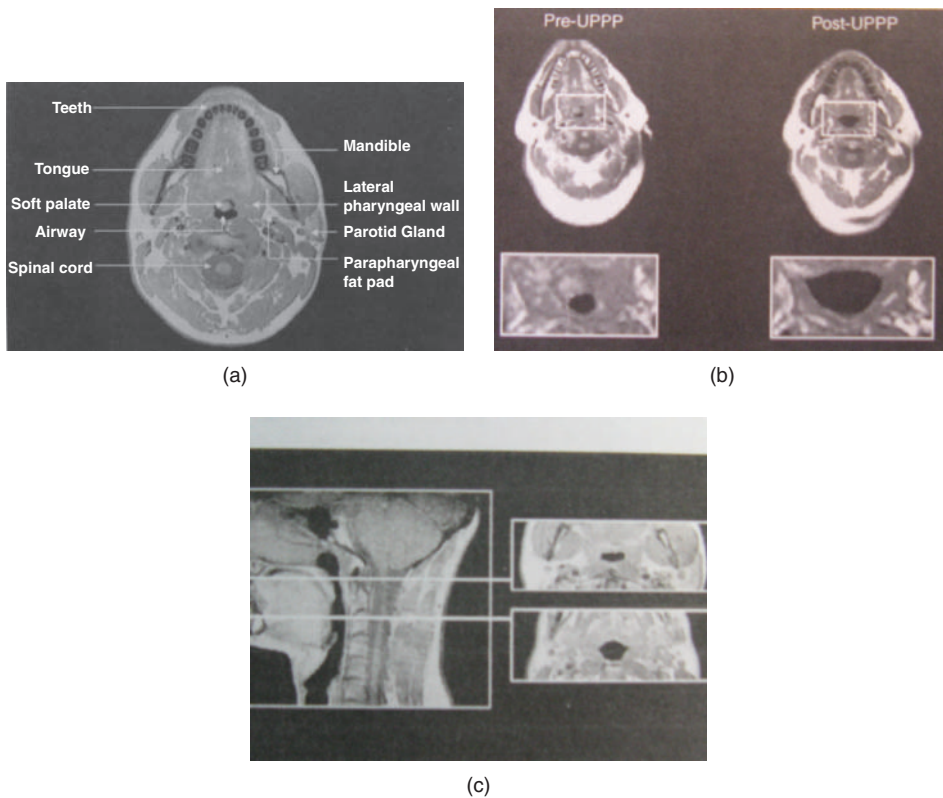


Figure 8.1 Cross-section MRI views of the airway: (a) general view, (b) the airway pre- and post-uvulopalatopharyngoplasty surgery, and (c) the airway at two levels: retropalatal and retroglottal. (Lee-Chiong T, ed. *Sleep: A Comprehensive Handbook*. Hoboken, NJ: John Wiley & Sons. 2006. Used with permission).

Nasalpharyngoscopy (fiber optic pharyngoscopy)

Nasalpharyngoscopy, also known as fiber optic pharyngoscopy, is frequently used by the otolaryngologist to evaluate the airway. A flexible tube with a fiber optic light and camera allow for both dynamic and state-dependent visualization of the airway from the nose down to the larynx (Figure 8.2).

During this evaluation, the effect of inspiration with the nose and mouth closed, termed the Mueller maneuver, is observed.⁶ This clinical technique replicates the effect of obstructive events in the airway, thereby indicating the impact of apnea events on the airway as well as identifying the specific site of obstruction associated with OSA. One study found that 60% of the sleep apnea patients had complete occlusion of the airway, 40% had multiple sites of obstruction, and there was reduced size along with increased collapsibility of the airway that correlated with an increase in the apnea-hypopnea index.⁷

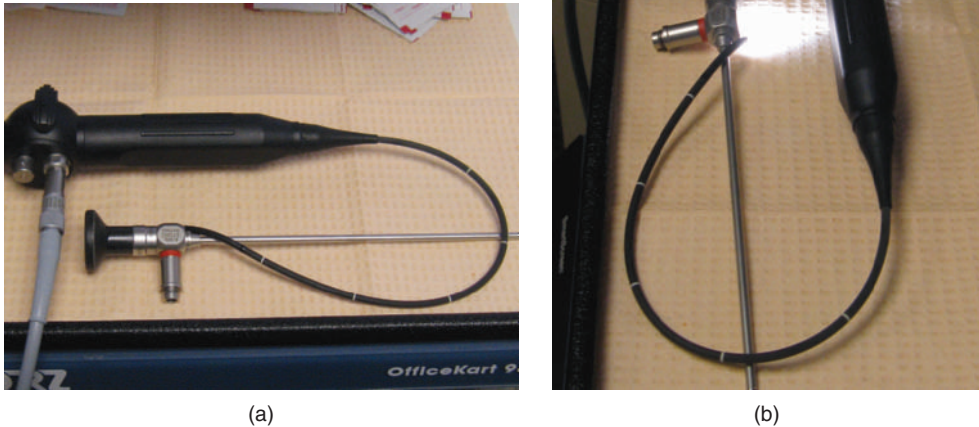


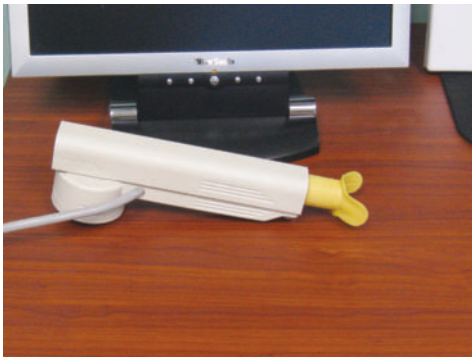
Figure 8.2 View of rigid and flexible fiberoptic scope used to visualize the nasal airway and oropharynx down to the level of the epiglottis: (a) the rigid and flexible scopes and (b) the scopes showing the illumination.

Pharyngometry

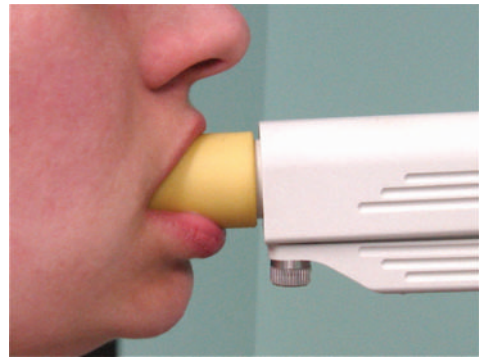
Pharyngometry, also known as acoustic reflection imaging (ARI), is another technique that has been utilized to evaluate the airway in patients with OSA and/or snoring (Figure 8.3). This modality emits a sound wave through the mouth that is helpful in the identification of the site(s) of narrowing from the oropharyngeal junction to the hypopharynx.

ARI may also determine the impact of mandibular repositioning on the airway and to what extent tongue space may increase. The technique for this modality is not standardized in terms of how it is performed, and it is therefore subject to the procedural handling by each individual user. The advantage of ARI is that it may assist the dentist in determining the potential for the success of OA therapy.⁸ It was demonstrated that when the airway volume was increased as measured by ARI, there was a 60% chance that an OA would have a favorable prognosis. If the airway volume was unchanged, the possibility that an OA would not be of benefit was 95%. This modality may also be helpful with the postinsertion assessment of the OA relative to whether any further adjustments to the therapeutically induced maxillomandibular relationship may be helpful or necessary.

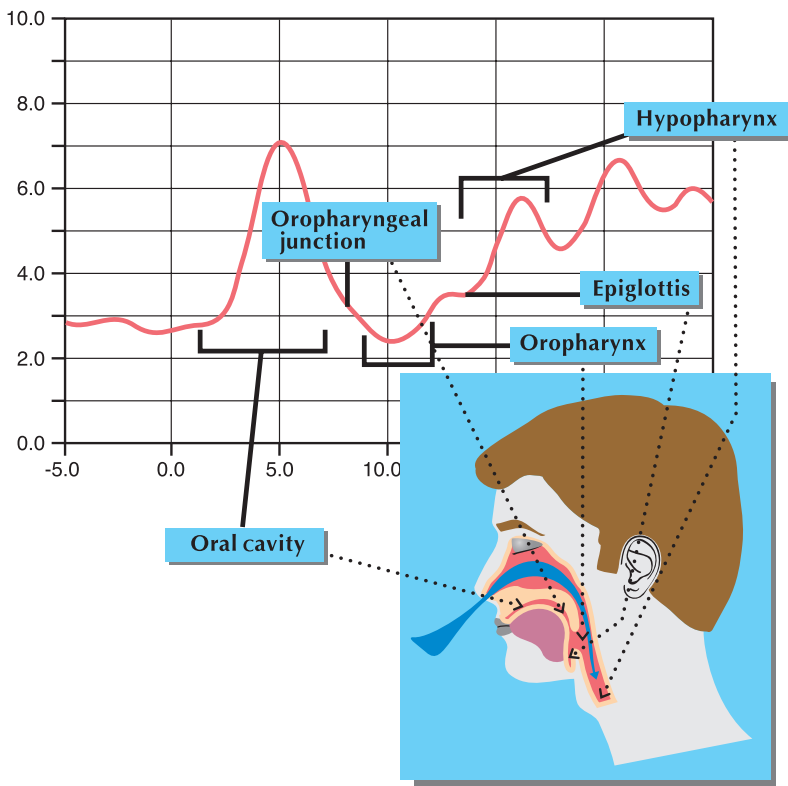
One study found that ARI could assist in predicting the risk for SRBD as related to gender and ethnic differences.⁹



(a)



(b)



(c)

Figure 8.3 Acoustic reflection imaging—pharyngometer: (a) general view of the wave tube that emits the sound waves and the oral mouthpiece—computer monitor can be seen in the background, (b) close-up view of the testing equipment in use, and (c) diagram of the image seen on the monitor that demonstrates the areas of the airway as they would be viewed at the various levels of the airway.

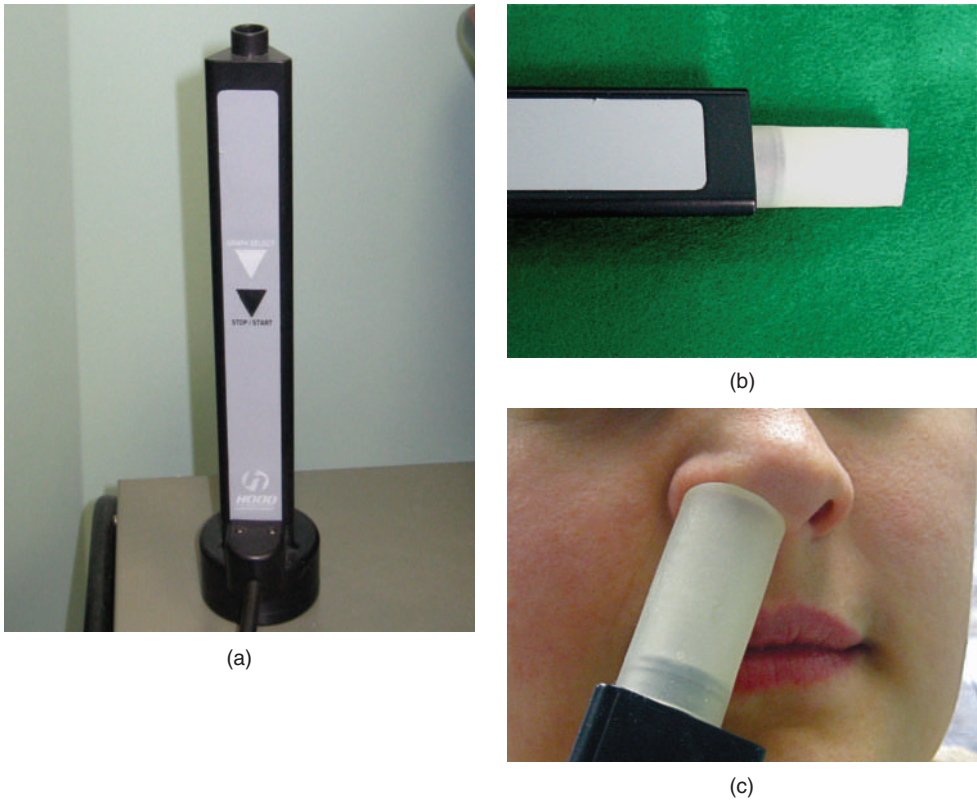


Figure 8.4 Rhinometer used to acoustically image the nasal airway: (a) view of the rhinometer tube that emits the sound wave, (b) close-up view of the nasal interface used to emit the sound wave into the nasal airway, and (c) rhinometer in use—note the tight seal of the nasal interface.

This finding may have some possible impact in the future to use this device as a screening tool as well, but further evaluation and research are warranted.

A companion device, the rhinometer, may be utilized to evaluate the nasal area from the external nasal valve to the posterior choanae (Figure 8.4). This modality does not evaluate the nasopharynx; therefore, the area from the posterior choanae to the superior aspect of the soft palate is not analyzed. To do this, adjunctive imaging would need to be ordered as deemed necessary.

Cephalometrics

Cephalometrics has been utilized for many years to assess a large number of craniomandibular osseous structures as well as to determine their impact on the airway as it relates to SRBD. The standards of practice on OAs

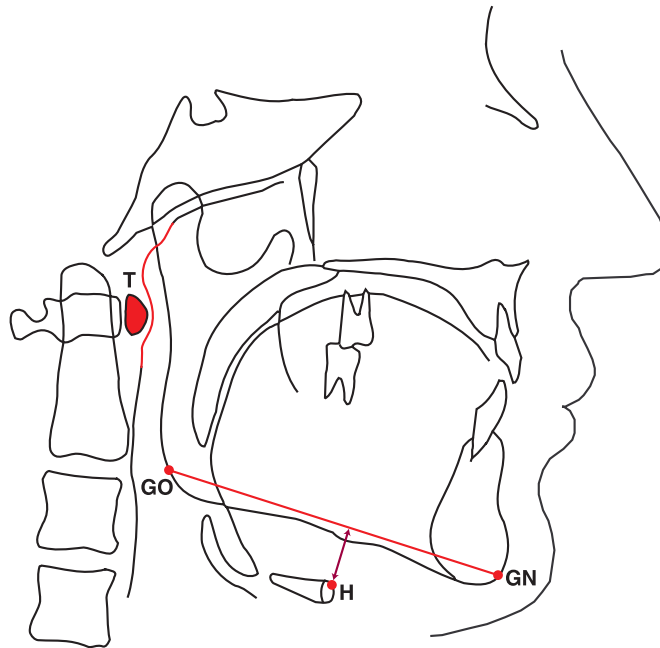


Figure 8.5 Cephalometric tracing to assess hyoid position. Note the perpendicular distance from the mandibular plane (Go-Gn) to the superior aspect of the hyoid (H). Note the anterior tubercle (T) of C-1 that is impacting the posterior aspect of the airway.

do not specifically recommend this imaging modality as part of the treatment, but the standards do indicate that cephalometrics can be used as an option if the clinician deems it necessary.¹⁰

At the current time, the use of cephalometrics seems to have a limited role. Historically, the airway has been visualized from an anteroposterior perspective to determine if it was compromised. However, this perspective was limited and it did not visualize the area in three dimensions.

In addition, a wide variety of classical cephalometric values have been reviewed to see if they can predict risk for SRBD. The one finding that has been repeatedly reliable as well as indicating a risk for a SRBD is hyoid position (Figure 8.5).^{11,12} Outcomes demonstrated that the more inferior the hyoid was relative to the mandibular plane and the larger the distance between the hyoid and the mandible, the greater the risk for SRBD.

Another study also found that the lowered position of the hyoid bone was significant as a predictor of OSA independent of obesity.¹³ In addition, the presence of a short mandibular corpus (body) was a relevant factor, and the study also demonstrated that a predisposition to OSA was associated with maxillary and mandibular retrognathism as well as a reduced facial height and deep bite.

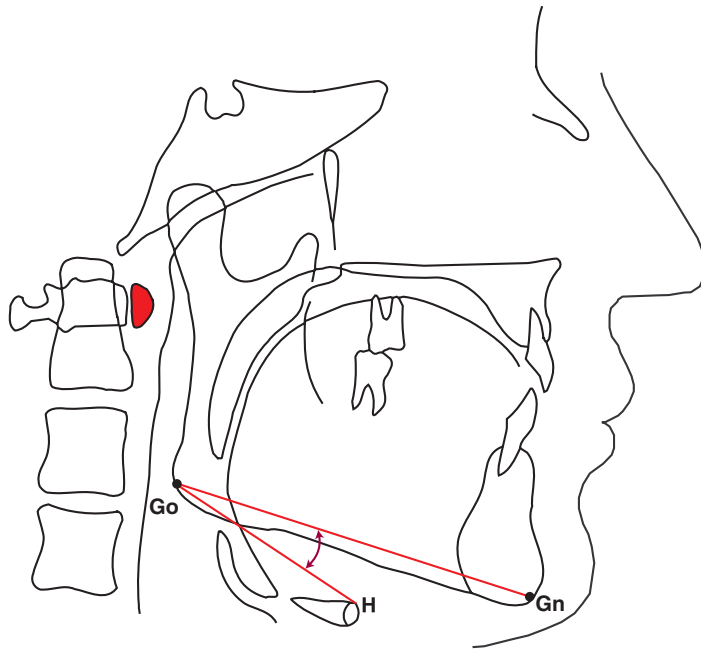


Figure 8.6 Cephalometric tracing to assess hyoid position. The angle from the mandibular plane (Go-Gn) to the superior aspect of the hyoid (H). As this angle increases, the hyoid moves down and posterior.

Another way of viewing this image is to look at the angle formed by the Go-Gn line to the hyoid (Figure 8.6). The greater this angle becomes, the lower and more inferior the hyoid is and hence the risk for OSA is increased.¹⁴ This finding is relevant particularly to those who order cephalometric X-rays on a regular basis for orthodontic procedures.

The observation of the hyoid position needs to be observed because of its correlation as a potential risk factor for SRBD, and this finding alone should stimulate the need for further and more detailed investigation.

Another cephalometric method that can be used is to assess the position of the hyoid bone by simply using a line drawn from the most inferior-anterior point of the third cervical vertebrae to the point menton (Me) of the mandible (Figure 8.7). As such, the hyoid should be on the line or above it. If the hyoid is below this line, then it is considered that the risk for OSA increases.

A study also looked at the tubercle of the first cervical vertebrae.¹¹ There may be instances when this tubercle is protruded forward, thus creating a narrowing of the airway in the pharynx from the posterior aspect. The clinician may need to consider this possibility on the basis of the assessment of the patient's posture relative to the cervical spine.¹⁵ As head posture

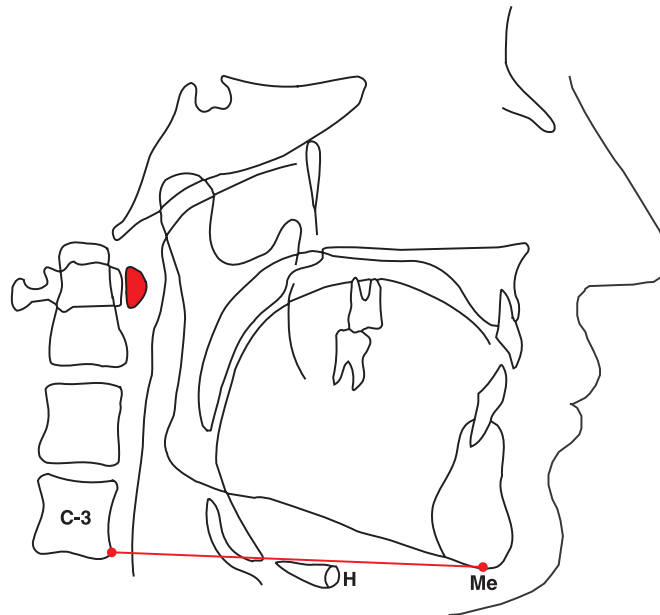


Figure 8.7 Cephalometric tracing to assess hyoid position, relative position of the hyoid (H), related to a line from menton (Me) to the inferior–anterior tip of the third cervical vertebrae (C-3).

changes, oftentimes in response to a compromised airway, the cervical spine is also impacted which may then impact the hyoid position.

Another possibility for compromise of the posterior pharyngeal wall may be the presence of osteophytes or small bony outgrowths associated with cervical spondylosis or a condition often termed ankylosing spondylosis (Figure 8.8).¹⁶ This study demonstrated the impact of osteophytes on the compression of the posterior aspect of the pharyngeal wall and the improvement following their surgical removal. The presence of the osteophytes frequently may be associated with symptoms such as difficulty swallowing, throat pain, shoulder pain, and headaches.

A study assessing head shape as a predictor for snoring resulted in the development of the craniofacial risk index (CRI).¹⁷ The CRI uses age, body mass index, and 14 cephalometric measurements as means of determining risk for snoring, and it was found to be effective 75% of the time.

Cone beam computed tomography

Cone beam CT, also known as cone beam volumetric imaging (CBVI), is an imaging technology that has the potential to assess the airway along with the craniofacial structures in a three-dimensional perspective (Figure 8.9). A study that used CBVI to review the airway configuration in subjects with



Figure 8.8 Osteophyte of the cervical spine and the impact on the airway from the posterior aspect.

OSA demonstrated the difference in airway volume and shape when comparing controls to individuals with OSA.¹⁸ The ability to evaluate the airway volume as well as the anteroposterior dimension was found to be useful in discerning individuals at risk for SRBD.

Because this is new technology, the full scope of its possible use has not yet been determined. With what is known to date, cone beam imaging has been termed “virtual laryngoscopy,” and a study indicated that a broad range of possibilities exists for this technology.¹⁹ For example, it is feasible to employ the Mueller maneuver during CBVI because of the 9-second scan time.

Comparison of cone beam imaging to cephalometrics also demonstrated a moderate degree of variability in the assessment of the upper airway volume and area.²⁰ This finding could be used to plan the therapy,²¹ and the cone beam modality may be a valuable tool in establishing or modifying mandibular position for OA therapy or in planning for surgical intervention.

Panoramic radiograph

The panoramic radiograph should be utilized in the management of SRBD, especially if an OA is considered as part of the management plan (Figure 8.10). This imaging modality should be used to evaluate the dental

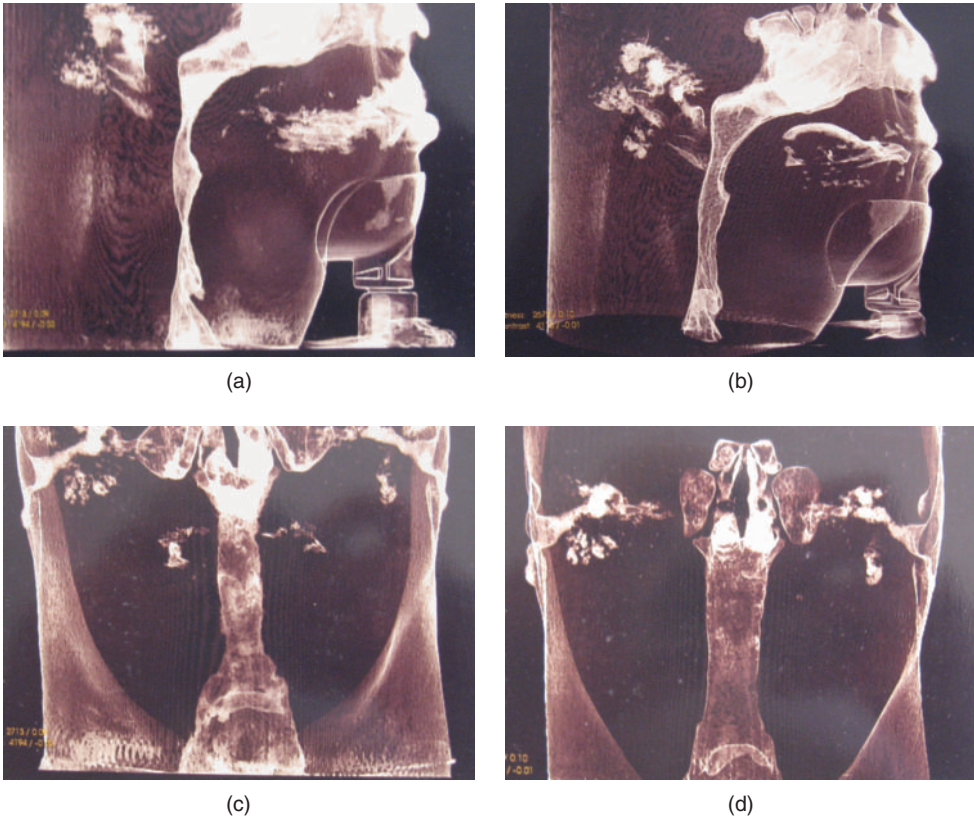


Figure 8.9 Cone beam images of a sleep apnea patient with and without the oral appliance in place; (a) and (b) are sagittal views and (c) and (d) are frontal views: (a) airway at normal jaw position, (b) airway with the mandible repositioned, (c) airway at normal jaw position, and (d) airway with the mandible repositioned.



Figure 8.10 Panoramic radiograph.

structures of the patient and to look for any pathology that may be present. It can also be helpful to screen for any gross temporomandibular joint (TMJ) changes, to evaluate the nasal airway structures (e.g., turbinates and nasal septum), and to assess the sinuses.

Tomography

Tomography, also known as tomograms, or other more advanced TMJ imaging modalities are not a standard of care for the SRBD patient, particularly if OA therapy is planned or being utilized. However, if temporomandibular disorder symptoms or signs are present, then additional more advanced and specific imaging may be indicated.²² Tomography may be performed if condylar displacement or arthritic changes are of concern and more detailed osseous imaging is needed. In addition, MRIs can be performed if a soft tissue problem is of concern, particularly if a disc displacement within the TMJ is suspected.

Imaging is not needed to purely assess TMJ position when the mandible is being repositioned with an OA for dental management of SRBD. It has been demonstrated that mandibular repositioning OAs do not precipitate TMJ problems with long-term use.²³ In addition, there does not appear to be any alteration in TMJ function.

CONCLUSION

The use of imaging relative to SRBD has the potential to aid in determining the risk for these disorders as well as to assist with the diagnosis and management, particularly with application to the use of an OA. However, imaging is not a replacement for sound clinical diagnosis, and it should be utilized as an adjunct in the diagnosis, treatment planning, and treatment progress.

In the future, the utilization of imaging will take on a more definitive role as additional research demonstrates the usefulness of the various technologies. Also, the cost effectiveness and the availability of conducting an imaging study may be of value to both the clinician and the patient as an aid to improved diagnosis and treatment outcome.

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Section 3

Management of the Sleep-Related Breathing Disorder Patient



Positive airway pressure therapy for sleep-related breathing disorders

CONCEPTUAL OVERVIEW

For patients with obstructive sleep apnea (OSA), snoring, or upper airway resistance syndrome (UARS), the primary type of therapy is positive airway pressure (PAP)¹⁻³ (Figure 9.1). However, a significant challenge to both patients and health care practitioners is compliance (acceptance/adherence) to PAP therapy.⁴

Upper airway patency is maintained by PAP through a mechanism involving the creation of a “pneumatic splint.” The PAP device produces a pressurized airflow that is delivered to the patient via a mask interface. This airflow subsequently creates a positive distension of the upper airway as well as changes in lung volume.

There are three primary types of PAP modes: (1) continuous positive airway pressure (CPAP), (2) bilevel positive airway pressure (BiPAP), 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.

INDICATIONS FOR PAP THERAPY

For SRBD that involve collapse of the oropharyngeal soft tissues or narrowing of the upper airway (Figures 9.2–9.4), such as what occurs with OSA,



Figure 9.1 PAP is the primary type of therapy for OSA and SRBD. (© ResMed, 2008. Used with permission).

PAP is effective and accepted as appropriate therapy. There are published parameters for the use of PAP in the management of OSA.¹

In essence, the objectives for managing OSA with PAP can include the following:⁵⁻¹³ (1) reduction of excessive daytime sleepiness (EDS) that is related to other comorbid issues, such as motor vehicle accidents, cognitive

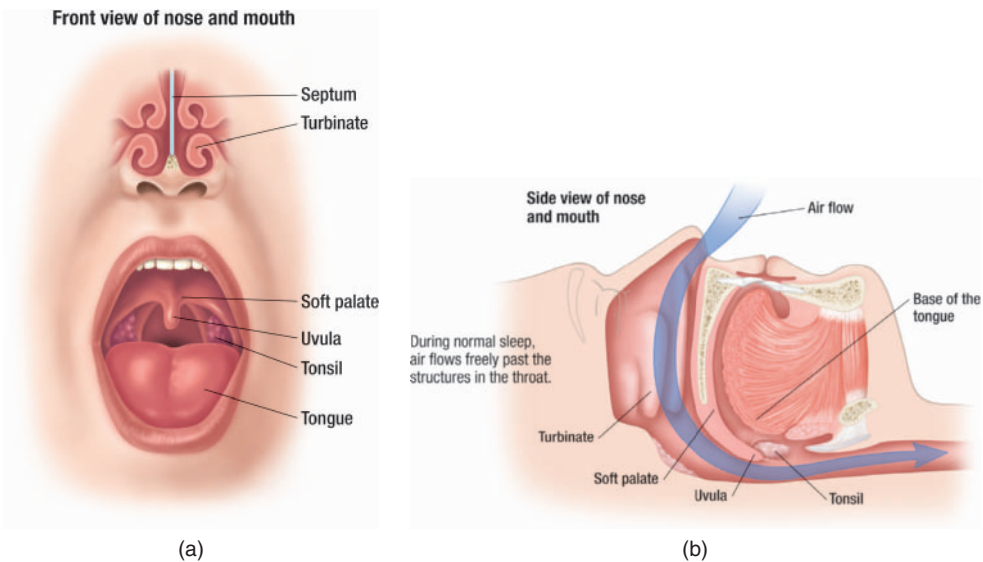


Figure 9.2 (a) Frontal view of normal oral and nasal airway. (© Krames Medical Publishing, 2009. Used with permission). (b) The normal upper airway allowing for an unobstructed pathway for breathing. (© Krames Medical Publishing, 2009. Used with permission).

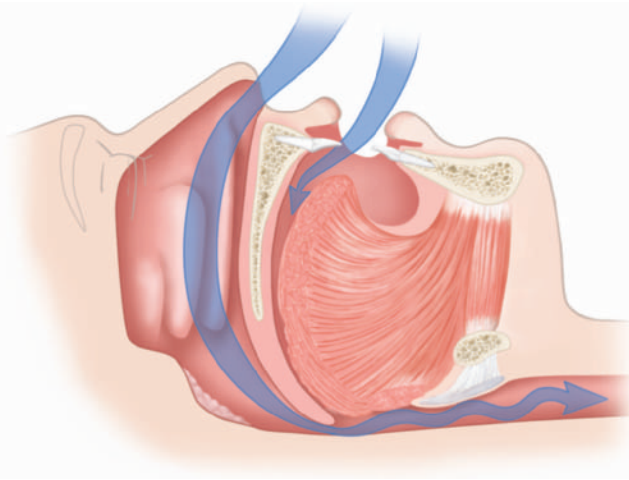


Figure 9.3 The partially obstructed upper airway that may result in snoring and/or hypopneic episodes. (© Krames Medical Publishing, 2009. Used with permission).

impairments, mood disorders; (2) prevention of cardiovascular sequela, such as high blood pressure; and (3) improvement of sleep for the bed partner, which usually involves the disturbing noises of snoring as well as the observation of apneic episodes.

PAP can be effective in obtaining these objectives and thereby improving the quality of life of individuals with OSA as well as the sleep of the bed partners.

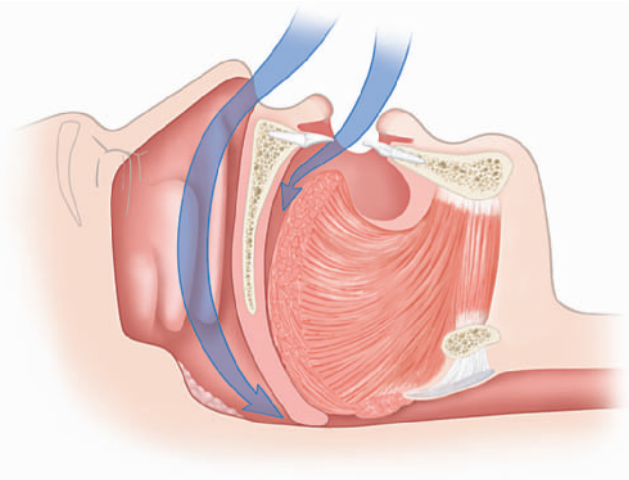


Figure 9.4 The completely obstructed upper airway that may result in apneic episodes. (© Krames Medical Publishing, 2009. Used with permission).

Besides effectiveness for management of moderate to severe OSA cases, PAP therapy may be effective for the management of mild OSA.¹ 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 hypertension (HT).^{10–14}

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 the use of an OA. The remaining possible conservative options include weight loss, sleep positional changes, sleep hygiene modifications, or other alternative therapies.

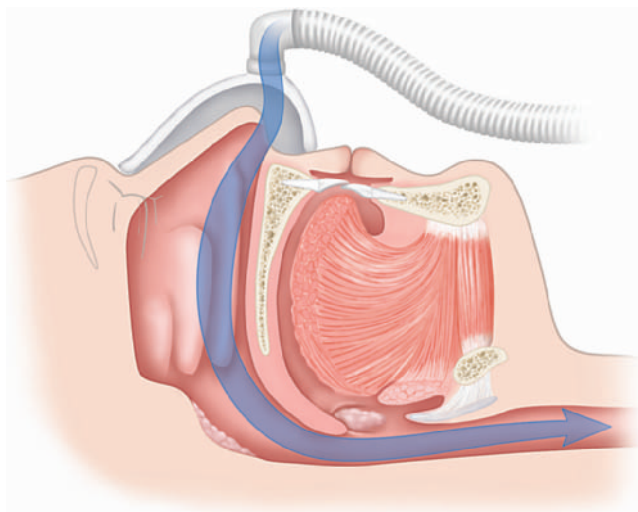
As with other areas of medical care, there are Medicare guidelines for PAP therapy reimbursement, which can serve as references for the indications of PAP therapy.¹⁵ If there is an apnea–hypopnea index (AHI) of 5–14 episodes/hour that represents mild OSA, then there must also be symptoms or signs of 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 polysomnogram (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 they may be performed during the latter portion of a single sleep study, also known as a split-night study.

MECHANISMS OF ACTION

The flow of pressurized air from the PAP device most often travels through the nasal passages and upper airway, past the soft palatal tissues and tongue, and down the lower airway to the lungs. This pressurized airflow creates an airway patency in the area of the nasal valve as well as in the oropharyngeal area by a combination of anterior displacement of the tongue base and soft palatal tissue along with distension of the lateral pharyngeal walls. In essence, the PAP functions as a “pneumatic splint” (Figure 9.5) that precludes the upper airway from collapsing during sleep.^{16,17}

Compared to atmospheric pressure, the intraluminal pressure from the PAP is positive, which results in a dilation of the upper airway. This increase in the cross-sectional area and volume of the upper airway has been demonstrated with computed axial tomography scanning as well as with magnetic resonance images (Figures 9.6 and 9.7).^{18–20} Subsequent to the opening of the airway by the “pneumatic splint,” there is a reduction of the upper airway muscle activity,^{21,22} thereby lowering muscle resistance.



A mask over the nose gently directs air into the throat to keep the airway open.

Figure 9.5 The effectiveness of PAP in preventing collapse of the upper airway, also referred to as a “pneumatic splint.” (© Krames Medical Publishing, 2009. Used with permission).

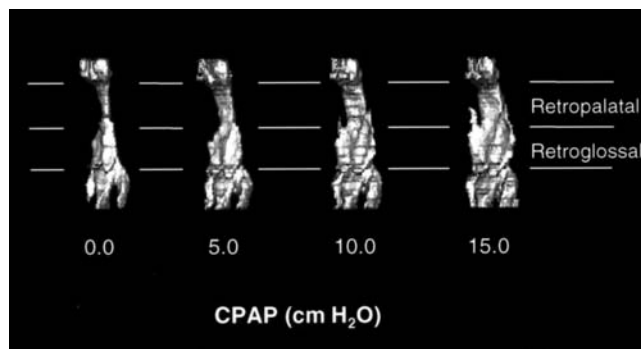


Figure 9.6 Three-dimensional surface renderings of the upper airway in a normal subject demonstrating increased upper airway volume in the retropalatal and retroglossal regions with progressively greater CPAP (0–15 cm H₂O). (Schwab R. Imaging for the snoring and sleep apnea patient. In: Attanasio R and Bailey D, eds. Sleep Disorders: Dentistry’s Role (Dental Clinics of North America; 45:4). Philadelphia: W.B. Saunders. 2001; 784. Reprinted with permission.)

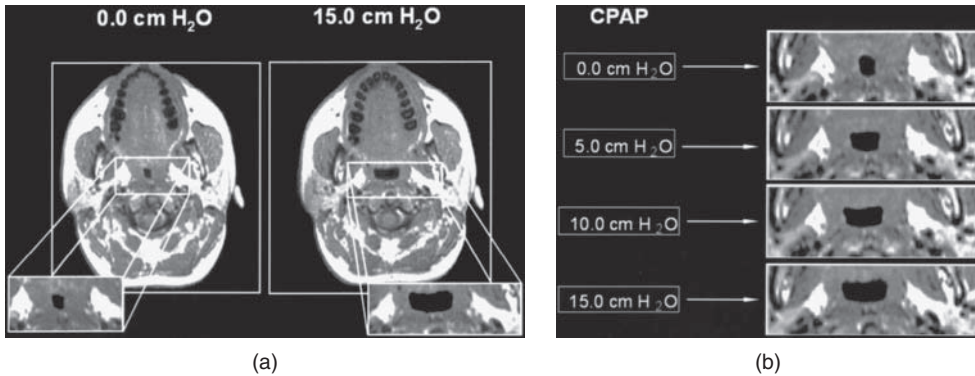


Figure 9.7 (a) Axial magnetic resonance image of the retropalatal region in a normal subject with CPAP of 0 cm H₂O and of 15 cm H₂O. (b) Axial magnetic resonance image with CPAP ranging from 0 to 15 cm H₂O. Progressive increases in CPAP result in airway enlargement. (Schwab R. Imaging for the snoring and sleep apnea patient. In: Attanasio R and Bailey D, eds. *Sleep Disorders: Dentistry's Role* [Dental Clinics of North America; 45:4]. Philadelphia: W.B. Saunders. 2001; 785. Reprinted with permission.)

PATIENT COMPLIANCE (ACCEPTANCE/ADHERENCE)

Patient compliance, also referred to as acceptance and adherence, of PAP therapy is regarded as willingness of the patient to use the PAP device at home for at least 1 week.⁴ Compliance is regarded as the patient using PAP therapy for more than 4 hours per night for more than 70% of the days.^{23,24}

Through the use of memory cards, modems, or wire transfer of data from the PAP device to a computer, there can be an assessment of patient compliance (Figures 9.8 and 9.9). It is important to obtain adherence data early after the onset of PAP therapy since PAP users who are adherent during the initial 1–3 months are more inclined to continue with PAP therapy.^{25,26}

However, in those individuals initially using PAP therapy, nonacceptance can vary from 5 to 50%, and by 3 years, an additional 12–25% no longer continue with PAP therapy.^{4,27,28} When an aggressive systematic follow-up program for PAP treatment is employed that includes appropriate mask interface selection and fitting, heated humidification, patient education, objective assessment of adherence, and early intervention for adverse side effects, PAP user compliance can significantly increase with results varying from 40 to 80%.^{23,29–34} These statistics are similar to adherence outcomes for chronic diseases managed by oral medications.

A poor fit of the mask interface, nasal symptoms (e.g., dryness, congestion, sore throat), and insufficient support from homecare providers



Figure 9.8 An example of a data memory card for a PAP unit (© ResMed, 2008. Used with permission).

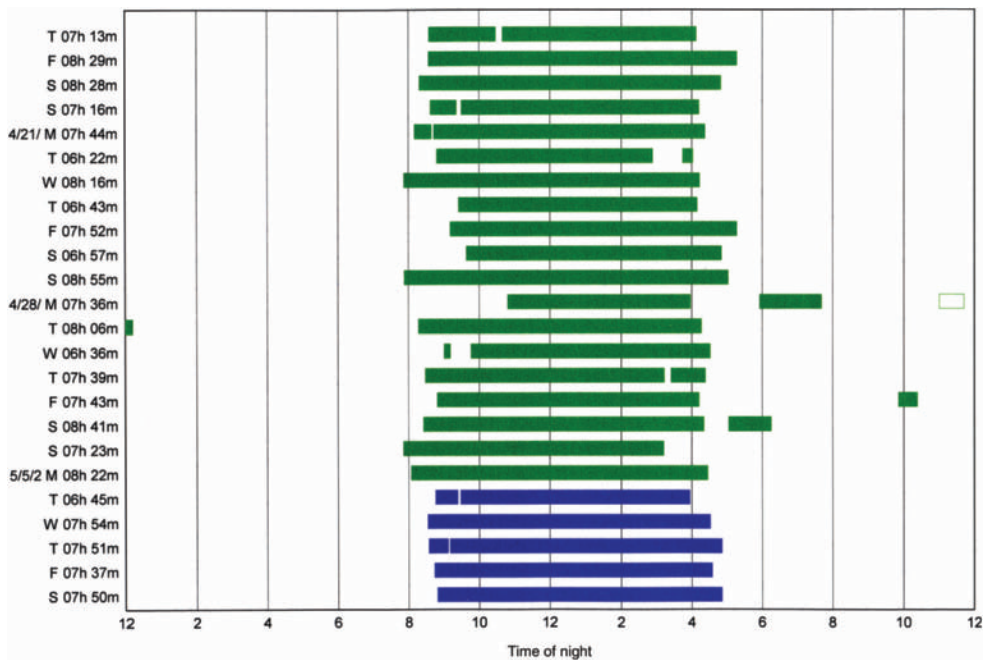


Figure 9.9 An example of a usage profile. The printout reflects nightly usage relative to start and stop times. (© ResMed, 2008. Used with permission).



Figure 9.10 An example of a PAP device without a humidifier (ResMed S8 Elite) (© ResMed, 2008. Used with permission).

(e.g., follow-up) are typically the most common contributory factors to non-adherence to PAP therapy.³⁵

TYPES OF PAP MODES

Although CPAP has been used since its inception in 1981, there have been technological advancements resulting in other delivery modes for PAP: BiPAP,³⁶ APAP,^{37–39} and, most recently, Flexible CPAP (Figures 9.10 and 9.11).⁴⁰

CPAP

The use of CPAP for the treatment of adults with OSA was first described in the literature in 1981.⁴¹

CPAP mode devices maintain a fixed or constant flow rate of air pressure during both inspiration and expiration. Through management that includes appropriate mask interface and possible use of airway humidification, many individuals find CPAP devices to be compatible for obtaining good sleep hygiene. Humidification can either be heated or at ambient



Figure 9.11 An example of a PAP device which incorporates a built-in heated humidifier (ResMed S8 Elite with H3i) (© ResMed, 2008. Used with permission).

room temperature, although PAP users typically prefer heated. CPAP devices are usually less expensive when compared to the other PAP modes.

APAP

APAP mode devices employ a sensor-based mechanism that automatically adjusts the flow rate of air pressure by continually monitoring the upper airway patency.^{37,38} Depending on the particular device, one or more respiratory factors are assessed, such as airflow limitations and impedance, which result in an air pressure that is constant during a respiratory cycle but variable during the course of a sleep period.^{42–45} Adherence of PAP therapy may be improved because of the allowance of a variable mean pressure during the sleep period (Figure 9.12).

Although the built-in algorithms will differ per manufacturer, the common objective is to analyze airflow and provide feedback to the device to allow for a pressure change. So as not to cause a waking arousal, these changes in airflow pressure are made on a gradual basis. The lowest and highest pressure limits of the device are formatted by the health care practitioner. The APAP device can then provide the pressure as needed per the varying respiratory requirements during the sleep period, which is

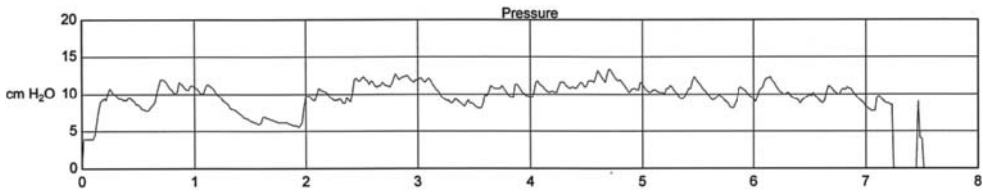


Figure 9.12 An example of PAP versus time for APAP treatment during a sleep period. The PAP unit autotitrated between a preset upper pressure limit of 20.0 cm H₂O and a lower pressure limit of 4.0 cm H₂O.

usually the lowest effective airflow pressure needed to maintain upper airway patency.

BiPAP

BiPAP mode devices allow for the provision of airflow pressures that reflect an inspiratory pressure higher than the expiratory pressure (Figure 9.13). Because some CPAP users find the continuous pressure during both inspiration and expiration to be uncomfortable, particularly the expiratory pressure, the BiPAP device's lower pressure during expiration can make

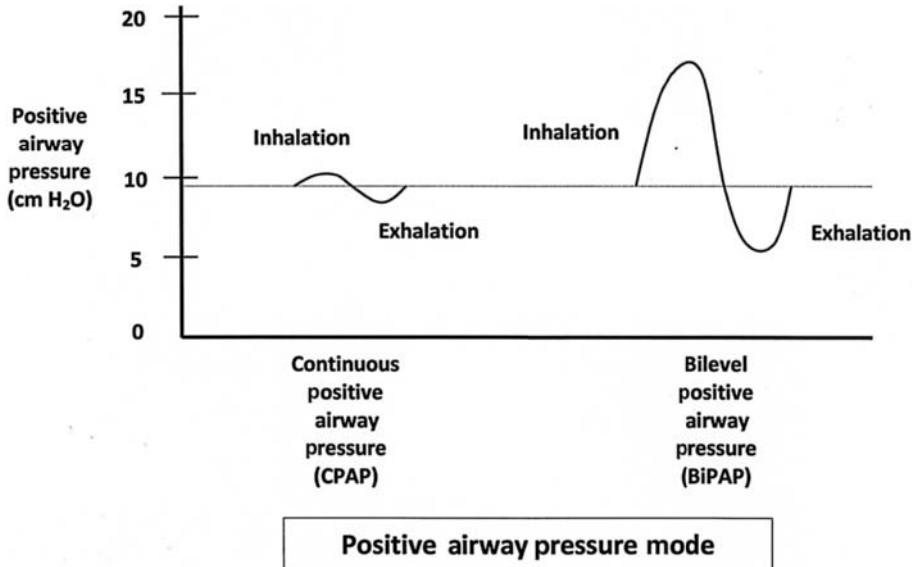


Figure 9.13 A comparison of inhalation and exhalation between CPAP and BiPAP regarding PAP. CPAP maintains a relatively steady positive pressure for both inhalation and exhalation. BiPAP provides a higher inhalation pressure and a lower exhalation pressure.

the device more tolerable to the user. A BiPAP device may lend to greater adherence for individuals who experience either aerophagia (i.e., forced ingestion of air into the stomach) or a reduced ventilatory drive.⁴⁶

Flexible CPAP

A recent technological advancement to further improve PAP therapy adherence has provided a CPAP variant that allows for a Flexible CPAP. By allowing the airflow pressure to go below the prescribed therapeutic airflow pressure in early expiration followed by a return of airflow pressure to the prescribed amount at the end of expiration, the Flexible CPAP mode can offer improved patient tolerance.

The C-Flex (Respironics, Murrysville, PA) is one model of this mode, and it allows for an alternating airflow pressure between inspiration and expiration on a breath-by-breath basis. During expiration, the device's airflow pressure decreases in proportion to the individual's expiratory airflow. By the end of the individual's respiratory expiration, the C-Flex's airflow pressure returns to the prescribed level of PAP.

A study demonstrated that adherence with the C-Flex was higher than with the traditional CPAP, although there were no significant differences between the two groups relative to subjective sleepiness or functional assessments.⁴⁰ Another study found no significant difference between the two groups relative to adherence and also found no significant differences relative to EDS and functional outcomes.⁴⁷

TYPES OF PAP MASK INTERFACES

Not only are the airflow pressure level and mode inherent to successful adherence with PAP therapy, but so is the challenge of selecting the optimal mask interface between the patient and the device. Mask interface issues can include discomfort from pressure of the mask against the nose or other facial areas, air leaks that can create noise or cause irritation of the eyes, and positional shifts of the mask that result from positional changes of the body during sleep.

As such, the development of various types of PAP mask interfaces by manufacturers has been robust. In addition, each type of mask interface is available in various sizes, typically small, medium, and large. Because the initial several weeks of PAP therapy are critical in establishing patient adherence, the American Academy of Sleep Medicine has recommended close follow-up evaluations to assess and manage issues arising from the PAP mask and/or device.²

Types of PAP mask interfaces (Figures 9.14–9.19) include the following: nasal mask, nasal pillows, full-face mask, oral-nasal mask, and oral mask. All PAP mask interfaces employ a varying type of headgear to maintain mask position. Sometimes it is necessary to also utilize a chin strap to



Figure 9.14 An example of a PAP nasal mask interface (ResMed Mirage Activa). (© ResMed, 2008. Used with permission.)

prevent opening of the mouth for those individuals who mouth breathe during sleep.

To obtain an optimal seal, there are typically two types of material utilized for the actual area of the mask that interfaces with the patient's face. One is a thin, flexible plastic sleeve that inflates from the airflow pressure and thus presses an "air cushion" against the contours of the face, and another is a soft, gel-type material that adapts to the contours of the face.

One of the more challenging areas to obtain a seal is the bridge of the nose. When there are air leaks in this area, it is common for there to be a subsequent irritation of one or both eyes.⁴⁸

The most common and traditional type of PAP mask interface is the nasal mask that fits over the entire nose, thereby creating a seal against the area above the upper lip but below the nares, against the cheek areas, and against the bridge of the nose.⁴⁹⁻⁵¹ For most PAP device users, opening of the mouth with subsequent mouth leaks is not an issue. The soft palatal tissues are pressed anteriorly against the posterior aspect of the tongue as a result of the positive airflow pressure in the nasopharynx.



Figure 9.15 An example of a PAP nasal mask interface being worn (ResMed Mirage Activa). (© ResMed, 2008. Used with permission.)



Figure 9.16 An example of a PAP nasal pillow interface system that utilizes interchangeable tube positions. (ResMed Swift II). (© ResMed, 2008. Used with permission.)



Figure 9.17 An example of a PAP nasal pillow interface system being worn (ResMed Swift II). (© ResMed, 2008. Used with permission.)

If a chin strap is not successful in maintaining mouth closure, if it is uncomfortable for the PAP user, or if there is an air leak causing eye irritation or a disturbing noise, then one of the other types of mask interfaces can be employed.

Another common type of PAP mask is the nasal pillows interface, which is composed of small nipple-type flexible rubber components that fit into



Figure 9.18 An example of a PAP full face mask interface. (ResMed UltraMirage Full Face). (© ResMed, 2008. Used with permission.)



Figure 9.19 An example of a PAP full face mask interface being worn. (ResMed UltraMirage Full Face). (© ResMed, 2008. Used with permission.)

the nares (Figures 9.20 and 9.21). The position of the pillows is maintained by a small plastic holder connected to a head strap similar to the one used for the nasal mask. The pillows are inflated with the PAP device's airflow pressure, thus creating a seal against the nares. As with the nasal mask, some users may need to also employ a chin strap if they are mouth breathers.

Persistent mouth breathers for whom the chin strap is unsuccessful may find the oral-nasal, full-face, or the oral device interfaces helpful. Not having a patent nasal airway (e.g., nasal congestion) may be another reason for considering the use of such type of mask interface alternatives.



Figure 9.20 An example of a PAP nasal pillow interface system with soft flexible pillows.



Figure 9.21 An example of a PAP nasal pillow interface system with soft flexible pillows being worn.

An oral-nasal mask or a full-face mask may be an appropriate alternative.⁵²⁻⁵⁴ Airflow pressure to the nasal and oral airways is applied simultaneously with the use of an oral-nasal mask. For patients with mouth air leaks and drying of the tissues in the upper airway, a full-face mask interface can be effective, although it can be challenging to obtain a good mask fit around all borders. With both these types of interfaces, there are valves or orifices built-in to the mask component that provides the ability for inspiration of ambient air in the circumstance of losing airflow pressure from a power source failure of the PAP device.

The availability of the oral mask interface allows the opportunity to bypass the nose completely (Figure 9.22).⁵⁵ Because of the airflow pressure



Figure 9.22 An example of a PAP oral appliance interface. (© OPAP—Aleksy Kozlov, 2009. Used with permission.)

against solely the oral tissues, it is usually necessary to provide heated humidification to minimize drying of those tissues. The oral mask can be uncomfortable to the user by virtue of its bulky design that incorporates flange components that fit behind and in front of the user's lips. Just as with the various nasal-involved type of masks, there can also be air leakage around the borders of the oral mask.

Most PAP devices employ the option to turn on a "mask-off" alarm that alerts the user of a significant air leak, which can occur from dislodgement of the mask interface from an adequate seal to the skin surface. Typically, the alarm awakens the PAP user and/or bed partner, and a simple readjusting of the mask interface resolves the leak and the alarm automatically ceases.

Each of the PAP mask interfaces needs the ability to clear out the user's exhaled CO₂. This is usually accomplished with the placement of a slit or orifice outlet either on the mask itself or at the junction of the mask and the hose that connects to the PAP device. This type of CO₂ outlet creates a necessary intentional leak.

From the aforementioned, it is obvious that mask fitting and comfort are critical factors and essential to enhanced patient adherence to PAP therapy. Typically, the prescribing sleep physician delegates this responsibility to either the respiratory therapist at the durable medical equipment company that will be providing the PAP device or the sleep technologist at the sleep laboratory that may provide the PAP unit.

DETERMINATION OF PAP PRESSURE LEVELS

At either the split-night or two-night sleep study attended by the sleep technologist, the mask interface type is selected, and it then becomes necessary to establish the therapeutic level of airflow pressure from the PAP device.^{1,56} This determination of effective PAP level is called PAP titration.

During the PAP titration portion of the sleep study, the sleep technologist typically uses remote control to incrementally increase the airflow pressure to determine levels when there is resolution of respiratory events, such as apneas, hypopneas, snoring, oxygen desaturations, and respiratory effort-related arousals. The effective PAP titration should normalize airway flow in the supine position as well as in rapid eye movement sleep. Protocols for PAP titration have been established.⁵⁷

The attended PSG, or sleep study, provides an opportunity for a sleep technologist to account for patient discomfort, mask air leaks, mask position shifts, changes in body positions, stages of sleep, and other technical aspects such as the loss of PSG leads.^{29,57-60} For example, it has been demonstrated that the therapeutic PAP pressure level was significantly higher in the supine position than in the lateral position in most individuals who were diagnosed with OSA.⁶⁰ Also, a study found that the therapeutic PAP pressure level was decreased with elevation of the bed's head

portion,⁵⁹ and another study demonstrated that weight loss also allowed for a decrease of airflow pressure.⁶¹

The sleep physician subsequently reviews the recorded results of the PAP titration portion of the sleep study compared to the results from the non-PAP baseline portion of the sleep study. A therapeutic level of PAP is established and prescribed, and the patient is provided with a PAP device programmed to that therapeutic level of pressurized air. Pressure levels for effective PAP therapy can vary, but they are typically between 4 and 20 cm H₂O.

BENEFITS OF PAP

Successful PAP therapy for the management of OSA can result in physical benefits for the user. It is well documented that PAP therapy will improve both EDS and quality of life.^{3,62-67} In fact, one study found that some subjects diagnosed with OSA demonstrated improvements in EDS following only one sleep period of PAP therapy.⁶⁸

By precluding apneic and hyponeic events, PAP therapy has been shown to be of benefit in reducing blood pressure in hypertensive patients who have sleep apnea,⁶⁹ decreasing nocturnal blood pressures,⁷⁰⁻⁷² and reducing diurnal blood pressures.^{11,12} Also demonstrated in studies has been the improvement of neuropsychologic functioning with PAP therapy even in individuals diagnosed with mild OSA.^{73,74}

As such, sleep quality is improved through the use of PAP therapy. Individuals diagnosed with sleep-disordered breathing who were users of PAP on a consistent basis showed improvement in EDS, motor vehicle accidents, memory loss, concentration deficiencies, mood disorders, and cognitive function outcomes.⁷⁵⁻⁷⁷

ADVERSE SIDE EFFECTS OF PAP

There are adverse side effects associated with PAP therapy, and, if not addressed, these adverse side effects will decrease acceptance and adherence of PAP therapy.⁷⁸

Adverse side effects of PAP therapy are common. A listing of the most common include dry nasal passages, rhinorrhea, and congestion, all of which can affect from 25 to 65% of PAP users.⁷⁹ These particular PAP users often also have had nasal symptoms prior to the use of PAP therapy. Other user complaints can include claustrophobia, irritation of the eyes, difficulty breathing the pressurized airflow, sore skin areas over the bridge of the nose (Figures 9.23 and 9.24) or the upper lip related to the mask interface, and even noise generated by the PAP device.



Figure 9.23 Common pressure areas of mask interface systems that may result in skin irritation on the bridge of the nose and upper lip areas. (© ResMed, 2008. Used with permission.)

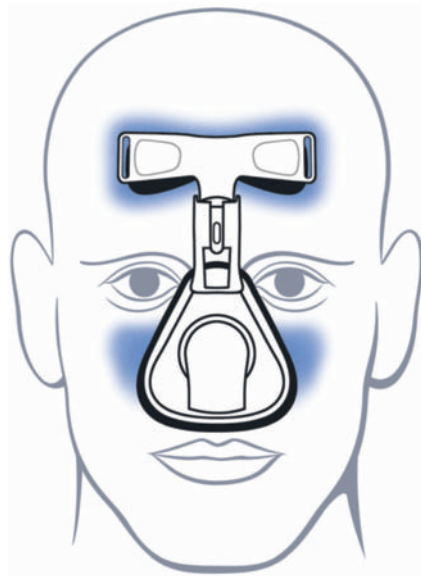


Figure 9.24 Areas of resistance built into some mask interface systems, thus reducing pressure on sensitive areas. (ResMed Mirage system). (© ResMed, 2008. Used with permission.)

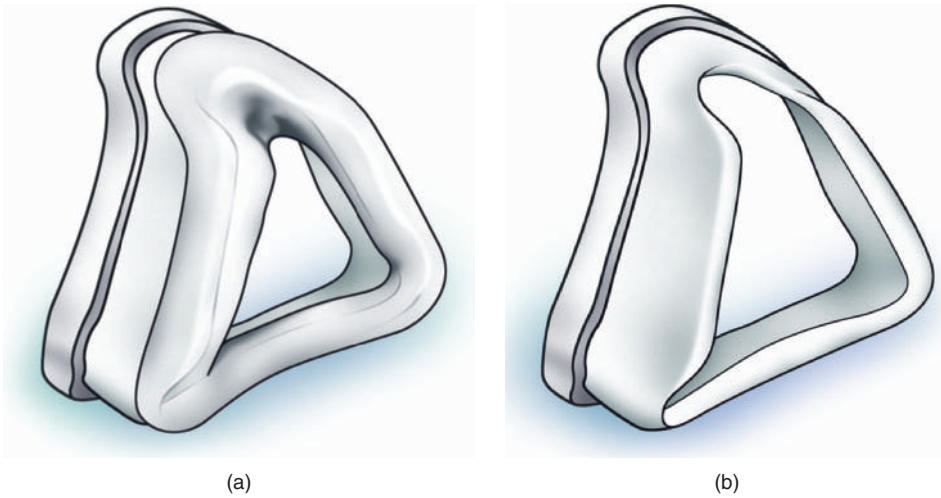


Figure 9.25 An example of a dual-wall cushion technology designed to provide support and stability for a wide fit range and additional dispersion of weight and pressure, limiting contact of the cushion with the most sensitive areas of the face. (a) Outer lining of the dual-wall cushion and (b) inner lining of the dual-wall cushion. (ResMed Mirage system). (© ResMed, 2008. Used with permission.)

Many of the adverse side effects are mask interface related.⁸⁰ Despite the ability that PAP devices can take into account for air leaks at the mask interface (Figures 9.25–9.27), significant leaks that can occur with mouth opening may preclude the PAP device from producing ample air-flow pressure to maintain airway patency.⁸¹ The end results of these large



Figure 9.26 Cutaway shows how air pressure flows into the outer cushion of a dual-wall mask interface to enhance the seal, thus reducing the need for excessive head strap tension. (ResMed Mirage system). (© ResMed, 2008. Used with permission.)



Figure 9.27 An example of an adjustable forehead support portion of a mask interface system that reduces pressure on a common sensitive area of the skin. (ResMed Mirage system). (© ResMed, 2008. Used with permission.)

air leaks can include dryness of the air passages as well as waking arousals, thereby counteracting the effectiveness of PAP therapy in managing OSA.

Sleep position of the head against a pillow while wearing a PAP mask may lead to discomfort from the pressure of the pillow against the mask or it may also lead to air leak from dislodgement of the mask by the pillow. Use of a specialized pillow, such as the PAPillow[®], allows for the designed contours of the pillow's edge to allow the mask to extend beyond without pressure interference (Figure 9.28).

Nasal congestion is also common, and heated humidification of the PAP device's airflow may improve this situation. At times, it may be necessary to also treat the congestion with nasal steroid spray, nondrowsy antihistamines, or decongestants that the individual can use prior to sleep. A full-face mask or oral mask may also be considered,^{52–54} but another alternative is surgical treatment if a nasal obstruction, such as a narrow nasal passage, can be identified as a contributing factor.^{55,82}

Sometimes a PAP user will complain of difficulty breathing with the device's airflow pressure levels, but this is usually more common with CPAP than with BiPAP, APAP, or Flexible CPAP devices. Also, all that may be necessary is an adaptation period for the user to start the sleep period with an airflow pressure that is lower than the therapeutic level, and then the programmed PAP unit gradually increases the pressure over time as the patient accommodates. This is typically referred to as “ramp time.”



Figure 9.28 (a) CPAP user with a standard bed pillow. (© PAPillow, 2009. Used with permission). (b) CPAP user with a PAPillow. (© PAPillow, 2009. Used with permission.)

Noise complaints used to be more common with the older PAP devices. Most contemporary devices are significantly quieter. The noise that generally bothers either the PAP user or the bed partner is the sound created by the intentional leak of the PAP mask component as air is emitted from the orifice located in the mask or at the junction of the mask and the air tube that connects to the PAP unit. However, typically the bed partner will prefer such device-generated noise than the noise generated from the individual's snoring.

OTHER CONSIDERATIONS

Humidification

PAP devices can be fitted with either a cold passover (ambient temperature) humidifier or a heated humidifier. The initial PAP devices only had the cold passover humidification as an option. Subsequent generations of PAP devices incorporated heated humidification that could deliver more moisture than the cold passover version.

Since the mucosa of the nasal airway can dry and become tender from the passage of cold air, resistance in the nasal airway can increase without the use of any humidification. Studies have demonstrated that this nasal airway resistance caused by PAP therapy without humidification can result in significant air leak at the mask interface area from mouth opening, which in turn may cause further nasal airway resistance.^{35,83} This may result in a cycle of nasal airway resistance, mask interface leaking, mouth breathing, increased nasal airway resistance, and worsening of the OSA. As a result of the decreased efficacy of the PAP therapy and decrease in patient comfort, PAP user compliance may be reduced.

With cold (passive) humidification, the PAP device generates air pressure that subsequently passes through a container that is partially filled with water. This mechanism allows for the air to absorb moisture as it passes over the surface area of the water in the container.

Heated humidification employs the use of a hot plate in the container of water, and the PAP device typically allows the user to modify the amount of heated humidification through a temperature control. As a result, the pressurized air has the advantage of being both warm and humid.

For the most part, PAP users tend to agree that heated humidification is more comforting to the nasal passages than is cold (passive) humidification. Both cold passover and heated humidification modalities result in greater PAP user satisfaction relative to no humidity. However, there were less adverse side effects with heated humidification when compared to cold passover humidification.^{29,30}

Because an increase in infectious complications may be associated with PAP humidification,⁸⁴ it is recommended that the PAP user clean all the components involved (i.e., humidifier, hose, and mask) on a regular basis per the manufacturer's instructions.

Ramp

Most PAP devices offer an optional "ramp" feature that allows the PAP user the opportunity to set a "ramp time," which is a period of gradual increase in airflow pressure from a low starting level to eventually reaching the prescribed therapeutic level. This period of time allows the patient to fall asleep when the airflow pressure is lower, thereby increasing the acclimation and comfort, and thus the successful adherence to PAP therapy.

Traveling with CPAP

PAP devices need to be protected from damage during travel. PAP users who derive benefits from their PAP device should carry their devices in a carry-on bag, whether in automobile transit or air travel. Most airport security screening personnel are familiar with PAP devices, and they are allowed as a carry-on item. Nonetheless, it is recommended that the PAP user still obtain and carry a letter/prescription from their health care practitioner that the PAP device is of medical necessity.

Most PAP devices have features to account for variable voltage sources, especially for international travel, and the devices may also have a built-in compensatory mechanism for altitude adjustments. It has been shown that PAP devices at higher altitudes will provide less airflow pressure as a result of the decreased air density.⁸⁵ In these circumstances, PAP users may consider wearing an OA as a substitute treatment during travel.

Sleep apnea in the perioperative period

There is a greater risk of complications from general anesthesia relative to individuals with SRBD. As such, the disorder should be diagnosed and managed prior to the use of general anesthesia. In addition, these same individuals must be closely monitored postoperatively to preclude any SRBD problems. It is not uncommon for individuals with SRBD to bring and use their PAP devices at the hospital during the postoperative period.

CONCLUSION

PAP is the primary type of therapy for patients with OSA, snoring, and UARS. There are three primary types of PAP modes: CPAP, BiPAP, and APAP. Flexible CPAP is a more recent development as a possible fourth PAP mode.

PAP creates a “pneumatic splint” that maintains upper airway patency, thereby improving the continuity and quality of sleep as well as the overall quality of life for individuals diagnosed with OSA. Studies have demonstrated the effectiveness of PAP for the management of OSA.

The significant challenge to successful PAP therapy, however, is adherence (compliance) since individuals can experience adverse side effects as well as difficulties tolerating the airflow pressure. PAP therapy is usually effective if used on a regular basis.

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10

Surgical treatment for sleep-related breathing disorders

CONCEPTUAL OVERVIEW

The *International Classification of Sleep Disorders-2 (ICSD-2)* regards sleep-related breathing disorders (SRBD) as being “characterized by disordered respiration during sleep.”¹ A subcategory of SRBD is obstructive sleep apnea (OSA). In order to effectively treat OSA, it is imperative to assess which of the upper airway structures are contributory to the diagnosed disorder.

The upper airway can be regarded as being composed of three anatomical zones² (Figures 10.1 and 10.2):

1. The nasopharynx (i.e., the nasal turbinates to the hard palate)
2. The oropharynx that includes both the retropalatal area (i.e., the hard palate to the caudal margin of the soft palate) and the retroglossal area (i.e., the caudal margin of the soft palate to the epiglottis)
3. The hypopharynx (i.e., the base of the tongue to the larynx)

Although obstruction of the upper airway may include multiple anatomical sites, closure of the upper airway is most commonly located in the retropalatal and retroglossal areas (Figures 10.3–10.5).² Vibration of the airflow past the pharyngeal soft tissues can result in snoring, and a narrowing or collapse of the upper airway may result in obstructive respiratory disturbances during sleep, such as hypopnea or apnea. Surgery of the upper airway may be considered as a management option, especially for patients for whom treatment involving positive airway pressure (PAP) therapy or oral appliance (OA) therapy is not effective or tolerable.

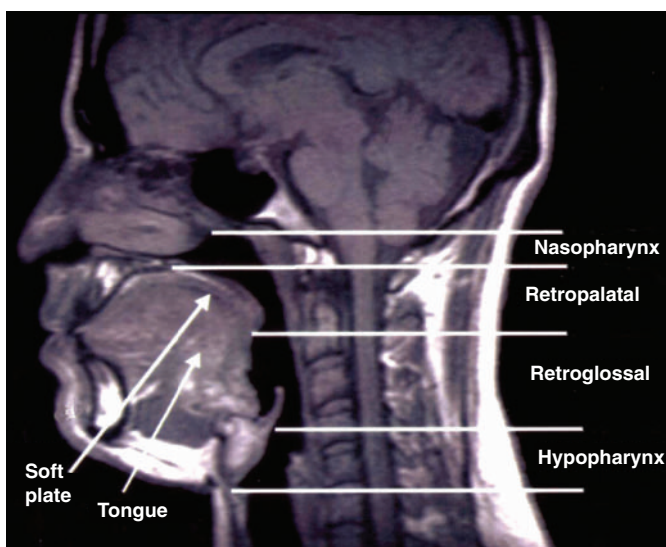


Figure 10.1 Midsagittal MRI of a normal subject. It indicates the regions of the upper airway. (Schwab R. Imaging for the snoring and sleep apnea patient. In: Attanasio R and Bailey D, eds. Sleep Disorders: Dentistry's Role (Dental Clinics of North America, 45:4). Philadelphia: W.B. Saunders. 2001; 761. Reprinted with permission.)

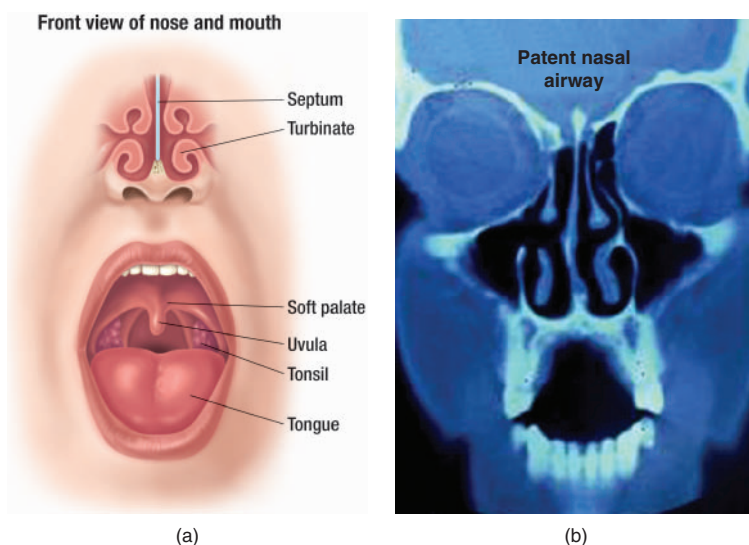


Figure 10.2 (a) Normal frontal view of the upper airway. (© Krames Medical Publishing, 2009. Used with permission.) (b) Coronal CT scan of a normal airway with clear paranasal sinuses, relatively straight septum, and normal turbinates, resulting in a patent nasal airway. (Aragon S. Surgical management for snoring and sleep apnea. In: Attanasio R and Bailey D, eds. Sleep Disorders: Dentistry's Role (Dental Clinics of North America, 45:4). Philadelphia: W.B. Saunders. 2001; 868. Reprinted with permission.)

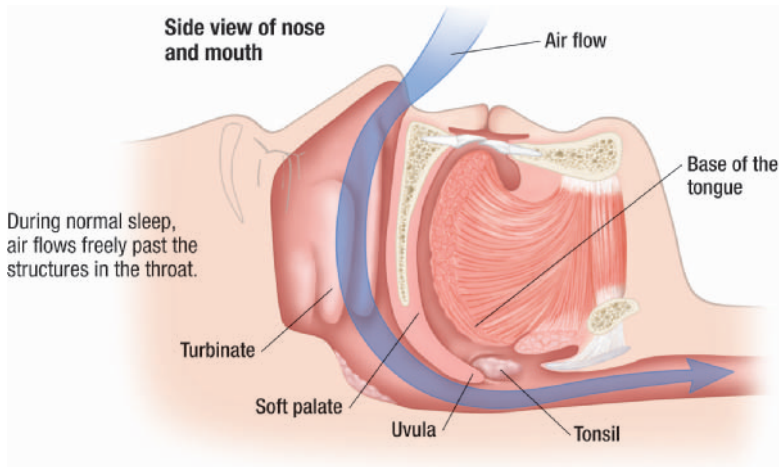


Figure 10.3 Normal sagittal view of the upper airway. (© Krames Medical Publishing, 2009. Used with permission).

The goal of this chapter is to provide an overview of the more common types of surgical intervention techniques that are employed for the management of snoring and OSA.

INDICATIONS FOR SURGICAL INTERVENTION

The purpose of surgical intervention is to improve airway patency during sleep by enlarging the upper airway and correction of any disproportionate anatomy.

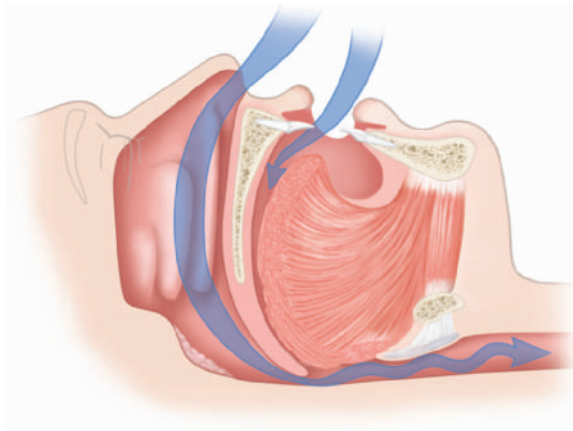


Figure 10.4 Sagittal view of partially blocked upper airway. (© Krames Medical Publishing, 2009. Used with permission.)

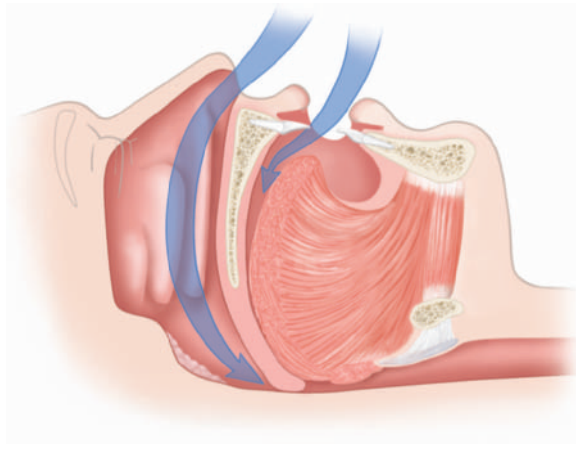


Figure 10.5 Sagittal view of completely blocked upper airway. (© Krames Medical Publishing, 2009. Used with permission.)

Surgery is indicated (1) after unsuccessful nonsurgical conservative therapeutic interventions such as PAP, behavioral approaches (e.g., weight loss, sleep hygiene), and OA; or (2) when an abnormal soft tissue structure is identified as being responsible for the OSA.³

It has been shown that only a very small percentage of patients with OSA have a readily identifiable anatomical area of narrowing or collapse that contributes to the upper airway resistance or collapse.⁴ Most OSA patients present with a diffuse soft tissue blockage or disproportionate anatomical component of the upper airway that is not always readily observable,⁴ and thus warrants a complete evaluation.

PRESURGICAL EVALUATION

The purpose of the presurgical evaluation is to identify site-specific areas in the upper airway that contribute to or cause the OSA. These findings become part of the process of determining which surgical approach, if any, is warranted to address the anatomical obstruction.

Because different areas of the upper airway may be involved in the etiology of the SRBD, a complete patient evaluation is necessary to (1) assess the probable areas of anatomical involvement in airway narrowing or collapse and (2) determine if any existing pathologies such as neoplasms or cysts are present. Most common clinical findings associated with OSA are disproportionate anatomy such as a large or elongated soft palate (Figure 10.6), a thickened or elongated uvula (Figure 10.7), an enlarged tongue particularly at the base (Figure 10.8), a deviated nasal septum (Figures 10.9 and 10.10), nasal turbinates that are hypertrophic (Figures 10.10 and 10.11),



Figure 10.6 Elongated palatal tissue.

presence of nasal polyps (Figure 10.12), and a hypoplastic or retrognathic mandible.

Ideally included in the evaluation is a polysomnogram (PSG) to help determine the extent of the breathing disorder, a comprehensive history, and a comprehensive head and neck clinical examination that includes a nasopharyngolaryngeal examination through the use of a flexible fiberoptic endoscope. The findings from the clinical examination can also be complemented by imaging studies (e.g., cephalometrics, magnetic resonance imaging (MRI), computed tomography (CT) scan).⁵

The outcomes of an ideal presurgical clinical examination would serve as predictors for the locations of any narrowed or collapsed areas of the upper airway. Unfortunately, because of its subjective nature, clinical



Figure 10.7 Elongated uvula.



Figure 10.8 Enlarged tongue.

examination in and of itself is insufficient for complete assessment of the upper airway in identification of SRBD.⁶ In addition, clinical examinations, including the use of fiberoptic endoscopy, are generally conducted when the patient is not in a true sleep state.

Although the literature does not provide a definitive means of establishing a predictive value regarding which patients would benefit from all surgical techniques,⁷⁻⁹ there have been two classification systems typically used during clinical examinations with respect to pharyngeal anatomy. The

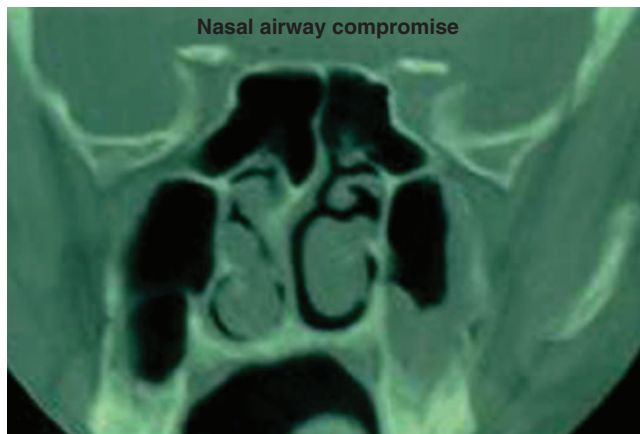


Figure 10.9 Coronal CT scan of an impaired nasal airway because of pansinusitis leading to purulent rhinosinusitis. (Aragon S. Surgical management for snoring and sleep apnea. In: Attanasio R and Bailey D, eds. *Sleep Disorders: Dentistry's Role* (Dental Clinics of North America, 45:4). Philadelphia: W.B. Saunders. 2001; 869. Reprinted with permission.)

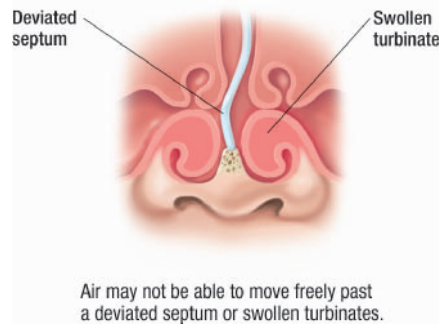


Figure 10.10 Frontal view of the upper airway demonstrating a deviated septum and swollen turbinates. (© Krames Medical Publishing, 2009. Used with permission.)

first, known as the Mallampati classification, was based on palate position, and it was used as a prognostic indicator relative to the potential difficulties of tracheal intubation.¹⁰ Two other clinical staging systems for SRBD were subsequently developed, known as the Friedman classifications, related to tongue position and tonsil size with respect to the palate^{11,12} (Figures 10.13 and 10.14). The Friedman tongue position classification has been investigated as a prognostic indicator for the presence and severity of obstruction in the hypopharynx region.¹³

Incorporating a classification with respect to position of the tongue, tonsils, and palate into a complete head and neck examination is warranted

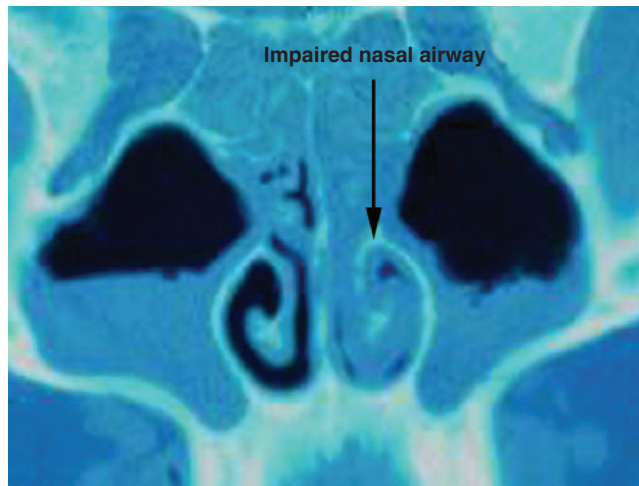


Figure 10.11 Coronal CT scan of a compromised nasal airway because of marked septal deviation, turbinate hypertrophy, and maxillary sinusitis. (Aragon S. Surgical management for snoring and sleep apnea. In: Attanasio R and Bailey D, eds. *Sleep Disorders: Dentistry's Role* (Dental Clinics of North America, 45:4). Philadelphia: W.B. Saunders. 2001; 869. Reprinted with permission.)

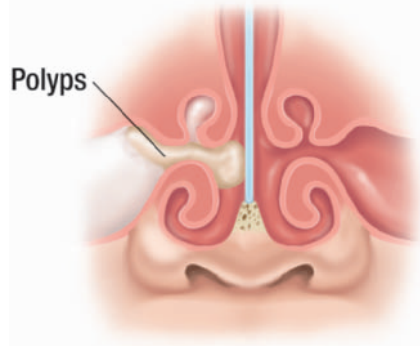


Figure 10.12 Frontal view of the upper airway demonstrating polyps. (© Krames Medical Publishing, 2009. Used with permission.)

for any patient prior to undergoing any surgical intervention for snoring and OSA since the findings may direct the surgeon in the selection and site of surgical treatment.

TYPES OF SURGERY

On the basis of the outcomes of the presurgical evaluation, surgical treatment is site-specific. There are a variety of surgical procedures available, ranging from minimally invasive to more invasive. The purpose of the minimally invasive procedures is to obtain relatively small changes in the structures and biomechanics of the upper airway, whereas the more invasive procedures are meant to achieve significant removal of tissues and sometimes also a repositioning of anatomical structures (e.g., advancement of the mandible and/or maxilla).

Tracheostomy

Tracheostomy was the first surgical procedure introduced for the treatment of OSA.¹⁴ However, since the employment of PAP therapy and other surgical procedures, tracheostomy is no longer the standard surgical treatment for OSA.¹⁵ Also, when compared with PAP therapy and the other surgical procedures, tracheostomy is regarded to have adverse psychological, social, and esthetic effects.

This procedure completely bypasses any obstruction in the upper airway because it is performed distal to both the larynx and the pharynx. As such, this procedure has been demonstrated to result in significant improvement of OSA.¹⁶

Despite the aforementioned adverse effects, tracheostomy is still indicated when (1) the severe OSA is life-threatening from an immediate

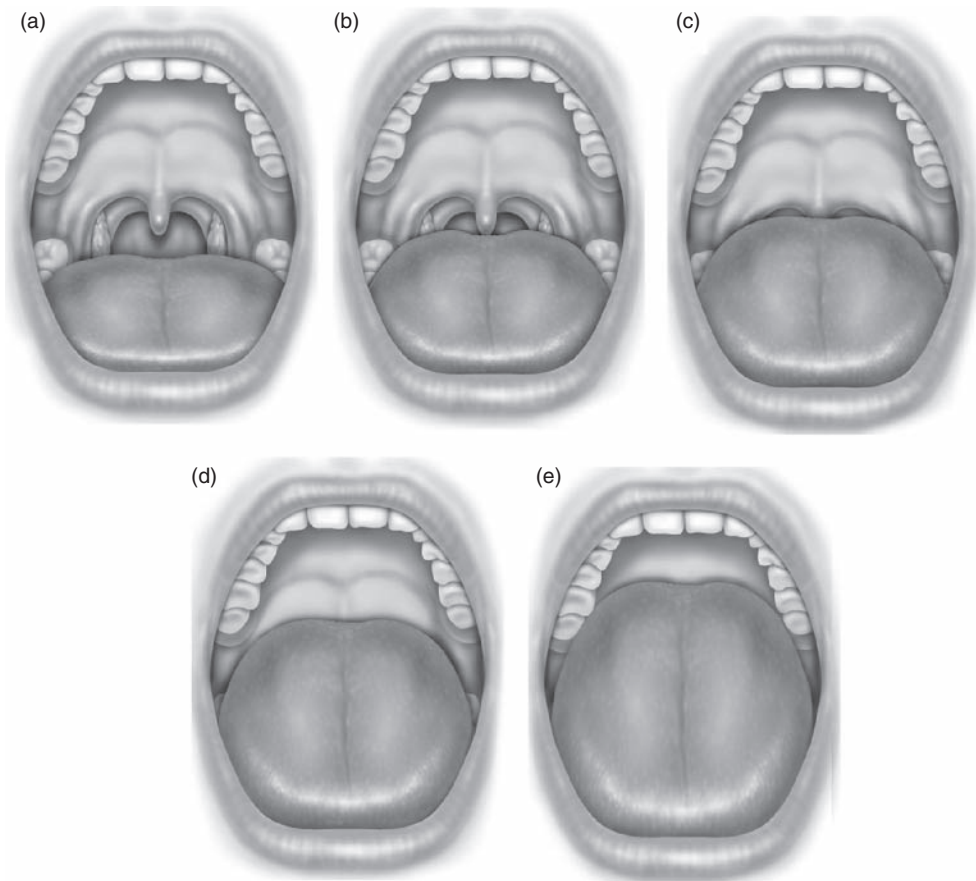


Figure 10.13 Friedman tongue position classification. (a) FTP I allows visualization of the entire uvula and tonsils/pillars. (b) FTP IIa allows visualization of most of the uvula, but the tonsils/pillars are absent. (c) FTP IIb allows visualization of the entire soft palate to the base of the uvula. (d) FTP III allows visualization of some of the soft palate, but the distal structures are absent. (e) FTP IV allows visualization of the hard palate only. (Friedman M, Soans R, Gurpinar B, et al. Interexaminer agreement of Friedman tongue positions for staging of obstructive sleep apnea/hypopnea syndrome. *Otolaryngol Head Neck Surg.* 2008; 139:373. Reprinted with permission.)

obstruction or associated with significant comorbidity, (2) PAP therapy is either not tolerated or it does not address the severe deoxygenation, and (3) airway control is needed during upper airway reconstruction.

Nasal surgery

Even though narrowing and collapse of the upper airway occur mostly in the retropalatal and retroglottal areas, the nasal portion of the upper

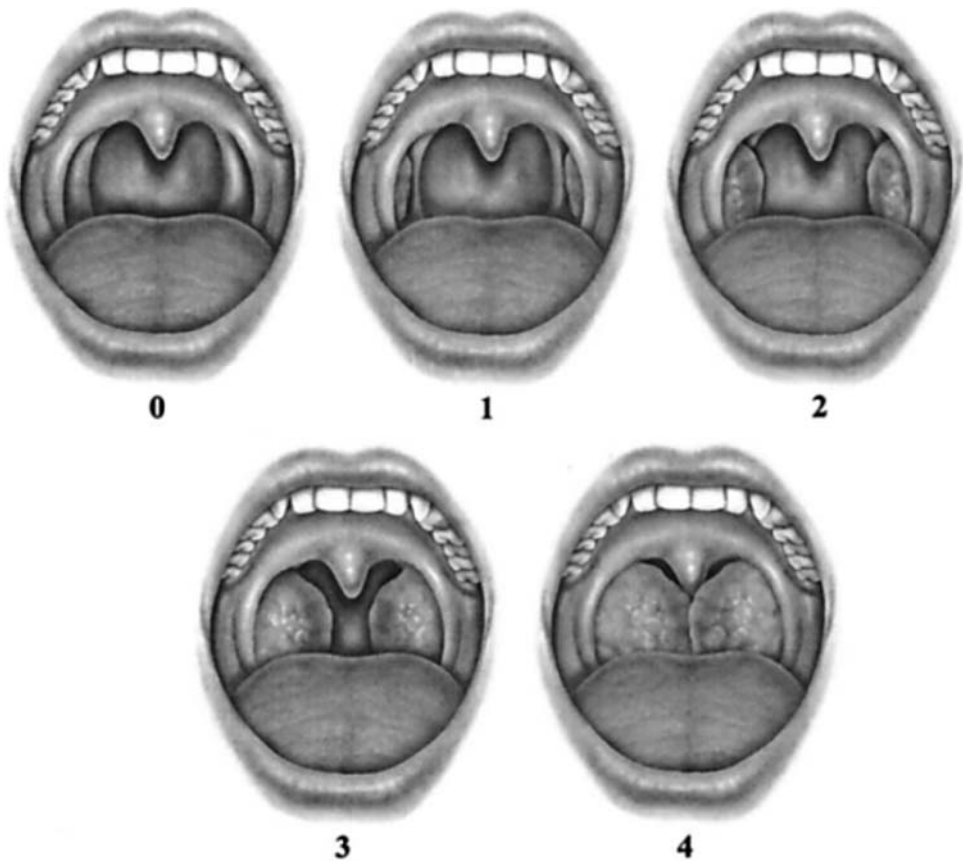


Figure 10.14 Tonsil size is graded from 0 to 4. Tonsil size 0 denotes surgically removed tonsils. Size 1 implies tonsils hidden within the pillars. Tonsil size 2 implies the tonsils extending to the pillars. Size 3 tonsils are beyond the pillars but not to the midline. Tonsil size 4 implies tonsils extended to the midline. (Friedman M, Ibrahim H, Lee G, et al. Combined uvulopalatopharyngoplasty and radiofrequency tongue base reduction for treatment of obstructive sleep apnea/hypopnea syndrome. *Otolaryngol Head Neck Surg.* 2003; 129(6):614. Reprinted with permission.)

airway can have a significant effect on these sites. One of the functions of the nasal airway is to allow for a level of resistance in the upper airway. If there is an increase in the level of resistance in the nasal airway, there can be a related increase in pharyngeal negative pressure that may contribute to the narrowing and even obstruction of the airway in the velopharyngeal and hypopharyngeal areas.⁵

Impaired nasal breathing can result in individuals who are sensitized from exposure to high allergens or in individuals suffering from rhinitis. This experience of impaired nasal breathing during sleep can result in poor sleep quality and excessive daytime sleepiness. It is not uncommon

during sleep for nasal congestion to cause an individual to mouth breathe, and therefore rotating the mandible down and back. When this occurs, the base of the tongue can narrow the airway space in the pharynx. Thus, nasal airflow resistance could have an impact on the severity of SRBD.^{17,18}

Investigations have demonstrated that sleep quality worsened with experimental nasal occlusion.^{19–21} Individuals are at greater risk for developing SRBD if they have rhinitis and nasal congestion during sleep,^{22,23} severe septal deviation or inferior turbinate hypertrophy,^{24,25} and nasal polyposis.²⁶

The primary purpose of nasal surgery is to improve the patency of the nasal airway which can subsequently improve SRBD.^{24,27–29} However, the rate of success for nasal surgery is regarded as low when success is defined as a $\geq 50\%$ reduction of AHI (apnea–hypopnea index) and < 20 AHI score. One study demonstrated a worsening of the OSA by a surgical correction procedure of a deviated septum.³⁰ At times, the purpose of the nasal surgery may only be to improve the compliance of using PAP by providing the patient with a clear nasal airway. Thus, the indications for nasal surgery include obstruction of airflow from deviation of the septum, enlarged turbinates, nasal polyps, or narrowing of the nasal valve.

The more common nasal surgeries include septoplasty, turbinoplasty, turbinatectomy, polypectomy, adenoidectomy, and enlargement of the nasal valve.

Tonsillectomy

It is not uncommon for hypertrophic palatine tonsillar tissues to be responsible for obstruction in the posterior pharyngeal region of the airway. Using the definition of surgical success as $> 50\%$ reduction of AHI and < 20 AHI score, tonsillectomy has been demonstrated to be effective in 80% of adults with severe OSA and 100% effective in adults with mild OSA.³¹ Although tonsillectomy and adenoidectomy are primarily performed in children, tonsillectomy is regarded as an accepted treatment modality for adults with OSA.³²

In an effort to lessen the degree of postoperative discomfort that can accompany the surgical or electrical cautery excision of tonsillar tissue, tissue ablation through the use of temperature-controlled radio frequency has been shown to be effective.^{33,34}

Uvulopalatopharyngoplasty

The most common surgical procedure for increasing the oropharyngeal airway space is the uvulopalatopharyngoplasty (UPPP). This procedure was initially described in 1964 as a surgical means of addressing snoring, and it was later modified to also address OSA.^{1,35,36} UPPP involves reduction of tissues in the areas of the soft palate, uvula, and the lateral and posterior

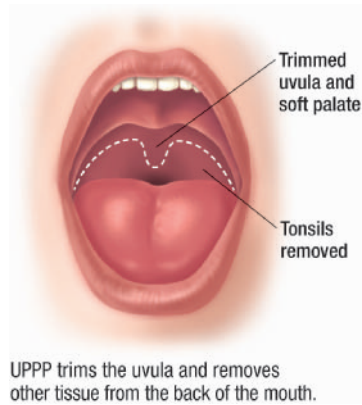


Figure 10.15 Depiction of UPPP surgical procedure. (© Krames Medical Publishing, 2009. Used with permission.)

walls of the pharynx (Figures 10.15–10.17).³⁷ It is common for a tonsillectomy to be performed at the same time as the UPPP.

The degree of removal of soft tissues can vary and is dependent on the site-specific issues of each patient. The more radical UPPP procedure does not necessarily equate to more effectiveness, but it can lead to increased complications. The more common postoperative complications can include surgery-associated pain, infection, velopharyngeal insufficiency, speech disturbance, and nasopharyngeal stenosis. Nonetheless, UPPP is regarded as a safe surgical procedure.³⁸

As with the other surgical procedures, the criteria for success include a $\geq 50\%$ reduction of the AHI and a < 20 AHI score. The success rate for treatment of OSA can range from 25 to 75%.^{39–44} The more the obstruction



Figure 10.16 Pre-UPPP: elongated palatal tissue.



Figure 10.17 Post-UPPP.

is limited to the retropalatal area, the greater the success rate. The most common reason for reduction in the success rate is the presence of anatomical obstruction in the retroglottal or hypopharyngeal area, for example the base of the tongue⁴⁵ or other tissues.⁴⁶ One study demonstrated that the Friedman classifications for palate position and tonsil size were a valuable prognostic indicator for UPPP successful outcomes.¹²

Tongue-base suspension

Even though OSA patients often have multiple sites of obstruction or narrowing of the airway,³⁶ the most common anatomical areas with obstruction are the base of the tongue and the retroglottal areas.⁴⁷ When the base of the tongue may be identified as the primary site of obstruction, a tongue-base suspension (TBS) surgical procedure can be indicated (Figure 10.18).

To help counterbalance the collapse of the base of the tongue in the hypopharynx, a permanent suture is looped through the base of the tongue and attached to a titanium screw that has been placed in the geniotubercle portion of the mandible. The tension of the suture decreases the collapsibility of the base of the tongue against the posterior wall of the pharynx during sleep.^{47,48} This procedure may result in a "tongue base dimple due to the suspension suture"⁴⁹ (Figure 10.19).

There is a low success rate of 20% with this procedure.⁵⁰ However, when TBS is combined with UPPP, the success rate significantly increases to above 80% in patients with severe OSA.⁴⁹

Surgical reduction of the tongue

A more radical surgical procedure for an enlarged base of the tongue occluding the retroglottal area is the surgical reduction of the posterior aspect

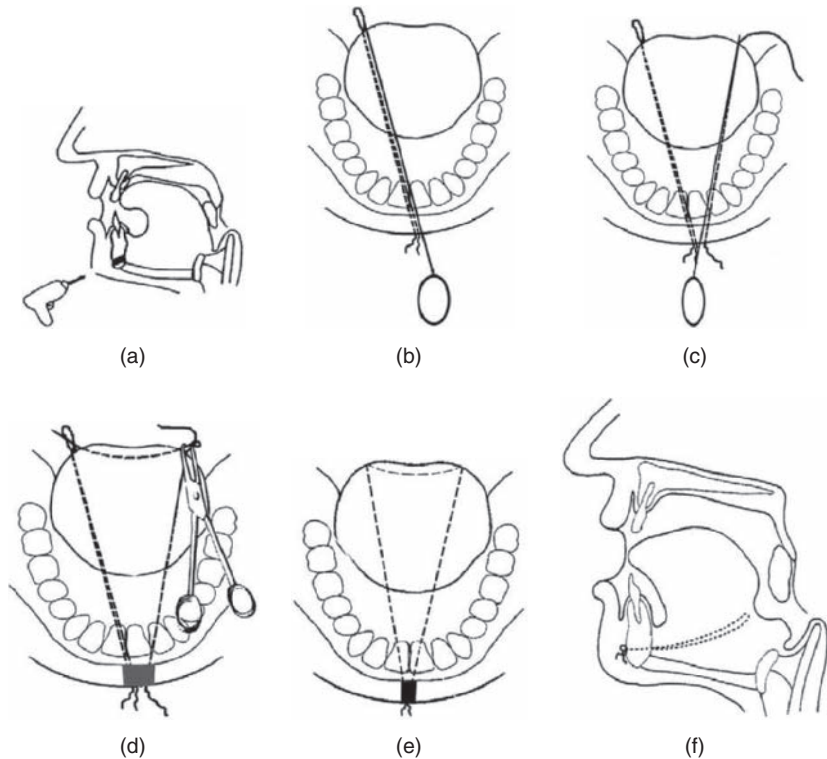


Figure 10.18 Schematic illustrations of the operative procedure. (a) Submental incision and the creation of the small hole on the mandible. (b) Passing the silk suture loop to the tongue base with a suture passer. (c) Passing the suture. (d) Passing the suture across the tongue. (e) and (f) Tying the suture on the mandibular hole. (Omur M, Ozturan D, Elez F, et al. Tongue base suspension combined with UPPP in severe OSA patients. *Otolaryngol Head Neck Surg.* 2005; 133:220. Reprinted with permission.)

of the tongue. This can be accomplished with a midline glossectomy that excises a rectangular portion of the tongue.⁵¹ Another technique is the linguloplasty that is more aggressive in the amount of tissue removal with the postsurgical defect, resulting in an anterior advancement of the base of the tongue. This latter procedure can significantly improve the success rate to 75%.⁵²

Because of the significant edema at the surgical site, both surgical procedures necessitate the use of a tracheotomy. Postoperative complications can include difficulty swallowing, disturbed taste, and bleeding.

Tissue ablation with radio frequency

Radio frequency (RF) for the ablation or removal of soft tissue has been used for the management of a hypertrophic prostrate and other body

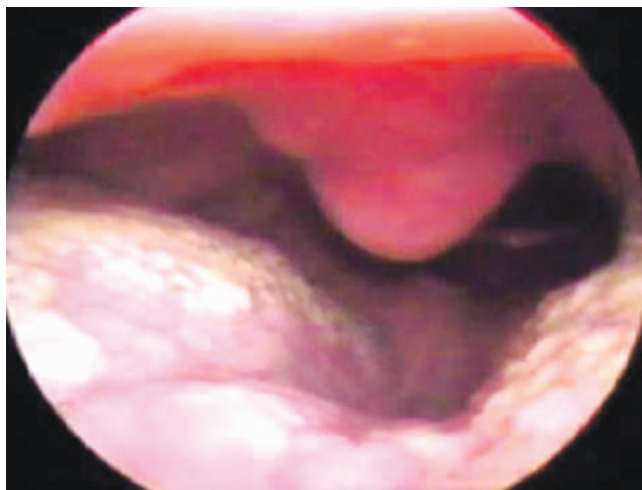


Figure 10.19 The tongue base dimple because of the suspension suture. (Omur M, Ozturan D, Elez F, et al. Tongue base suspension combined with UPPP in severe OSA patients. *Otolaryngol Head Neck Surg.* 2005; 133:220. Reprinted with permission.)

areas. An RF generator produces a low heat energy that can be used to denature tissue protein in the area surrounding the electrode probe without any destruction of surface tissue. RF has been demonstrated in animal studies to be effective in the reduction or ablation of excess or redundant tongue tissue.⁵³

As with the other surgical procedures, the success of RF is dependent on the site-specific diagnosis of the tissue obstruction or narrowing in the airway. Palatoplasty with RF therapy reduced snoring in 60–100% of the patients.^{54–57} Interestingly, it has been demonstrated that there may be an 11–41% potential for relapse of snoring 12–18 months following initial RF ablation.^{53,58} When there is an unsuccessful response to RF of the retropalatal area, potential airway obstructions in the nasal and/or hypopharyngeal areas should be considered.

RF ablation can also be applied to the base of the tongue. In one study of OSA patients for whom UPPP was not successful, the AHI was significantly reduced following RF ablation of the tongue base.⁵⁹ Another study of OSA patients who had multisite obstructions of the airway demonstrated that ablation of soft palatal tissue and the base of the tongue with RF significantly reduced the AHI.⁶⁰

Complications of RF ablation can include ulceration, infection, dysfunction of the soft palatal tissues, and velopharyngeal insufficiency.

A more recent technique employs a submucosal minimally invasive lingual excision (SMILE) in which the RF wand is placed within the tongue base to remove tissue inside as compared to the outer surface of the tongue

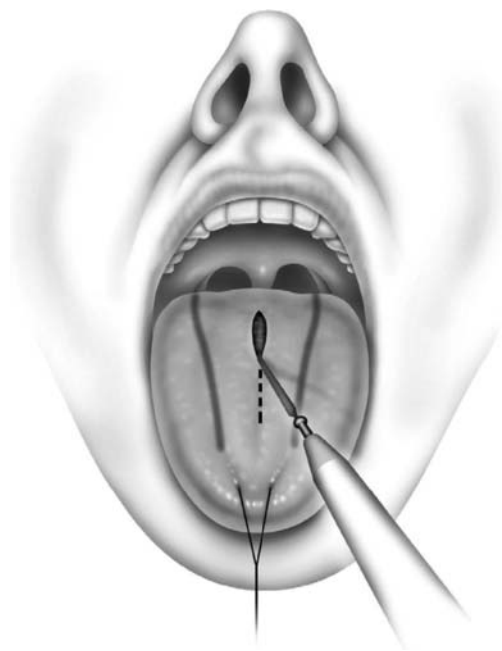


Figure 10.20 Midline incision for the SMILE technique. Note the marked course of the lingual arteries on either side of the incision. (Friedman M, Soans R, Gurpinar B, et al. Evaluation of submucosal minimally invasive lingual excision technique for treatment of obstructive sleep apnea/hypopnea syndrome. *Otolaryngol Head Neck Surg.* 2008; 139:379. Reprinted with permission.)

with the traditional RF approach (Figures 10.20 and 10.21). The results of studies demonstrate significantly increased success rates.^{61,62}

Mandibular osteotomy with genioglossus advancement

Attached to the geniotubercle area of the mandible is the genioglossus muscle of the tongue. A mandibular osteotomy with genioglossus advancement (MOGA) is a surgical procedure that involves the sectioning of a rectangular block of bone surrounding the geniotubercle and advancing that section anterior to the labial surface of the mandible (Figures 10.22–10.25). The outcome is a tension applied to the tongue, which prevents a collapse of the posterior aspect of the tongue into the retroglottal airway during sleep. This technique does not involve any movement of teeth or maxillo-mandibular osseous structures other than the conservative portion of the geniotubercle.

Expansion of the airway resulting from this procedure has been shown to have a 42–75% success rate.^{50,63,64}

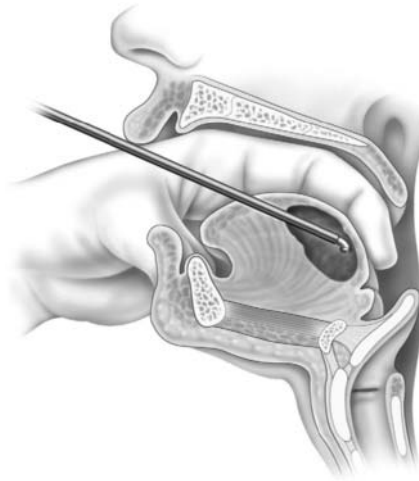


Figure 10.21 SMILE technique: coblation wand is introduced through the incision and advanced toward the vallecula. Submucosal tissue excision is guided by palpation with the nondominant hand. (Friedman M, Soans R, Gurpinar B, et al. Evaluation of submucosal minimally invasive lingual excision technique for treatment of obstructive sleep apnea/hypopnea syndrome. *Otolaryngol Head Neck Surg.* 2008; 139:380. Reprinted with permission.)

Hyoid myotomy and suspension

Another surgical procedure that results in an expansion of the retroglottal and hypopharyngeal areas is the hyoid myotomy and suspension (HMS). This is particularly effective when there is hypopharyngeal constriction

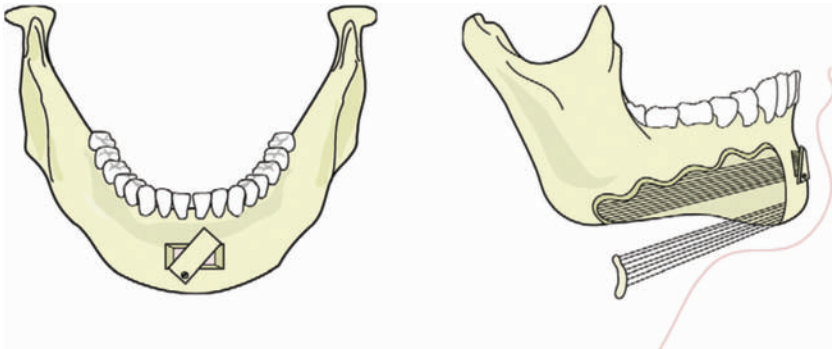


Figure 10.22 The genioglossus advancement procedure. A rectangular window of symphyseal bone consisting of the geniotubercle is advanced anteriorly, rotated to allow bony overlap, and immobilized with a titanium screw. (Li KK. Hypopharyngeal airway surgery. *Otolaryngol Clin North Am.* 2007; 40:848. Reprinted with permission.)



Figure 10.23 Intraoperative view showing the extent of the rectangular osteotomy. (Li KK, Riley RW, Powell NB, et al. Obstructive sleep apnea surgery: genioglossus advancement revisited. *J Oral Maxillofac Surg.* 2001; 59:1183. Reprinted with permission.)

from lateral pharyngeal tissue that is excessive or from an epiglottis that is retrodisplaced.

The procedure involves suspending the hyoid bone over the ala portion of the thyroid via nonresorbable sutures, which should preclude a hypopharyngeal collapse or narrowing since there will be increased tension on the muscles of the tongue (Figure 10.26).

The success rate for this procedure has been shown to vary from 17 to 65%.^{46,65-67}

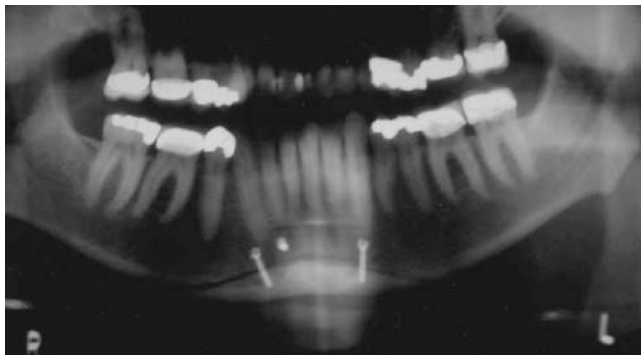


Figure 10.24 Panoramic radiograph showing the reconstituted border fixed with two screws. Note the genial tubercle/genioglossus muscle complex (GGC) bone flap is stabilized with a single screw. (Li KK, Riley RW, Powell NB, et al. Obstructive sleep apnea surgery: genioglossus advancement revisited. *J Oral Maxillofac Surg.* 2001; 59:1184. Reprinted with permission.)

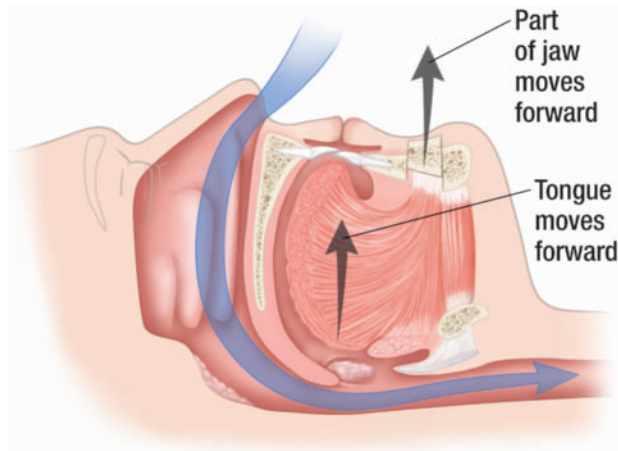


Figure 10.25 Depiction of genioglossus advancement surgical procedure. (© Krames Medical Publishing, 2009. Used with permission.)

Maxillomandibular advancement

A more aggressive surgical procedure involving repositioning of the osseous maxillomandibular components is maxillomandibular advancement (MMA). Although tracheostomy is considered to be the most effective treatment for OSA, MMA is regarded as the next most effective because the procedure involves the retropalatal and retroglottal areas as well as increasing the tension of the genioglossal muscle, thereby significantly improving the airway space⁶⁸ (Figure 10.27). Success rate for MMA is 75–100%.^{46,58,69,70}

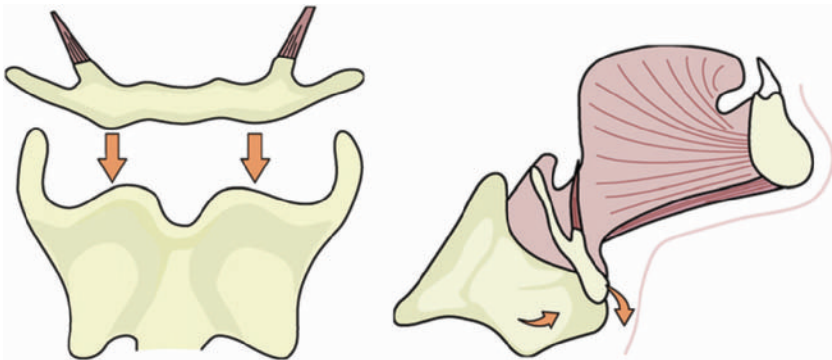


Figure 10.26 The hyoid advancement procedure. The hyoid bone is isolated, the inferior body dissected clean, the majority of the suprahyoid musculature remains intact. The hyoid is advanced over the thyroid lamina and immobilized with sutures placed through the superior aspect of the thyroid cartilage. (Li KK. Hypopharyngeal airway surgery. *Otolaryngol Clin North Am.* 2007; 40:848. Reprinted with permission.)

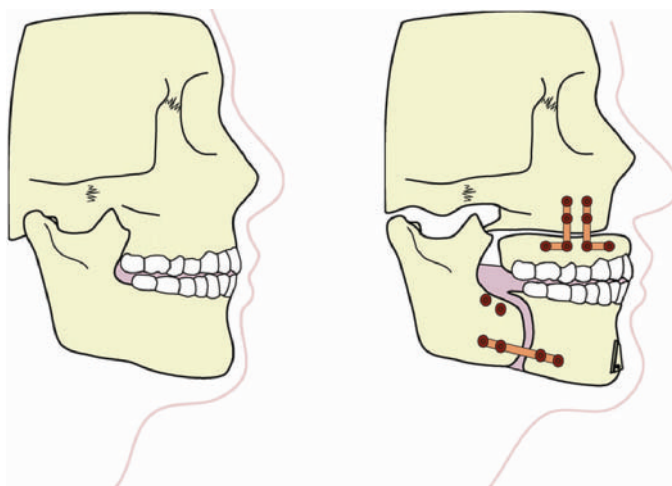


Figure 10.27 The maxillomandibular advancement osteotomy procedure. LeFort I maxillary osteotomy with rigid plate fixation and a bilateral sagittal split mandibular osteotomy with bicortical screw fixation. The advancement is at least 10 mm. (Li KK. Hypopharyngeal airway surgery. *Otolaryngol Clin North Am.* 2007; 40:849. Reprinted with permission.)

Complications of MMA can include long-standing anesthesia or paresthesia, malrelationship of the maxilla and mandible, infection, and bleeding.

Other considerations

The commercial market has made available two types of expendable devices known as nasal dilators. One is an internal device placed within the nasal passages, and the other is an external nasal strip placed on the outside of the nasal passages. The concept of these devices is to increase the nasal breathing ability by dilation of the nasal valve and thereby decreasing the nasal resistance.⁷¹

Both have been studied in patients with PSGs.^{72–75}

Results demonstrated that although there may be a subjective improvement relative to snoring, the snoring and AHI are not improved for most patients with SRBD.

CONCLUSION

The gold standard for treating OSA is PAP. However, not all OSA patients are compliant or adhere to the use of a PAP device, and for those individuals there are nonsurgical options, such as OA therapy, or surgical

intervention. When considering treatment options, the decisions should be site-specific relative to obstruction or narrowing of the airway spaces.

To determine the site-specific areas of disproportionate anatomy as well as to rule out any pathology of the upper airway, a complete history, clinical evaluation, and appropriate imaging of the patient are necessary.

Surgery is not without its complications, but the patient must weigh the benefits and risks in making the decision when electing to pursue surgery.

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11

Oral appliance therapy for sleep-related breathing disorders

CONCEPTUAL OVERVIEW

Oral appliance (OA) therapy has been used for the management of sleep-related breathing disorders (SRBD) since the early 1930s.¹ Subsequent investigations into the obstruction of the upper airway led to the study of OA therapy as a nonsurgical treatment option. These early research endeavors identified OA devices that were of two different designs: (1) mandibular repositioning appliance (MRA) that advanced the mandible in a protrusive position and (2) tongue-retaining device (TRD) that held the tongue in an advanced position. Both these OA designs demonstrated promise in reducing the upper airway obstruction.²⁻⁴ Following these early discoveries, there has been a myriad of studies assessing the clinical efficacy and long-term use of OA therapy for the management of SRBD.

The first standards of practice guidelines relative to the use and effectiveness of OA therapy for obstructive sleep apnea (OSA) and snoring were published in 1995 by the American Academy of Sleep Medicine (AASM, formerly the American Sleep Disorders Association).⁵ Case studies comprised 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.⁶

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 medicine⁶ and (2) a practice parameters update.⁷ 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 continuous positive airway pressure (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 United States Food and Drug Administration (FDA), and OAs are regarded as class II medical devices.⁸ 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.

HISTORY OF OAs

In the early part of the 1900s, the first OA that repositioned the mandible was used to address mandibular retrognathia.⁹ Of interest, this particular device also had a positive secondary effect on the airway. In the 1950s, OAs were utilized mainly to stimulate growth of the mandible for correction of a malocclusion, and they were, for the most part, not recognized for their role in airway improvement during sleep.

In the 1960s and 1970s, research related to the impact of OAs on the growth of the mandible was increasing. During this time, the impact on the airway from OAs designed to reposition the mandible was also being investigated.¹⁰ The research demonstrated the impact that airway compromise and obstruction had on growth of the mandible as well as the rest of the craniofacial complex. Accordingly, the resolution of airway compromise along with the resultant improved breathing had the potential to beneficially impact human growth and development.

The first available OA that was designed specifically for the management of SRBD was a TRD developed by a physician who was attempting to treat his own sleep breathing issues.^{11,12} Fabricated out of a flexible material with a bulb-like receptacle in the anterior portion, the TRD was intended to pull and maintain the tongue in a forward position during sleep so that the tongue would not collapse into the airway against the posterior pharyngeal wall, especially since there can be a tendency for the musculature to exhibit decreased muscle tone during sleep (Figures 11.1a–11.1c).

Other OA designs were introduced in the 1980s and 1990s, all of which were intended to reposition or advance the mandible to improve the upper

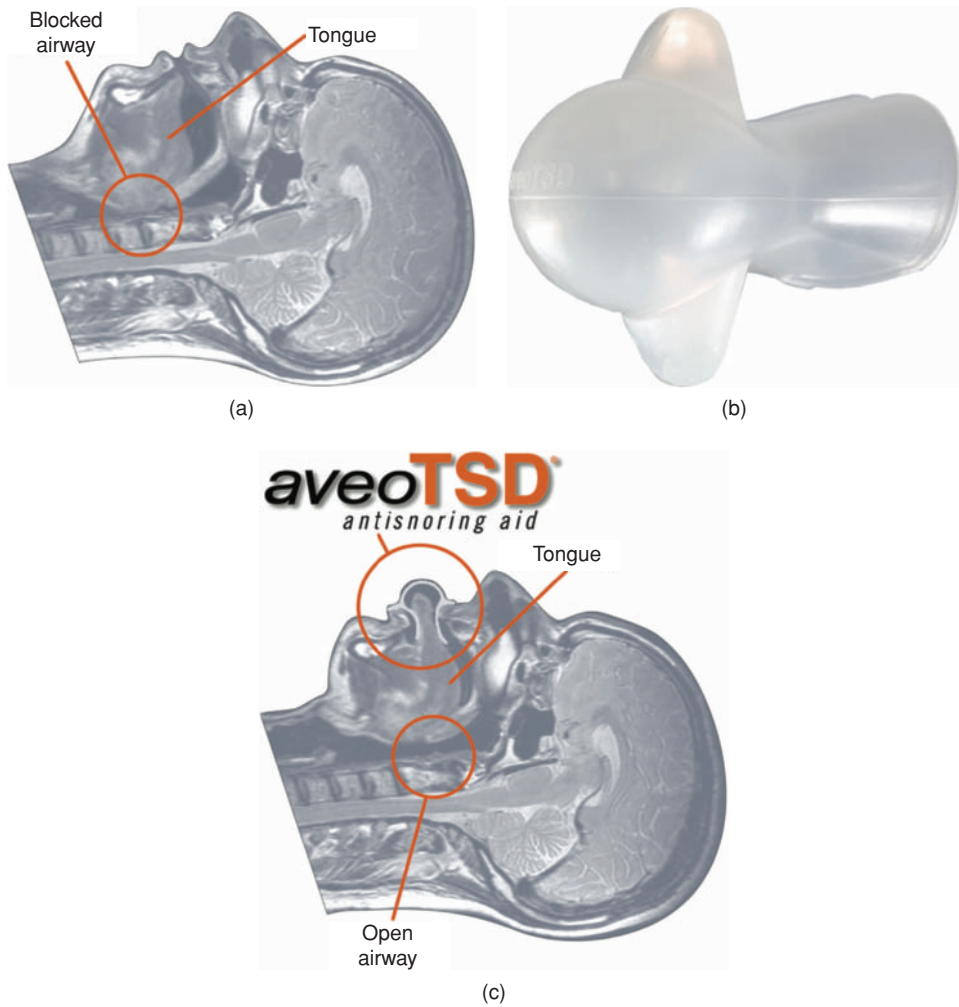


Figure 11.1 (a) Sagittal MRI of head: supine position demonstrating blocked airway from base of tongue impinging against posterior pharyngeal airway. (Courtesy of Innovative Health Technologies, 2009. Used with permission.) (b) Sample of a TRD: “aveoTSD”. (Courtesy of Innovative Health Technologies, 2009. Used with permission.) (c) Sagittal MRI of head: supine position demonstrating an opened airway from the use of the “aveoTSD” to maintain an advanced position of the tongue. (Courtesy of Innovative Health Technologies, 2009. Used with permission.)

airway patency. All these OA designs were referred to as anterior repositioning or mandibular repositioning OAs, with the term MRA often used.

The devices that were introduced in the 1980s were mostly a one-piece design that joined the maxillary and mandibular acrylic components together, and they were referred to as monoblocs (Figures 11.2a and 11.2b). However, this particular design did not allow for any mandibular

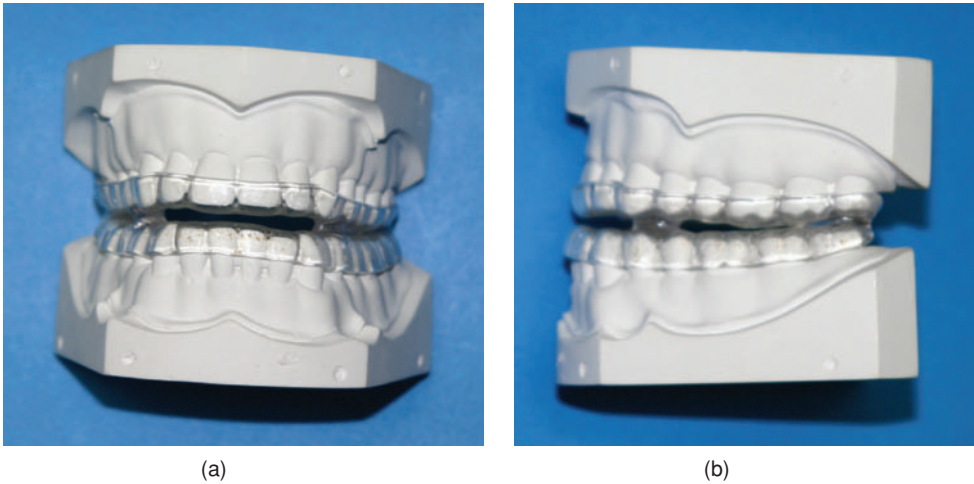


Figure 11.2 (a) Frontal view of monobloc type of MRA design. (b) Sagittal view of monobloc type of MRA design.

movement, such as the teeth grinding component of bruxism, and some patients reported discomfort with the masticatory muscles and/or temporomandibular joints (TMJs) after wearing this rigid type of design. In addition, if an alternative maxillomandibular relationship was deemed necessary for therapeutic or patient comfort purposes, it was not possible to modify the MRA without having to either (1) remake the entire OA or (2) separate the two components and reconnect them in a different maxillomandibular relationship with acrylic.

From the 1990s to the present time, most devices have been fabricated as separate maxillary and mandibular acrylic-type components that are joined together in a specific fashion that is unique to their respective manufacturer's mechanism, material, and purpose. Although the means by which the separate components are held together vary by manufacturer, the majority of them (1) account for bruxism by allowing for some degree of mandibular lateral excursions and (2) permit incremental anteroposterior adjustability (titration) for the dentist to easily modify the maxillomandibular relationship for optimum therapeutic effectiveness and patient comfort.

TREATMENT PROTOCOL, OBJECTIVES, AND CONTRAINDICATIONS

Because snoring and OSA are regarded as medical conditions, the dentist and physician need to cooperate with each other regarding the pretreatment assessment and management plan for the patient. The AASM⁶ and

the American Academy of Dental Sleep Medicine¹³ recommend similar treatment protocols for utilizing OA therapy in the management of OSA and snoring.

Both protocols outline the roles of the dentist and physician:

1. Assessment by a sleep physician: (a) the dentist can refer the patient to the physician to diagnose the SRBD via an overnight sleep study, and then the patient returns to the dentist if OA therapy is medically indicated, or (b) the patient is initially referred to the dentist by the sleep physician if OA therapy is medically indicated via an overnight sleep study.
2. The sleep physician provides the dentist with a written referral as well as copy of the diagnostic sleep study report.
3. The dentist assesses if the patient is a candidate for OA therapy, and the patient is advised of the appropriate OA design(s) for that patient as well as of the fees. Examples of OA designs should be shown to the patient, and a rationale for the appropriate OA design should be explained to the patient and documented in the patient record.
4. An informed consent about the risks and benefits of OA therapy for SRBD is obtained from the patient and documented in the patient record.
5. OA therapy is initiated by the dentist, which includes titration appointments as needed to obtain optimum effectiveness relative to the patient's initial symptoms.
6. Subsequent to the titration period, the patient is referred back to the sleep physician for medical assessment by the sleep physician relative to the OA's therapy effectiveness.
7. The final resolution of SRBD is determined by the sleep physician, typically with an overnight sleep study. If the initial medical diagnosis was primary snoring, then the dentist may complete OA therapy without the referral back to the sleep physician.
8. If medical assessment by the sleep physician demonstrates continued SRBD, then the dentist may need to consider additional titration of the MRA. The sleep physician may also recommend alternative treatment options, if appropriate.
9. An annual dental follow-up assessment is recommended for all OSA or snoring patients who have pursued OA therapy, especially since SRBD can have the tendency to worsen over time.

The AASM documents relative to its practice parameters and evidence-based review of literature lay the scientific foundation for the AASM's recommendations about the use of OA therapy for the management of OSA or snoring.^{6,7} The AASM defined the three levels of recommendations used in those documents as follows:

- *Standard*: "This is a generally accepted patient-care strategy, which reflects a high degree of clinical certainty."

- *Guideline:* “This is a patient-care strategy, which reflects a moderate degree of clinical certainty.”
- *Option:* “This is a patient-care strategy, which reflects uncertain clinical use.”

When considering the recommendations in the practice parameters, these definitions will serve as references for the dentist when engaging in the management of patients with OSA and/or snoring.

Assessment prior to initiation of treatment

Regarding assessment and diagnosis of SRBD relative to OA therapy, the AASM’s practice parameters made the following recommendation:⁷

The presence or absence of OSA must be determined before initiating treatment with oral appliances to identify those patients at risk due to complications of sleep apnea and to provide a baseline to establish the effectiveness of subsequent treatment. Detailed diagnostic criteria for OSA are available and include clinical signs, symptoms and the findings identified by polysomnography. The severity of sleep related respiratory problems must be established in order to make an appropriate treatment decision. (Standard)

Treatment objectives

Once the pretreatment assessment and diagnosis have been accomplished and OA therapy has been established as a treatment option for the SRBD patient, then the following treatment objectives for OA therapy in the management of SRBD, specifically OSA and snoring, are recommended per the AASM practice parameters:⁷

- “For patients with primary snoring without features of OSA or upper-airway resistance syndrome, the treatment objective is to reduce the snoring to a subjectively acceptable level. (Standard)”
- “For patients with OSA, the desired outcome of treatment includes the resolution of the clinical signs and symptoms of OSA and the normalization of the apnea-hypopnea index and oxyhemoglobin saturation. (Standard)”
- “Oral appliances are appropriate for use in patients with primary snoring who do not respond to or are not appropriate candidates for treatment with behavioral measures such as weight loss or sleep-position change. (Guideline)”
- “Although not as efficacious as CPAP, oral appliances are indicated for use in patients with mild to moderate OSA who prefer OAs to CPAP, or who do not respond to CPAP, are not appropriate candidates for CPAP, or who fail treatment attempts with CPAP or treatment

with behavioral measures such as weight loss or sleep-position change. (Guideline)”

- “Patients with severe OSA should have an initial trial of nasal CPAP because greater effectiveness has been shown with this intervention than with the use of oral appliances. Upper airway surgery (including tonsillectomy and adenoidectomy, craniofacial operations and tracheostomy) may also supersede use of oral appliances in patients for whom these operations are predicted to be highly effective in treating sleep apnea. (Guideline)”

Dental contraindications

The following dental contraindications for OA therapy in the management of SRBD, specifically OSA and snoring, are identified per the AASM evidence-based review of literature:⁶

- “Patients need to have an adequate number of healthy teeth (not compromised by periodontal disease) in the upper and lower dental arch to use an MRA.”
- “The patient should have the ability to protrude the mandible forward and open the jaw widely without significant limitation in order to be fitted with an MRA.”
- “Moderate to severe TMJ problems or an inadequate protrusive ability may be contraindications to OA therapy—mild TMJ problems may be lessened by the forward jaw position.”
- “Significant bruxism may be a contraindication to OA therapy.”
- “Patients with full dentures are generally unable to use an MRA but some of these patients may be treated with a TD” (tongue device).

Table 11.1 summarizes the indications and contraindications for OA therapy in the management of SRBD, specifically OSA and snoring.

Even though the presence of severe temporomandibular disorders (TMD) may be overstated, the dentist needs to (1) recognize any preexisting TMD prior to the initiation of OA therapy for SRBD and (2) discern whether the TMD may need to be addressed accordingly. In many instances, the TMD is not an intracapsular disorder of the TMJ itself, but rather more of a disorder involving the soft tissue components of the TMJ and/or the musculature of the head and neck.

Frequently, the masticatory and cervical muscles are tender to palpation, and they may have ability to refer pain to the area of the TMJ (Table 11.2).¹⁴ The ability for the musculature to refer pain to a location remote from the trigger points of the involved musculature is known as myofascial pain. Because the pain referral is from soft tissue, it typically can be relieved by exercise, physical medicine, and the use of muscle relaxation medications. In addition, the presence of sleep bruxism needs to be assessed since

Table 11.1 Indications and contraindications for oral appliance therapy.

Indications	Contraindications
Treatment of snoring alone (benign snoring)	Poor dental status
Patient with mild to moderate obstructive sleep apnea who <i>prefers</i> oral appliance over continuous positive airway pressure (CPAP) or did not respond to CPAP	Inadequate healthy teeth
Patient with severe sleep apnea who either failed CPAP or was intolerant to it	Active periodontal disease
	Inadequate mandibular function or limited range of motion
	Severe temporomandibular disorders

Source: Adapted from Kushida CA, Morgenthaler TI, Littner MR, et al. Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances: an update for 2005. *Sleep*. 2006; 29(2):240–262.

Table 11.2 Myofascial pain and trigger point referral for the most common muscles of the jaw and neck associated with oral appliance therapy.

Muscle	Common site for referral
Temporalis	Forehead and over the eyes Maxillary teeth (anterior portion to the anterior teeth, middle portion to the bicuspid, posterior portion to the molars)
Masseter	Ear and around the temporomandibular joint (TMJ) area Supraorbital region Mandibular posterior teeth
Lateral pterygoid	TMJ area Mid-face and infraorbital area
Medial pterygoid	TMJ and surrounding area Posterior face and mandible
Sternocleidomastoid	Face, supraorbital, back and top of the head, under the mandible Ear, behind the ear, forehead (only muscle that will refer across the midline to the other side of the forehead)
Diaphragm	Anterior teeth Neck, under the mandible at the posterior aspect (may feel like a sore throat)
Posterior cervicals	Face and TMJ area Forehead and the temples Back of the head

Source: Adapted from Travell JG and Simons DG. *Myofascial Pain and Dysfunction: The Trigger Point Manual*. Baltimore: Williams & Wilkins. 1983.

it can be associated with snoring and OSA, and it may be a precipitating mechanism.^{15,16}

FUNCTION OF MRA THERAPY

For the management of OSA and snoring, the basic function of OA therapy with an MRA design is to reposition the mandible in an increased vertical or open position as well as in an advanced or protrusive position relative to the maxilla. The basic function of a TRD design is to hold the tongue in a more forward position. Although the exact mechanism of the OA on the airway is not fully understood, it is believed that OA therapy improves the patency of the upper airway by increasing the pharyngeal volume or size and/or reducing the collapsibility of the airway through muscle tone improvement.

The upper airway of OSA patients is more narrowed during sleep than those who do not have OSA.¹⁷ Studies have shown that anterior repositioning of the mandible and tongue increases the size and volume of the upper airway, typically in the retropalatal and retroglottal areas.^{18–24} Not all studies, though, demonstrate such airway size increase. One study found that there was no change in the airway size with mandibular advancement.²⁵ Lending to the suggestion that increasing the airway size beneficially affects the severity of OSA are the studies that reflect a reduced apnea–hypopnea index (AHI) with increased mandibular advancement.^{26–29}

Repositioning the mandible also impacts the muscles that support the pharyngeal airway and comprise the tongue. The action of mandibular repositioning may do the following: (1) prevent the tongue from collapsing posteriorly into the oropharyngeal area and (2) stabilize the musculature that supports the airway. A study demonstrated that improvement in the airway is impacted by the palatoglossus muscle on the tongue base when accompanied by mandibular opening.³⁰

The musculature that is related to the tongue and the pharynx may also be beneficially affected by mandibular repositioning. With the mandible advanced, the musculature of the pharynx and the tongue are stabilized, thus preventing these structures from collapsing, narrowing, and obstructing the pharynx during snoring or OSA. Studies on mandibular and tongue advancement with OA therapy found increased muscle tone of the upper airway and decreased genioglossus muscle tone.^{31,32}

In addition, the repositioning of the mandible also positively impacts the velopharyngeal area and thereby improves the patient's ability to nose breathe.³³ It is believed that the MRA's impact on the palatopharyngeus creates an increased amount of tension on the soft palate, and the repositioning moves the tongue base forward and away from the inferior surface of the soft palate, resulting in increased patency of the nasopharynx for improved breathing (Figures 11.3–11.6).

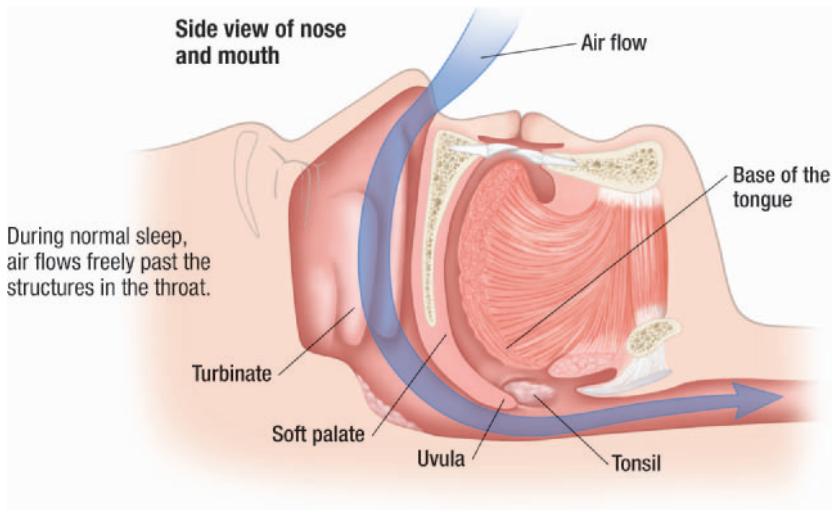


Figure 11.3 Normal sagittal view of the upper airway. (© Krames Medical Publishing, 2009. Used with permission.)

DESIGN CHARACTERISTICS OF MRAs

For MRAs to be optimally effective and reduce the impact of side effects, there are several design characteristics that should be considered when selecting an MRA design. The selection should be based on (1) specific and related medical and dental conditions of the patient, (2) patient preference,

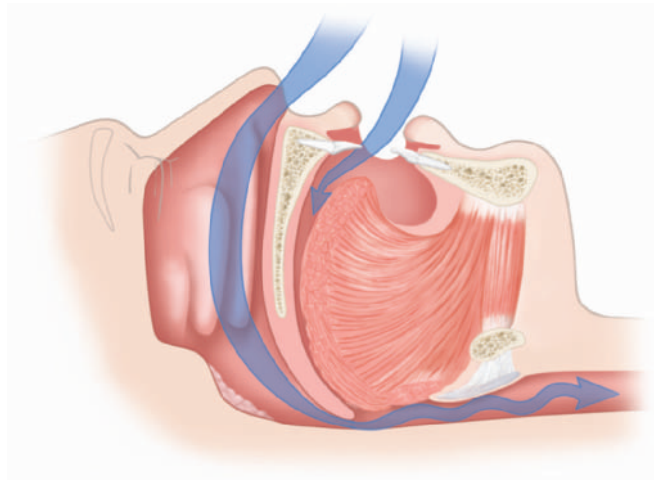


Figure 11.4 Sagittal view of partially blocked upper airway. (© Krames Medical Publishing, 2009. Used with permission.)

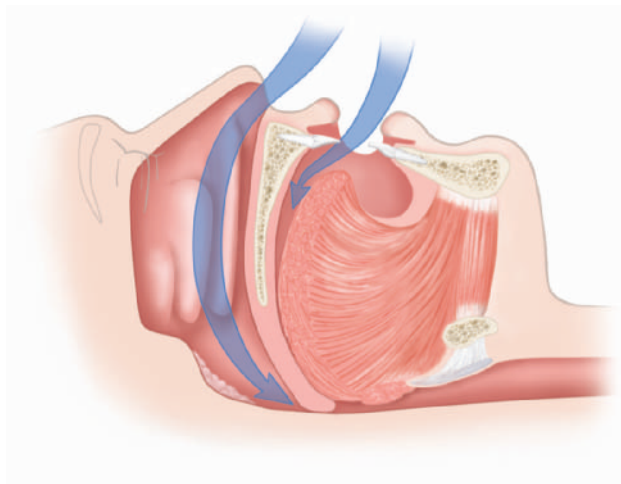


Figure 11.5 Sagittal view of completely blocked upper airway. (© Krames Medical Publishing, 2009. Used with permission.)

and knowledge, experience, and preferences of the dentist providing the service.

The following are suggested characteristics of MRAs for optimum effect in the management of OSA and snoring:

- *Adjustable*: It (1) is easily adjusted to accommodate changes in the dental status, (2) can be modified to address a new dental restoration, and (3) can be relined to improve retention or the fit.

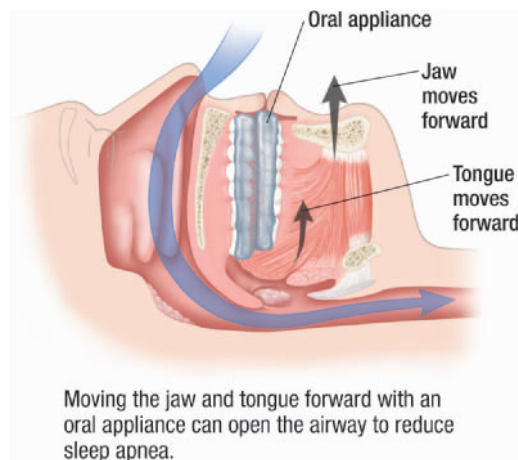


Figure 11.6 Sagittal view of an opened airway from the use of an OA. (© Krames Medical Publishing, 2009. Used with permission.)

- *Posterior support*: It (1) addresses sleep bruxism as well as SRBD and (2) provides adequate support for the TMJs.
- *Titration*: It incorporates a mechanism that allows incremental positional changes (e.g., anterior, posterior, vertical).
- *Full coverage of teeth*: It covers all the teeth to prevent tooth movement or unwanted supereruption, and maintains the teeth in the pretreatment position.
- *Mandibular mobility*: It (1) allows freedom of mandibular movement during sleep bruxism that is also termed as rhythmic masticatory muscle activity, which is commonly found in all patients; (2) improves comfort of the OA during sleep, especially with sleep bruxism; and (3) allows for normal function such as swallowing and licking the lips.
- *Lip seal and patent nasal airway*: It (1) allows for comfortable lip seal to limit mouth breathing during sleep and (2) improves blood oxygen saturation levels via an open nasal airway, otherwise adjunctive treatment of the nasal airway may be required.
- *Maximum tongue space*: It affords maximum space for the tongue to prevent further collapse of the tongue into the pharyngeal airway space, especially since encroachment of the OA into the tongue space may cause the tongue to be in a more posterior position in the mouth.

EFFECTIVENESS OF OAs

There are a number of studies and literature reviews that have evaluated OAs and their effectiveness, including the AASM evidence-based review of literature which included objective sleep data before and after OA therapy.⁶ Another literature review document also summarized in 2007 many of the studies published on OAs relative to their effectiveness for the management of SRBD.³⁴ Although the data in these documents support the use of OAs, it continues to be difficult to predict an outcome with OA therapy for any specific SRBD patient. Other than an objective sleep study that includes a polysomnogram, the currently accepted means by which OA effectiveness can be reliably documented through self-reports is with the use of the Epworth Sleepiness Scale (ESS). It has been shown that the ESS score was improved in OA users from 11.2 to 7.8,³⁴ and other self-report assessments also demonstrated significant reductions in ESS scores regarding the efficacy of OA therapy.^{27,35-37}

When using respiratory sleep data to assess the affect of OA therapy on severity of respiratory disturbances for individuals with OSA, the definition of success varied among the studies considered in the AASM review of literature: “the most stringent definition of success was a reduction to less than five respiratory events per hour of sleep while the most liberal definition was a reduction of 50% or more from the baseline AHI.”⁶

OAs have also been found to be effective in the improvement of cardiovascular disease. Three studies documented improvement in blood

pressure measurements with the use of OA therapy.^{35,38,39} One study found that in 161 patients who used an OA for 60 days, the improvement in the blood pressure went from 132.0/82.1 to 127.5/79.2. Another study indicated that the improvement in blood pressure was equivalent to that found with the use of CPAP therapy.

A study that evaluated oxidative stress after 1 year with the use of an OA demonstrated improvement in the AHI, ESS, and endothelial function.⁴⁰ Although the apneic events were not totally eliminated, there was improvement that offered encouragement for these conditions in the future.

Effectiveness of OA therapy has also been evaluated relative to other therapies, such as CPAP and surgery, as well as comparison among different OA designs.

Comparison of OAs with CPAP

There are several crossover studies that have compared MRA therapy with CPAP.^{37,41-45} The findings in each of the studies confirmed that CPAP was more efficacious in reducing the AHI to normal levels as well as controlling snoring in almost all patients, whereas OA therapy was able to do the same in about two-thirds of the patients. However, these studies also indicated that OA therapy was the preferred method of treatment. In addition, the use of an OA demonstrated better compliance when compared to CPAP. One such study found that in 41 patients who had utilized both an OA and CPAP, 71% preferred the OA.⁴⁶

Comparison of OAs with surgery

For comparison of OA therapy and upper airway surgery, there has been one randomized parallel study,⁴⁷ several related reports,⁴⁸⁻⁵¹ and two reviews of literature documents.^{6,34} Results found that after 1 year, 51% of the uvulopalatopharyngoplasty (UPPP) surgery patients had an AHI less than 10 compared to 78% of the OA patients. At the 4-year interval, 3% of the UPPP surgery patients and 63% of the OA group had an AHI less than 10.

The one surgery that appears to have the best prognosis as compared to OAs is maxillomandibular advancement surgery.⁵² In one study, 4 patients out of a total of 43 with an OA had an initial AHI of 50 that decreased to 12, and the AHI for the surgery group was reduced to 2.

FABRICATION AND DELIVERY OF THE OA

The following recommendation for the dental management, including the fitting of an OA, for patients with OSA is provided by the AASM practice

parameters for OAs and was based on committee consensus:⁶

- “Oral appliances should be fitted by qualified dental personnel who are trained and experienced in the overall care of oral health, the temporomandibular joint, dental occlusion and associated oral structures. Dental management of patients with OAs should be overseen by practitioners who have undertaken serious training in sleep medicine and/or sleep related breathing disorders with focused emphasis on the proper protocol for diagnosis, treatment, and follow up. (Option)”

Informed consent

There are a number of informed consent examples available for the use of OA therapy for the management of SRBD. Some of the OA manufacturers have ones that are unique to their specific device. The American Academy of Dental Sleep Medicine provides one that is available only to their members and it is generalized for all OAs.

Regardless of which one is selected or developed, it is advisable for the dentist to contact his or her malpractice insurance carrier or an attorney regarding specific content of the consent form. The major factors of concern are to be certain that the patient is advised of the need for ongoing follow-up care, the side effect potentials, and the use and care of the OA. It is also important that the patients have had any questions or concerns answered, and that this communication has been documented in the patient record. The follow-up visits should also be documented in detail, particularly relative to any side effects as well as improvement in the patient’s symptoms and sleep quality.

Making impressions

The actual fabrication of the OA starts with the process of obtaining impressions of the patient’s maxillary and mandibular teeth and related structures, which are subsequently poured into stone casts. The impression material should be stable and provide a detailed representation of the teeth and surrounding structures. Material selected for the impressions can be (1) a quality alginate, (2) one of the products on the market that is designed as an alginate substitute, or (3) a polyvinylsiloxane. The advantage of alginate substitute and polyvinylsiloxane materials is the allowance for the dentist to do multiple pours of the impression, which affords the option of maintaining a pretreatment cast for future reference. Retaining duplicates of the master casts as part of the record can be helpful in the event that there are concerns regarding tooth movement or changes in the maxillo-mandibular relationship. In addition, these retained casts may be needed for future use in the event that repairs to the OA are needed because of fracture or breakage.

The mandibular impression should provide adequate detail of the teeth and all the lingual anatomy with appropriate extension into these areas. If there are mandibular lingual tori present, these should be captured in

the impression so that the master cast has sufficient detail for the dental laboratory to provide relief around them or to design the OA's mandibular component to circumnavigate the tori.

The maxillary impression should also have adequate detail of the teeth and the hard palatal area. Depending on the OA design, it may or may not be necessary for the entire hard palate to be captured in the impression. If the anteroposterior adjustment (titration) mechanism is going to be located in the palatal vault region, then it would be necessary to capture that palatal aspect in the impression. If such mechanism design is not going to be employed in that palatal region, then it would not be as necessary to capture the complete palatal area since the hard palate will not be totally covered by the OA's maxillary component. For some patients, covering the entire hard palate has the potential of interfering with proper tongue space and position during function (e.g., swallowing).

After the impressions are obtained, they need to be poured in a relatively hard stone, usually the typical die stone used for fixed prosthodontics. These casts along with the interocclusal record (IR) will be sent to the dental laboratory for the fabrication of the appliance.

If there is any concern about the impressions for the fabrication of the OA or the cast that is needed, it is always best to contact the dental laboratory that will be fabricating the appliance for clarification and/or their specifications per the OA design.

Interocclusal record

The IR, or bite registration, is typically established by the dentist at the same appointment as when the final impressions are obtained. The IR allows the dentist to determine the maxillomandibular relationship, or the therapeutic window, at which he or she will begin OA therapy for the SRBD patient. Generally, the therapeutic window is the three-dimensional anteroposterior and vertical positioning of the mandible relative to the maxilla that addresses the OSA and/or snoring.

If the dentist elects to employ an OA design that has a titration mechanism connecting the maxillary and mandibular components of the OA, then he or she has the ability to make incremental adjustments of that maxillomandibular relationship to potentially (1) modify the effectiveness of the OA regarding the OSA and/or snoring and (2) assist the patient's masticatory muscles and TMJs to slowly adapt to the optimum position in that three-dimensional therapeutic window.

A study assessed two OA designs and the patient's AHI responses to each design.⁵³ One of the designs repositioned the mandible anteriorly, and the other design was a placebo with no mandibular repositioning. The repositioning OA reduced the AHI, whereas there was no AHI improvement with the other OA design. Two prior studies and three subsequent studies reflected similar findings.^{27,54-57} One of the studies used the same OA design, but compared AHI responses in two groups of patients relative to different amounts of mandibular advancement.²⁷ When comparing the

two groups of 50 or 75% of the maximum distance that the patient could protrude, there was a reduction to <10 AHI in 31 and 52% of the patients, respectively.

The collective findings of these studies appear to indicate that greater degrees of mandibular advancement would result in greater reductions in AHI scores. As such, the therapeutic window for making the IR is typically in the 50–75% range of maximum mandibular protrusion.

Relative to the vertical opening component of the three-dimensional therapeutic window, studies have evaluated this component relative to OA effectiveness, but with mixed results (www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfPMN/pmn.cfm).^{22,42} Although this vertical component needs further investigation, the typical vertical opening of the IR can range from 2 to 10 or more millimeters, depending on the uniqueness of each individual patient case.

In addition, the IR fabrication technique and specifications may be available from the dental laboratory or the manufacturer for a specific OA design, and the dentist will need to use his or her best assessment when taking into account the patient's particular clinical case.

Table 11.3 outlines factors to consider when making the IR, and the following goals are associated with its use:

1. Be able to maintain a lip seal with the OA seated on the dentition, which will foster nasal breathing during sleep.
2. Provide the least amount of strain on the masticatory musculature with the use of the OA.
3. Focus on the combined approach of mandibular advancement and vertical opening: (a) for optimum effectiveness of the OA and (b) to lessen the possibility of occlusal changes with the use of an OA.

Making the IR can be accomplished in a variety of ways. The use of a device called the George Gauge[©] has been a very standard technique (Figure 11.7).

The George Gauge[©] is composed of a handle to which a bite fork component is attached. The handle portion is designed to (1) hold the bite fork and (2) allow for the determination of the amount of advancement that is desired by virtue of a sliding scale on the one side. On the opposite side, there is the ability to adjust the thickness of that portion that fits over the mandibular anterior teeth by loosening the setscrew and sliding the lingual portion forward or backward. This allows for variations in the size of the teeth as well as accommodates for crowding or rotations of the anterior teeth. The device also has a midline mark indicator for the maxillary teeth that can be used as a reference point.

The bite fork component is designed such that two different vertical measurements can be obtained, 2 mm or 5 mm, in the interincisal area. The 5 mm bite fork is typically more frequently used as it allows for interarch spacing in the posterior areas necessary for most OA designs. If additional vertical spacing is desired, the incisal notch of the maxillary portion of the

Table 11.3 Proposed interocclusal record for oral appliances: initial starting position for mandibular repositioning appliance design.

Vertical	Start at between 5 and 7 mm interincisally (Edge to edge) Take into consideration Maintain a lip seal Able to nose breathe Inability to voluntarily snore Subjective improvement in airway
Advancement of mandible based on relationship of incisors and angles classification	Class I (full dentition) Incisors at edge to edge Class I division 2 Advanced 2–3 mm past edge to edge Class I (previous bicuspid extraction) Advanced 1–4 mm past edge to edge Class II division 1 to <5 mm overjet Advanced up to 5 mm—not beyond edge to edge Class II division 1 to >5 mm overjet Advance up to 5 mm—not beyond Titrate advancement over time as deemed necessary Class II division 2 Advance 2–4 mm beyond edge to edge Class III—prognathic mandible Minimal to no advancement Focus on vertical Class III—retrognathic maxilla (Pseudo class III) Minimal advancement (1–3 mm)

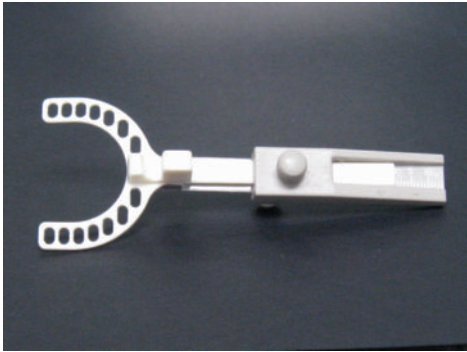
Special Considerations: In class III and more severe class II patients, a cephalometric radiograph should be done to evaluate the skeletal development of the patient.

bite fork can be modified by the addition of either a self-curing or light-curing acrylic. Once the proper bite fork has been selected, the amount of vertical should be verified with some type of measurement device such as a Boley gauge (Figure 11.8).

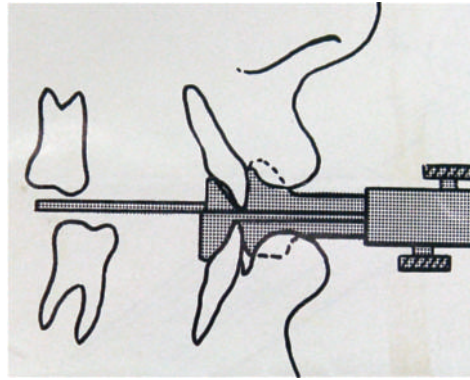
Occasionally, the groove of the bite fork that accommodates the maxillary teeth will need to be modified because of anatomical variation or crowding. The lingual portion of the groove is often adjusted or modified so that the bite fork is stable when seated on the anterior dentition.

Delivery of the mandibular repositioning appliance

Delivery, or fitting, of the MRA for SRBD is similar to any other type of OA, although there may be adjustments and considerations unique to the



(a)



(b)



(c)



(d)



(e)

Figure 11.7 (a) The George Gauge[®] for the IR. (b) Diagram of sagittal view of George Gauge[®] seated intraorally for making the IR. (c) Mandibular anterior clamp of George Gauge[®] adjusted to the lingual of the teeth for positioning of the anterior notch on the mandibular incisor teeth. (d) Sagittal view of George Gauge[®] in place ready for the IR material. (e) Frontal view of George Gauge[®] in place ready for the IR material.

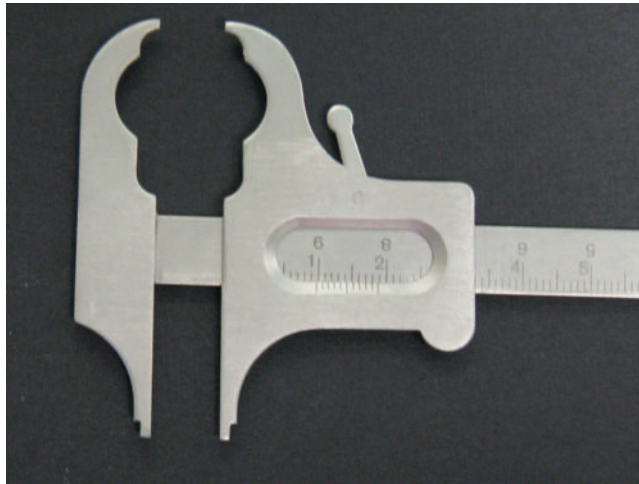


Figure 11.8 Boley gauge to assist in determining the optimum amount of vertical opening for the IR.

specific OA design being utilized. The major factors involved with the delivery of the MRA are the following:

Optimum fit: The device should be snug with adequate retention and should not cause discomfort to any of the teeth or to the masticatory musculature. It may initially feel tight to the patient because of the retention components of the OA, and there also may be masticatory muscle strain because of the mandibular advancement. If this is the case, the patient should be scheduled for a follow-up visit within a week after the delivery. At this time, the position of the mandible should be verified in accordance with the predetermined position that was established at the time of the IR.

Adjustment of the OA's occlusal tables: Many OAs have contact between the maxillary and mandibular components (posterior occlusion). This will need to be balanced and adjusted at the delivery appointment for comfort.

Verify a lip seal: Because a lip seal is optimum, this should be determined at this time. If the lip seal appears strained, possibly the vertical will need to be reduced at this time.

Determine the ability to nose breathe: Determine if the patient has the ability to adequately nose breathe. If this appears to be a problem and the lip seal is adequate, then oftentimes the use of nasal strips may be helpful initially along with the use of nasal irrigation with a saline solution (Figure 11.9). One product that is very popular with physicians and is easy to use is the Neil Med Sinus rinse. It is a combine saline and sodium bicarbonate mixture that is prepackaged and is mixed with water.



Figure 11.9 Neil Med Sinus Rinse[®]: over the counter product for nasal airway irrigation.

At this visit, the patient will need to be instructed in the proper use (removal and insertion), care, and the possible adverse effects associated with the use of an OA that repositions the mandible. Some of the manufacturers may have preprinted instructions that are available that can be given to patients when the appliance is delivered.

Follow-up care

Follow-up care visits should be for the purpose of monitoring the outcome of the OA therapy. The frequency of these visits is determined by the progress that the patient makes with the OA, but the first follow-up visit is

oftentimes within 2 weeks following the delivery of the OA. Oftentimes a patient will be appointed 4–6 times after the OA has been delivered over a 6-month time period, after which the patient will usually be aware of an improvement in their sleep as well as a decrease in the initial symptoms.

At each follow-up visit, the following may be addressed:

Adjust the OA: At these visits the overall fit, retention, posterior bite relationship, and comfort of the OA should be reviewed. Any needed adjustments should be made at this time and will be reviewed at a future follow-up visit.

Assess the symptoms: The original symptoms can be reviewed to determine if there is improvement. One of the key elements of major concern is snoring, an indicator that an adequate patent airway may not yet be achieved.

Look for side effects: At each follow-up visit, adverse outcomes of OA use should be evaluated, which includes maxillomandibular changes, tooth movement, masticatory muscle pain, and TMD-related findings. If there are any changes, then they need to be addressed immediately.

Review the plan: It is important to address the plan for the future. Because the patient has been sleep-deprived, they may not remember all the information related to the treatment from the previous visits.

Testing for efficacy

The AASM practice parameters for OA therapy of OSA and snoring state the following relative to follow-up sleep testing:⁷

- “Follow-up sleep testing is not indicated for patients with primary snoring. (Guideline)
- To ensure satisfactory therapeutic benefit from OAs, patients with OSA should undergo polysomnography or an attended cardiorespiratory (Type 3) sleep study with the oral appliance in place after final adjustments of fit have been performed. (Guideline)
- Patients with OSA who are treated with oral appliances should return for follow-up visits with the dental specialist. Once optimal fit is obtained and efficacy shown, dental specialist follow-up at every 6 months is recommended for the first year, and at least annually thereafter. The purpose of the follow-up is to monitor patient adherence, evaluate device deterioration or maladjustment, evaluate the health of the oral structures and integrity of the occlusion, and assess the patient for signs and symptoms of worsening OSA. Intolerance and improper use of the device are potential problems for patients using oral appliances, which require patient effort to use properly. Oral appliances may aggravate temporomandibular joint disease and may cause dental misalignment and discomfort that are unique to each device. In addition,

oral appliances can be rendered ineffective by patient alteration of the device. (Option)

- Patients with OSA who are treated with oral appliances should return for periodic follow-up visits with the referring clinician. The purpose of the follow-up is to assess the patient for signs and symptoms of worsening OSA. Close communication with the dental specialist is most conducive to good patient care. An objective reevaluation of respiration during sleep is indicated if signs or symptoms of OSA worsen or reoccur. (Option)''

While the management of OSA with OA therapy is the dentist's role in the overall treatment of a medical condition, the ultimate responsibility for the SRBD patient belongs to the physician. This is particularly important to recognize given the large number of health-related consequences associated with sleep apnea.

Once the OA appears to have achieved an optimum result, follow-up testing is important to document. The follow-up testing can be accomplished in a variety of ways depending on the comfort level of the dentist providing the service. There are two major areas where improvement is best determined, neurocognitively and cardiovascularly. The neurocognitive improvement may be assessed with the ESS as well as subjective reports relative to the patient's mood and memory.

The ESS should be repeated periodically throughout the OA therapy period. If it was used prior to initiating the treatment, then this will help to determine if the patient's tendency to be drowsy during the day has improved. This is always a significant improvement particularly since being sleepy and/or tired are often associated with sleep apnea.

Improvement in mood and memory indicates improvement in brain function associated with improved sleep and possibly increased oxygen saturation levels. Although these parameters are subjectively reported by the patient and/or significant other, at least this information can be compared both pre- and post-OA therapy.

Improvement in the cardiovascular status of the patient may best be determined by obtaining the patient's blood pressure since it has been shown that OAs can improve the patient's blood pressure.

At this time, the use of portable monitoring devices for home sleep studies to determine the efficacy of the OA therapy may be utilized. There are many ambulatory devices available, and both the option of using this device to test the OA as well as which specific one to use has to be determined by the individual practitioner. The use of such devices should be considered in light of the comfort level of the dentist as well as their understanding of the specific recommendations for their use.

One place where such devices may have a role is in the initial phase of the treatment as a means of determining the performance level of the OA. A clinical guidelines paper published in 2007 indicates that the use of a Type 3 device that measures 4–7 channels of data is a valid indication for

evaluating the response to non-CPAP treatment such as an OA.⁵⁸ This Type 3 device would allow for objective testing of the OA to determine if further adjustment or modification of the OA is warranted prior to referring the patient for a sleep study with the OA. At all times, the data from this testing should be shared with the sleep specialist and the patient's physician. In no way should the use of the portable monitoring devices be used to determine if the patient is at risk for OSA prior to them having a formal sleep study and diagnosis by the physician. This would most likely be viewed as practicing medicine without a license and is not within the scope of the practice of dentistry.

On the basis of the AASM practice parameters on OAs, the current recommendation is for the patient to have a formal overnight sleep study once the OA has been adjusted, titrated, and modified to the point of delivering an optimum outcome. Because a satisfactory, efficient, and cost-effective method of titrating an OA during the sleep study has not been developed as has been done for positive airway pressure (PAP), the overnight sleep study with the OA in place is the best way to test for the efficacy of the device.

SIDE EFFECTS AND THEIR MANAGEMENT

Side effects with the use of OAs that reposition the mandible can be a concern. The overall long-term side effects are not well known. In many cases, patients will continue to use the OA despite the side effects as long as they are experiencing a positive result and outcome. The main reason that people stop using the OA is related to a lack of effective outcome. Compliance with the use of an OA is related to the perceived benefit despite the side effects. In one study it was found that compliance ranged from 48 to 84% after 2 years of use.⁵⁹

The most common side effects or adverse outcomes and means by which they can be addressed are the following.

Excessive salivation

This is more prevalent at the onset of treatment and usually subsides with the continued use of the OA.

Intervention: If this persists, then it may indicate that there is mouth breathing as a result of inadequate lip seal or restricted nasal airway or both. Patients who were chronic mouth breathers prior to receiving the OA may have the most difficulty. If the nasal airway is open, then the adjunctive use of a chin-up strip may be helpful to correct this issue.

Face, muscle, and TMJ discomfort

This is most likely associated with the strain on the masticatory musculature associated with mandibular repositioning, which usually occurs in the

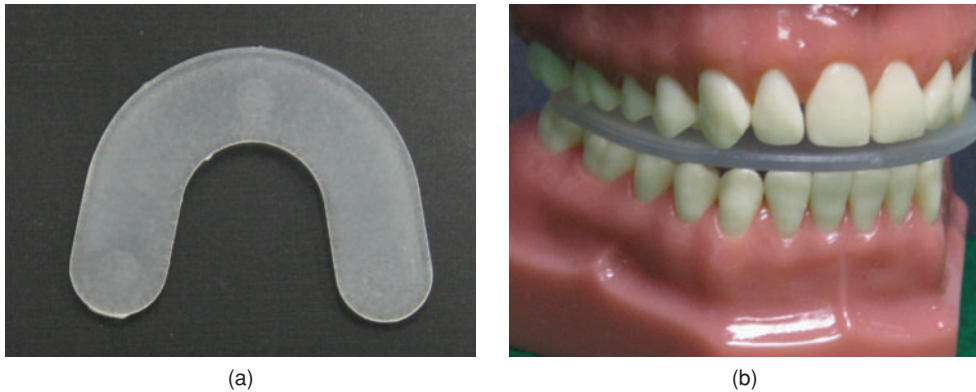


Figure 11.10 (a) Therapeutic Bite Rim[®]. (b) Therapeutic Bite Rim[®] in place.

early stages of OA therapy and typically resolves over time with continued use of the OA. Regarding any concern that OAs may cause a TMJ problem, this has not been found to be the case. In one study after a full year of OA therapy, the development of TMD was not found to be present and there were no morphologic changes involving the joints.²⁶

Intervention: At times, intervention is needed to resolve these issues, but they can usually be managed with mandibular range of motion exercises. One such exercise involves the use of a therapeutic bite rim (Figures 11.10a and 11.10b).

The patient places the rim between the maxillary and mandibular teeth and subsequently slides the mandible anteriorly and posteriorly up to ten times after removing the OA. Also, the application of moist heat to the masseter areas while doing the exercises can be helpful. Sometimes a short course of muscle relaxation medications, such as Flexeril, Skelaxin, or a similar pharmaceutical, may help when taken prior to initiating sleep. In some situations, referring the patient for physical therapy may also be indicated if the problem persists.

Maxillomandibular relationship or occlusal changes

One of the areas of major concern is a change in the maxillomandibular relationship or occlusion associated with the repositioning of the mandible for upwards of 6–8 hours a day. These changes may last for up to 30 minutes or longer after discontinuing the use of the OA.

Intervention: The use of the therapeutic bite rim can be helpful. In rare situations, the use of the OA has to be discontinued. Reducing the amount of advancement of the mandible and focusing on the vertical opening may also help, but there are no studies to date to confirm this. In many cases, a simple tongue-up exercise may also be helpful. The patient is instructed to open the mouth as wide as possible. With the mouth held open, the tongue

is placed up and as far back toward the soft palate as possible. While holding the tongue up in this position, the patient closes the mouth. The basis of this exercise is that it activates the muscles that retrude the mandible, thus counteracting the forces that advanced the mandible.

In some situations, patients will report that despite the feeling that the posterior teeth do not fully contact in occlusion, they would rather live with that side effect because the OA has had such a significant impact on their sleep. In these situations, the patient should be advised to continue to use the therapeutic bite rim exercise, do the tongue up exercises, and return for follow-up evaluation on a more frequent basis. It is imperative that this be documented in the patient record for medicolegal purposes. To further document the situation, photos may be taken periodically.

Opening of interproximal contacts

There have been reports on an antidotal basis as well as clinically of a separation of contacts with the use of OAs that leads to open contacts and food catching between the teeth.

Intervention: The best method to prevent this side effect is to be certain that all the teeth are covered and adequately surrounded so that there is little to no chance of tooth movement. This has been reported on a very limited basis, and patients need to be advised of this potential side effect in advance as part of the informed consent form.

CURRENT STATUS OF OAs

At this time, there are many different OA designs from which to select. The FDA currently views OAs as a singular group and does not distinguish them separately for snoring or OSA as it had done initially. In 2002, the FDA reclassified OAs as class II medical devices indicating that special controls may be needed.²⁶

The FDA Web site relative to OAs has listed more than 40 OAs as being cleared and receiving a 510(k) along with the name and contact information relative to the inventor or distributor. A listing of FDA-cleared OAs can be found on the FDA Web site by inputting the user code LRK (www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfPMN/pmn.cfm).

There are devices that also may be fabricated by the dentist or by a dental laboratory for individual use and are not openly advertised or marketed. In these cases, a 510(k) is not required. In many instances, an insurance company may ask for the 510(k) designation for the specific OA being utilized. Therefore, it is recommended that anyone who is providing treatment for OSA or snoring become familiar with the specific OA as it relates to the FDA.

There are numerous OAs available at this time and the majority of them are custom-fabricated. However, there are a few that are of a thermoplastic

design, which are commercially available as over-the-counter (OTC) products, also termed as boil-and-bite or boil-and-fit. One study found that the OTC OAs are not as successful as the custom-fabricated OAs.⁶⁰ In addition, the OTC devices were not found to be very effective as a trial appliance, and the failure rate associated with their use was 69% as compared to a 63% success rate with custom-fabricated OAs. The lack of success was primarily related to poor retention. In addition to the findings of this study, the OTC OAs are usually designed as one-size-fits-all and consequently are usually quite bulky.

Based on the 510(k) listing by the FDA along with an awareness of the available OAs, a listing of these appliances is provided that reflects information submitted by manufacturers or inventors who responded to a request to submit information on their specific OA. The results of this inquiry are summarized here in an effort to describe many of the different devices that are available currently.

Note: The OAs identified in this book do not constitute a full and complete listing of all OAs currently available. The list represents those OAs from manufacturers or inventors who responded to a request for information and figures relative to their respective OA. In like manner, the listing of these OAs in no way should be construed as a recommendation for the use of any one of these OAs. The ultimate choice of an OA is the responsibility of the individual providing the service along with the comfort level and results that have been achieved with particular OAs.

Sample of available OAs

Depending on experience and licensing, many of the OAs listed may be available from more than one dental laboratory.

Common name of OA:

TAP[©] (Thornton Anterior Positioner) (Figure 11.11)

Contact information:

866-AMISNOR

www.amisleep.com

Type of OA design:

Mandibular repositioning

Common name of OA:

EMA[©] (Elastic Mandibular Advancement) (Figure 11.12)

Contact information:

800-588-7898

www.openairway.com

Type of OA design:

Mandibular repositioning

Common name of OA:

Klearway[©] (Figure 11.13)



Figure 11.11 TAP[®] III Pro. (Courtesy of Airway Management Inc., 2009. Used with permission.)

Contact information:

800-828-7626

www.klearway.com

Type of OA design:

Mandibular repositioning

Common name of OA:

TheraSnore[®] (Figure 11.14)

Contact information:

800-477-6673

www.distar.com



(a)



(b)

Figure 11.12 (a) Sagittal view of EMA[®]. (Courtesy of Frantz Design Inc., 2009. Used with permission.) (b) Lingual view of EMA[®]. (Courtesy of Frantz Design Inc., 2009. Used with permission.)

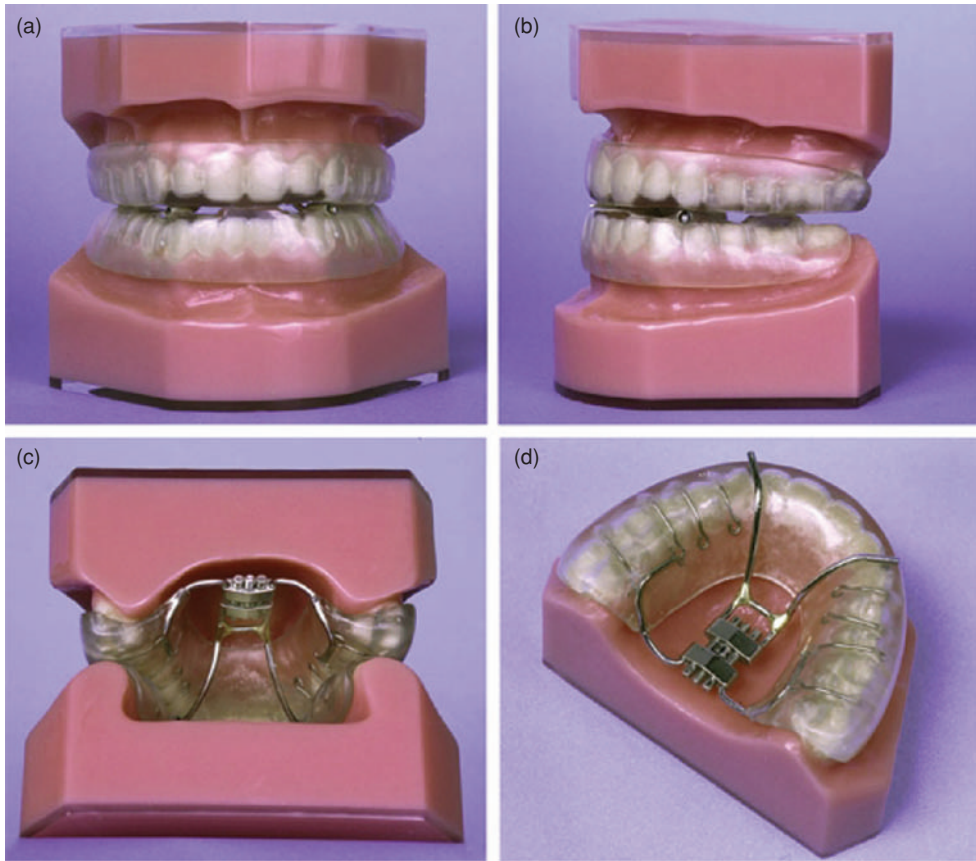


Figure 11.13 Various views of Klearway[®]. (a) Frontal view, (b) sagittal view, (c) lingual view, and (d) occlusal view of maxillary component. (Courtesy of Lowe A and Great Lakes Orthodontics Inc., 2009. Used with permission.)

Type of OA design:

Mandibular repositioning

Common name of OA:

PM Positioner[®] (Fixed and Adjustable) (Figure 11.15)

Contact information:

800-253-1196

www.pmpositioner.com/tech1.html

Type of OA design:

Mandibular repositioning

Common name of OA:

OASYS[®] (Oral/Nasal Airway System) (Figure 11.16)

Contact information:



Figure 11.14 TheraSnore[®]. (Courtesy of Distar, 2009. Used with permission.)

888-866-2727

www.oasysleep.com

Type of OA design:

Mandibular Repositioning

Common name of OA:

Silencer[®] (Figure 11.17)



Figure 11.15 (a) Adjustable PM Positioner[©]. (Courtesy of Parker J, 2009. Used with permission.) (b) Intraoral view of adjustable PM Positioner[©]. (Courtesy of Parker J, 2009. Used with permission.)

Contact information:

604-576-0952

www.the-silencer.com

Type of OA design:

Mandibular Repositioning

Common name of OA:

Elastomeric[©] (Figure 11.18)

Contact information:

800-828-7626

www.greatlakesortho.com



Figure 11.16 OASYS[©]. (Courtesy of Abramson M, 2009. Used with permission.)



Figure 11.17 Silencer[®]. (Courtesy of Halstrom D, 2009. Used with permission.)

Type of OA design:
Mandibular repositioning
Common name of OA:
Herbst[®] (Figure 11.19)
Contact information:
800-786-8848

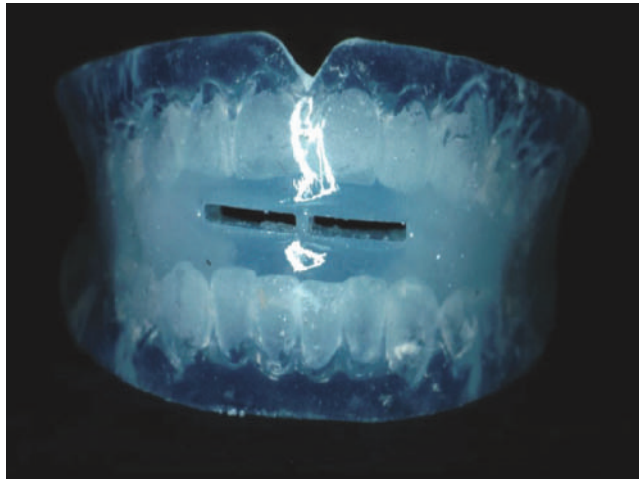


Figure 11.18 Elastomeric[®]. (Courtesy of Great Lakes Orthodontics, 2009. Used with permission.)



Figure 11.19 Thermoformed Herbst[©]. (Courtesy of Aleksy Kozlov, 2009. Used with permission.)

Brabant Dental Laboratory
Aleksy Kozlov (R&D Tech Supervisor)

Type of OA design:
Mandibular repositioning

Common name of OA:
Tongue Stabilizer Device (TSD) (Figures 11.20a and (11.20b)

Contact information:
800-828-7626
www.aveotsd.co.nz

Type of OA design:
Tongue retainer

Common name of OA:
SUAD[©] Figure (11.21)

Contact information:
888-447-6673
www.strongdental.com

Type of OA design:
Mandibular repositioning

Common name of OA:
Oral Positive Airway Pressure (OPAP) (Figure 11.22)

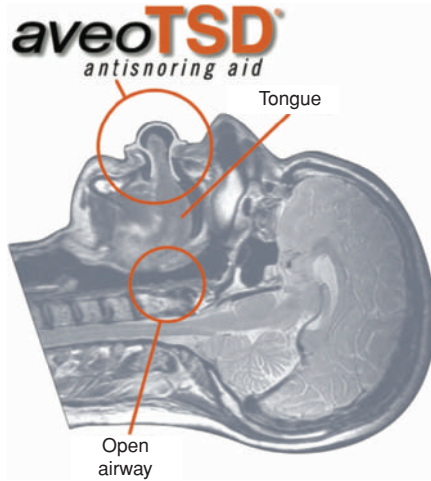
Contact information:
800-786-8848
Brabant Dental Laboratory
Aleksy Kozlov (R&D Tech Supervisor)

Type of OA design:
Positive airway pressure oral appliance

Common name of OA:
TAP[©] with PAP (Thornton Anterior Positioner) (Figure 11.23)



(a)



(b)

Figure 11.20 (a) aveoTSD[®]. (Courtesy of Innovative Health Technologies, 2009. Used with permission.) (b) aveoTSD[®] holding the tongue in advanced position. (Courtesy of Innovative Health Technologies, 2009. Used with permission.)



Figure 11.21 SUAD[®]. (Courtesy of Strong Dental Inc., 2009. Used with permission.)



Figure 11.22 An example of a PAP oral appliance interface. (© OPAP—Aleksy Kozlov, 2009. Used with permission.)

Contact information:

866-AMISNOR

www.amisleep.com

Type of OA design:

Mandibular repositioning combined with PAP



Figure 11.23 Combination of a TAP[©] oral appliance and PAP nasal pillows. (Courtesy of Airway Management Inc., 2009. Used with permission.)

CONCLUSION

The use of OA therapy for the management of OSA and snoring is recognized as an acceptable and oftentimes appropriate treatment option. The use of these devices by a dentist who is trained in sleep medicine provides an alternative for patients who desire an alternative to PAP therapy or surgery.

The dentist who has the desire to offer this service in his or her practice will experience a great deal of satisfaction when patients report that they are sleeping better and feel more alert and energetic.

The dentist who wishes to provide this treatment needs to embark on an educational mission similar to learning about any other specialty service in dentistry. The exciting aspect to this field is the impact that results from an overall health perspective may have when an OA has contributed to the improvement in a patient's sleep, overall health, and the quality of their life.

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Appendices

Appendix 1: Abbreviations for sleep medicine

The following is a listing of abbreviations that are used in sleep medicine and may be commonly encountered by the dentist:

AASM	American Academy of Sleep Medicine
ADD	attention-deficit disorder
ADHD	attention-deficit hyperactivity disorder
AHI	apnea-hypopnea index
APAP	autoadjusting positive airway pressure
BiPAP	bilevel positive airway pressure
BMI	body mass index
CHF	congestive heart failure
CPAP	continuous positive airway pressure
CRSD	circadian rhythm sleep disorders
CSA	central sleep apnea
DA	dopamine
<i>DSM</i>	<i>Diagnostic and Statistical Manual of Mental Disorders</i>
DSPD	delayed sleep phase disorder
ECG	electrocardiogram
EDS	excessive daytime sleepiness
EEG	electroencephalogram/electroencephalography
EMG	electromyogram
EOG	electrooculogram
ESS	Epworth Sleepiness Scale
FDA	Federal Drug Administration
GABA	γ -aminobutyric acid
GERD	gastroesophageal reflux disease
<i>ICSD</i>	<i>International Classification of Sleep Disorders</i>
<i>ICSD-2</i>	<i>International Classification of Sleep Disorders, Second Edition</i>

MRA	mandibular repositioning appliance
MRI	magnetic resonance imaging
MSLT	Multiple Sleep Latency Test
MWT	Maintenance of Wakefulness Test
NE	norepinephrine
NREM	nonrapid eye movement
NSF	National Sleep Foundation
OA	oral appliance
OSA	obstructive sleep apnea
PAP	positive airway pressure
PLMD	periodic limb movement disorder
PLMS	periodic limb movements during sleep (or PLM)
PSG	polysomnogram/polysomnography (sleep study)
REM	rapid eye movement
RERA	respiratory effort-related arousal
RLS	restless legs syndrome
RMMA	rhythmic masticatory muscle activity
SRBD	sleep-related breathing disorders
SRED	sleep-related eating disorder
SSRI	selective serotonin reuptake inhibitor
SSS	Stanford Sleepiness Scale
TMD	temporomandibular disorders
TMJ	temporomandibular joint
TRD	tongue-retaining device
UARS	upper airway resistance syndrome
UPPP	uvulopalatopharyngoplasty

Appendix 2: Glossary of terms for sleep medicine

The following is a listing of terms that are used in sleep medicine and may be commonly encountered by the dentist:

Advanced sleep phase syndrome: The sleep–wake times are earlier than the accepted times, which results in earlier bedtimes and earlier awakenings.

Apnea: Historically defined as the total cessation of breathing for 10 seconds or longer. In sleep medicine, apnea 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 stage of sleep, or from REM sleep to wakefulness.

Cataplexy: A finding oftentimes found in narcolepsy. A sudden loss of muscle tone associated with an emotional event such as laughter, surprise, excitement, or even anger.

Circadian rhythm: Oftentimes referred to as one’s internal clock. Associated with the light–dark cycle and occurs over about a 24-hour period.

Deep sleep/delta sleep: Oftentimes referred to as restorative sleep. This is associated with delta waves on an EEG. In the sleep study, this is known as stage N3 (may also be termed NREM stages 3 and 4). In some situations, deep sleep is associated with REM sleep because the ability to be awakened is more difficult.

Delayed sleep phase syndrome: The sleep–wake times are delayed, which may make 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.

- Epoch:** Refers to the amount of time that sleep is being recorded during a sleep study, typically for 20- or 30-second intervals.
- Fatigue:** The feeling of low energy levels, listlessness, or low motivation to be active with the inability to be sleepy or fall asleep.
- Fragmentation (sleep 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.
- Hypercapnia:** An elevation of CO₂ levels in the blood.
- Hypersomnia—excessive sleepiness or excessive daytime sleepiness (EDS):** The feeling of sleepiness at times when an individual should not be sleepy. Can also be associated with prolonged sleep.
- Hypnagogic hallucinations:** Images that may be visual, auditory, or tactile, which occur at the onset of sleep and may be related to the onset of REM.
- Hypnopomic 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. Oftentimes associated with narcolepsy and more common when cataplexy is present.
- 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.
- Insomnia:** The inability to initiate or maintain sleep and associated with sleep loss.
- K-complex:** A distinct sharp EEG waveform associated with 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. May be associated with excessive sleepiness.
- 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 used to test for the risk of narcolepsy and the onset of REM (stage R) within a 15-minute period.
- Nightmare:** An unpleasant/frightening dream that is associated with REM sleep.
- NREM (Non-REM) sleep:** Nonrapid eye movement sleep that is composed of three stages (N1, N2, and N3). These stages occur 4–6 times during a normal night's sleep.
- Parasomnia:** A disorder of sleep associated with arousal(s) or the transition between different sleep stages. Typically an event that disrupts sleep (sleepwalking, sleeptalking).
- Periodic breathing (Cheyne–Stokes respiration):** A type of breathing pattern that is a crescendo–decrescendo type of respiration.

- Periodic limb movement (PLM):** Also referred to as periodic limb movement disorder (PLMD) during a sleep study. An involuntary movement, usually of the leg, that occurs during sleep. May also involve flexion of the foot or extension of the large toe.
- Polysomnogram (PSG—also known as a sleep study):** Done during sleep (overnight) and records multiple physiologic events during sleep.
- Rapid eye movement (REM) sleep:** A period of sleep when the eyes typically move rapidly and the body is in a paralyzed state. Also known as *paradoxical sleep*.
- Respiratory disturbance index (RDI):** The apnea–hypopnea index (AHI) that also includes the RERAs.
- Respiratory effort-related arousals (RERAs):** 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.
- Sleep efficiency:** Usually measured in percent and is the amount of recorded time asleep to the amount of time in bed. The normal is considered to be 85%.
- Sleep hygiene:** The habits and lifestyle that contribute to good sleep, such as room temperature, noise, and the amount of light.
- Sleep latency:** The time between when the lights are turned off 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 speak. Considered to be associated with REM sleep and can be frightening, especially if breathing is also affected.
- Sleep–wake cycle:** The internal clock control of the sleep and wakefulness cycle (circadian rhythm).
- Slow-wave sleep:** Also referred to as delta sleep, stage N3 (NREM stages 3 and 4), or restorative sleep. This 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 an incomplete obstruction but may also be a symptom of sleep apnea.
- Total sleep time (TST):** The total amount of time actually spent in the sleep state (total of REM and NREM sleep).
- Upper airway resistance syndrome (UARS):** Events that do not seemingly meet the criteria for apnea and hypopnea but appear as RERAs on a sleep study. It appears similar to sleep apnea, but oftentimes the blood oxygen level does not drop.
- Zeitgeber:** (German for “time givers”) 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.

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