The Clinician's Handbook for Dental Sleep Medicine

SECOND EDITION

Steve Carstensen, DDS
Ken Berley, DDS, JD

QUINTESSENCE PUBLISHING

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Dedications

The impact of dentistry in health care is growing rapidly. I am dedicated to supporting the health care professionals who choose to push beyond their basic education to learn more about the impact they can have on community health. Those brave enough to say "yes" to opportunities can make a true difference in the lives of those who trust them. This approach would not be possible without the mentors who provided me with opportunities to grow—Dr L.D. Pankey, Dr Keith Thornton, Midge Carstensen, RDH, and countless others who have participated in reducing barriers to health at all stages of life. I hope every reader of this book is as fortunate as I am to have such strong support.

—Steve Carstensen, DDS

I would like to dedicate this book to my father, James A. Berley, who had undiagnosed OSA and died as a result of a stroke in the middle of the night. His death led directly to this book and changed my life. Also, I would like to dedicate this book to my wife, Patty Berley, RDA, who has stood by me through thick and thin, even when I was unworthy of her love.

—Ken Berley, DDS, JD

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Preface

Life is short. Before you know it, you will be looking at your professional life in the rear-view mirror trying to figure out how you spent the time you were given. How did it go so fast? As Benjamin Franklin said, "You may delay, but time will not." Now, in the sunset of my professional career, the thing that I am most proud of is that I committed to the screening and treatment of sleep-disordered breathing (SDB) early in my professional career. I sincerely derive comfort from the fact that my professional efforts have resulted in an improved quality of life for thousands of my patients. As I have said in many of my lectures, on a good day as a dentist I may be able to save a tooth or improve a smile. On a good day as a dental sleep medicine (DSM) practitioner, I may be able to save a patient's life. I am very proud that I have helped so many patients sleep. While the dental health of our patients is vitally important, nothing is more important than air. Without that, life doesn't exist.

With the rise of DSM, dentists are in a unique position to modify the anatomy of the upper airway and improve a patient's oxygen levels during sleep. The simple act of advancing or supporting the mandible during sleep can change the lives of millions of patients who are struggling. When we advance a patient's mandible, nocturnal arousals and sleep fragmentations are diminished because the airway is patent. The patient enjoys more restful sleep and an improved quality of life.

SDB and obstructive sleep apnea (OSA), which is a subset of SDB, are true epidemics in this country. Simply defined, SDB is difficulty maintaining a patent airway during sleep. The symptoms range from simple snoring to prolonged and complete closure of the patient's airway. The patient's tongue and soft palate are typically the culprits responsible for nocturnal airway resistance. But when we advance the mandible, the tongue and hyoid bone are placed under tension, which decreases resistance and reduces the likelihood of complete or partial airway closure.

Epidemiologists estimate that more than 200 million Americans snore nightly and at least 52 million have clinically significant OSA. That means that up to 50% of the adults and 10% of the children you see each day in your dental office likely have some degree of SDB. SDB and OSA are directly associated with hypertension, coronary artery disease, stroke, diabetes mellitus, dementia, and early death. SDB results in the activation of the sympathetic nervous system, resulting in the release of adrenaline and cortisol. When released as a result of the patient's struggle to breathe, adrenaline initiates a "fight or flight" sympathetic response. During sleep, our body is supposed to be resting and repairing, not fighting for oxygen. Opening a patient's airway with a mandibular advancement device (MAD) can change their life. Nothing that you do as a health care professional will be more impactful for your patients.

DSM was introduced approximately 40 years ago in an effort to advance the use of MADs as a treatment alternative to continuous positive airway pressure (CPAP). Now, after 40 years of growth and growing pains, oral appliance therapy (OAT) is an accepted alternative to CPAP and is the first-line treatment for SDB/OSA, with reimbursement available under most medical insurance policies and Medicare. While CPAP remains the first choice of treatment for the majority of physicians, the percentage of physicians who regularly recommend OAT for their patients with SDB/OSA is growing rapidly. This shift toward OAT as the therapy of choice has resulted in an explosion in the field of DSM. Now, over 30% of all dentists provide some level of OAT to their patients, and the number of full-time DSM practitioners is growing each year. With the acceptance of DSM as an effective treatment and the expansion of medical insurance reimbursement, the addition of DSM into a general dental practice can result in significant financial gain.

There are a number of dental professional service organizations that foster and support DSM, with the American Academy of Dental Sleep Medicine (AADSM) being the largest. All of the service organizations provide various levels of training and credentialing; however, the AADSM offers diplomate certification, which is generally recognized as the highest level of training and credentialing available. With that said, readers should be aware that no significant financial investment is needed in order to provide OAT and treat OSA. You do not have to enroll in an expensive program or purchase costly equipment to treat your patients. After reading and implementing the information and techniques discussed in this handbook, you will have the knowledge and techniques necessary to successfully practice DSM. Do not be deceived into thinking that you need to spend thousands of dollars to get your DSM practice started. That is not necessary. All you need is information and a true desire to help your patients live better lives.

You have my congratulations on your decision to treat your patients' SDB; you will not regret adding DSM to your practice. Thank you for allowing Steve and me the opportunity to instruct, assist, and inspire you in your pledge to treat your OSA patients. Please don't get discouraged as you begin. Find the CPAP failures that are currently experienced by patients in your practice and start by treating them. Learn and repeat!

Respectfully,
Ken Berley, *DDS, JD*

Acknowledgments

We would like to thank the team at Quintessence Publishing and Kristen Clark, our editor, for their guidance and assistance in the development of this handbook. Also, a big thank you to our families and friends who have tolerated and supported us throughout this process. Your understanding has been invaluable.

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Innocent sleep. Sleep that
soothes away all our worries.
Sleep that puts each day to rest.
Sleep that relieves the weary
laborer and heals hurt minds.
Sleep, the main course in life's
feast, and the most nourishing.

—*William Shakespeare, Macbeth*

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Clinical Guide for the Practice of Dental Sleep Medicine

The Sleep Deprivation Epidemic

An old Irish proverb says, "A good laugh and a long sleep are the best cures in the doctor's book." It is easy to champion the physical, mental, and social benefits of a good night's sleep. Sleep is mandatory for life. Humans can live approximately 4 days without water and 40 days without food but cease to function normally after missing a single night of sleep. Missing sleep for 10 days can be lethal. Even the early Greeks and Romans worshiped Hypnos and Somnus, their gods of sleep. We are all aware that every human must achieve a certain quality and quantity of sleep to function normally, but sadly, millions of individuals across the globe are unable to achieve a good night's sleep. For these individuals, life can be a real struggle. A simple task can turn into an adventure without adequate sleep. Despite all the medical and social advances of modern society, good sleep has never been harder to achieve.

We struggle with sleep for many reasons. People face various social, environmental, and physiologic barriers when attempting to sleep. Frequently, sleep quality is sabotaged by lifestyle decisions—the self-inflicted consequences of daily choices. Attempting to sleep in a noisy, hot, or bright environment is common. Too many people live sedentary lives with insufficient exercise, and sleeping is much more difficult if we are not physically tired. A great American weekend is now defined by watching football on the couch

or binge-watching the latest Netflix release. Alcohol consumption, which significantly exacerbates sleep-disordered breathing, is also at an all-time high. We are hyperconnected with a growing array of computer-driven screens and glowing devices. The blue light emitted from many screens stimulates serotonin production and contributes to insomnia. Our technologic advances have given us the ability to work anywhere on the face of the planet at any time during the day or night, and we can stay in constant contact with anyone in the world. The health-conscious person striving for a good night's sleep must be very disciplined in light of this sensory onslaught.

In addition to the societal and entertainment choices that rob us of sleep, our ability to sleep is directly affected by certain physical characteristics and anatomical deficiencies. First and foremost, the human ability for complex vocalization and speech has created an anatomical weakness—a smaller maxillofacial complex and subsequently a decreased airway diameter. The airway from the hard palate to the top of the trachea is collapsible, with collapse typically manifested during the deeper levels of sleep. If the airway is restricted, or collapses completely, the increased respiratory effort required to breathe can lead to arousals and other physical consequences. Airflow is restricted, and blood oxygen/carbon dioxide levels become unbalanced. The repeated cycle of airway restriction/collapse and recovery to resume breathing has many names. *Sleep-related breathing disorder* (SRBD) is the general term, with *obstructive sleep apnea* (OSA) being the most consequential subset.

In addition to the anatomical component of OSA, Western civilization has adopted a fast food and microwave meal–style of living. Poor diet has resulted in rampant obesity in the United States, and OSA is closely linked to obesity and diabetes mellitus. These diseases are almost inseparable and have long-term health consequences. As patients become larger, fat deposits in the base of the tongue and the lateral walls of the airway are enlarged, reducing the cross-sectional area of the oropharyngeal lumen and making it more likely to collapse. Therefore, obesity greatly contributes to or exacerbates OSA. According to the Centers for Disease Control and Prevention (CDC), approximately 65% of Americans are overweight or obese. In many states, more than 35% of the population is obese, with the percentages rising each year (Fig 1-1). Obesity hypoventilation syndrome (OHS) is a severe disease state, and over 90% of patients diagnosed with it have OSA.[2]

Finally, OSA appears to have some roots in the Western practice of bottle-feeding and the use of pacifiers and sippy cups. Nonproductive suckling may result in craniofacial respiratory complex collapse (CFRC) and increased dental crowding in children, exacerbating childhood sleep disorders.[3]

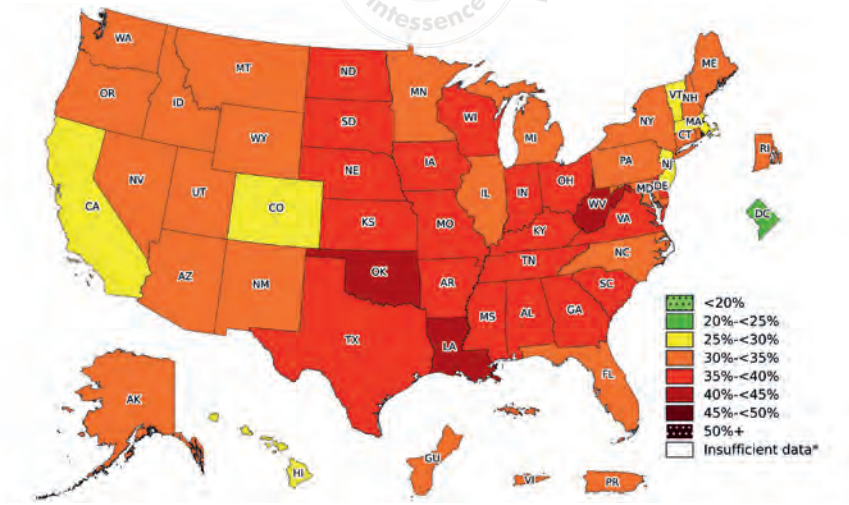

Fig 1-1 2022 obesity map. (Reprinted with permission from the CDC Behavioral Risk Factor Surveillance System.[1])

This coalition of factors, including a smaller orofacial complex, increased BMI, an aging population, and decreased levels of exercise, has resulted in an explosion of OSA in the United States. Often, individuals with OSA are unaware that they have difficulty breathing during sleep, and it is most commonly recognized as a problem by family members or bed partners who observe the individual during obstructive episodes. Individuals who sleep alone may be unaware of the condition because no one is present to poke them in the ribs. OSA patients may seek medical care for symptoms of fatigue or daytime sleepiness, but not all patients exhibit excessive daytime sleepiness (EDS). Sadly, OSA may remain undetected for years or even decades because patients become conditioned to the daytime sleepiness and fatigue associated with significant levels of sleep disturbance.

Lack of adequate air leads to a cascade of devastating physical and emotional consequences. Restricted airway function affects our ability to sleep and can give rise to insomnia, an altered arousal threshold, asphyxia, hypoxemia, hypercapnia, and sleep fragmentation. Increased respiratory effort and airway collapse can lead to EDS and contribute to a host of comorbid diseases.

The Rise Of Dental Sleep Medicine

This is an exciting time to be a dentist. The field of dental sleep medicine (DSM) is exploding. This is primarily because the majority of individuals with sleep disorders do not present to physicians for treatment of their sleep issues.[4] Instead, people typically seek medical care for comorbid diseases that are directly associated with SRBDs, in particular, hypertension, fatigue, stroke, cardiovascular disease, and diabetes mellitus. Unfortunately, most physicians do not screen for SRDBs, and the precipitating factors contributing to the patient's chief complaint frequently go undiagnosed.[5] However, many patients suffering from SRBDs also seek routine dental care, which provides knowledgeable dentists with a screening opportunity. Dentists who are properly trained to recognize the signs and symptoms of SRBDs can potentially provide life-saving referrals and oral appliance therapy (OAT) to sleep-deprived patients. Patients with SRBDs exhibit identifiable intraoral signs and symptoms, as well as physical and social symptoms.

DSM was founded on the principle that the mandible can be moved and supported to maintain an open airway during sleep. The tongue is attached anteriorly to the genial tubercles on the lingual aspect, and if the mandible is protruded with a mandibular advancement device (MAD), the tongue is advanced. This opens the airway and minimizes airway restriction. MADs work in the same manner as the head tilt, chin lift, jaw thrust maneuver used to open the airway in CPR. The simple act of holding the mandible in a stable or slightly protruded position during sleep may be adequate to prevent or mitigate the physiologic consequences of airway obstruction and resultant nocturnal hypoxemia. There is an ever-increasing number of MADs being cleared by the FDA to treat SRBDs, including snoring and OSA, and properly trained dentists are integral partners in the treatment of SRBDs.

Although still in its infancy, DSM is the fastest-growing discipline in dentistry. Screening and treating patients with SRBDs can greatly improve their health and generate a significant source of income for your dental practice.

Terminology

This section explains some useful terminology in the field of DSM.

Apnea

Apnea literally means "without air." An apnea is identified during a sleep study when the patient's airflow peak signal drops by ≥ 90% compared to the pre-event baseline for at least 10 seconds as measured using an oronasal

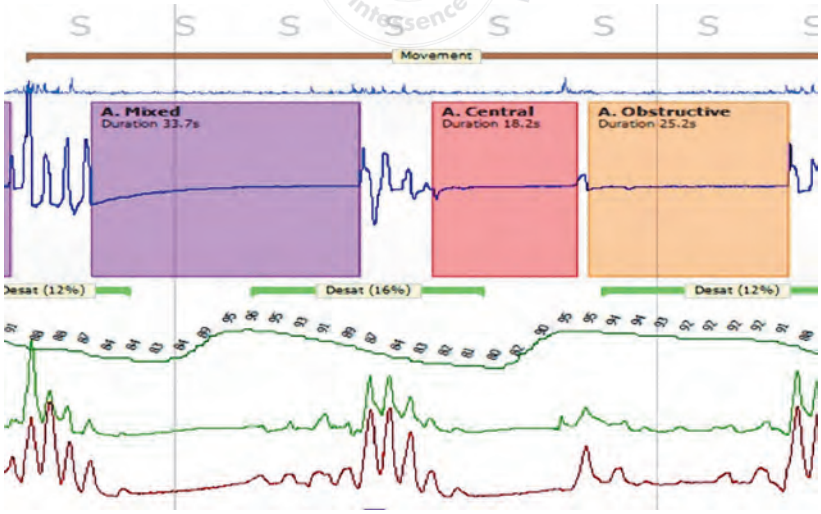

Fig 1-2 Screenshot of a home sleep apnea test (HSAT) indicating the different types of apneas. While the patient is in the supine position (S), the first event is scored as a mixed event because the respiratory effort belt signals are flat (no effort) for more than half of the event. Recovery breaths follow the mixed apnea, where the patient is gasping for breath. Then the next two events are scored as a central for 18.2 seconds, a small breath, then an obstructive event where the patient is trying to breathe but no air is entering the lungs. Notice that a desaturation follows each of the events.

thermal signal, positive airway pressure (PAP) device flow, or an alternative apnea sensor. There is no requirement for desaturation or arousal. Apneas can be classified as obstructive, central, or mixed, depending on the patient's respiratory effort during the event. If the patient is trying to breathe and cannot, the event is classified as obstructive. If the patient is not trying to breathe, the event is designated as central apnea. A mixed apnea is a combination of obstructive and central apnea, where the event is more central than obstructive (Fig 1-2).

Arousal

An arousal is a sudden change from a deeper level of sleep to a lighter level of sleep. Patients usually do not awaken and are unaware of the arousal, but daytime sleepiness and fatigue are common symptoms of frequent arousals.

Arousals are classified as respiratory arousals and environmental arousals. Respiratory arousals are caused by any respiratory event and are directly related to the patient's effort to breathe. As oxygen levels decrease and carbon dioxide levels rise during an apnea or hypopnea event, chemosensors in the brain arouse motor pathways to restore airway function and normal

respiration. Treatment of respiratory arousals can improve daytime symptoms. Environmental arousals occur as a result of non-respiratory stimuli. Many things can cause a patient to arouse during sleep. Anything from noise from a TV left on at night to the temperature in the bedroom or air quality and light control can be associated with a disruptive sleep environment.

Home sleep apnea tests

Frequently, home sleep apnea tests (HSATs) are used to diagnose OSA. However, HSATs typically cannot fully measure the quality and quantity of sleep. HSATs measure different bodily metrics, such as respiratory activity and breathing patterns. Common metrics measured during an HSAT include the following:

- Respiratory activity, breathing patterns, and chest motion
- Heart rate and blood oxygen level
- Actigraphy, or nocturnal movement and motor activity
- Sleep position and position changes
- Snoring events and intensity

Hypopnea

Hypopnea is a drop in peak signal excursion of at least 30% compared to the pre-event baseline for at least 10 seconds measured using nasal pressure (recommended sensor), PAP device flow, or an alternative hypopnea sensor. Additionally, to qualify as a hypopnea, the event must involve at least 3% oxygen desaturation from the pre-event baseline OR the event must be associated with a cortical electroencephalogram (EEG) arousal[6] (Fig 1-3).

Alternatively, Centers for Medicaid and Medicare Services (CMS) defines a hypopnea as a drop in peak signal excursion by at least 30% compared to the pre-event baseline for at least 10 seconds measured using nasal pressure (recommended sensor), PAP device flow, or an alternative hypopnea sensor AND oxygen desaturation of at least 4% from the pre-event baseline. Scoring hypopnea using a 4% desaturation is a Medicare requirement. When treating a Medicare patient, make sure that the appropriate scoring parameters were used by the sleep lab or the patient will not qualify for benefits under Medicare.

Polysomnography

A polysomnogram (PSG), also called an in-lab sleep study, is a continuous recording of specific physiologic markers and variables over a full night of

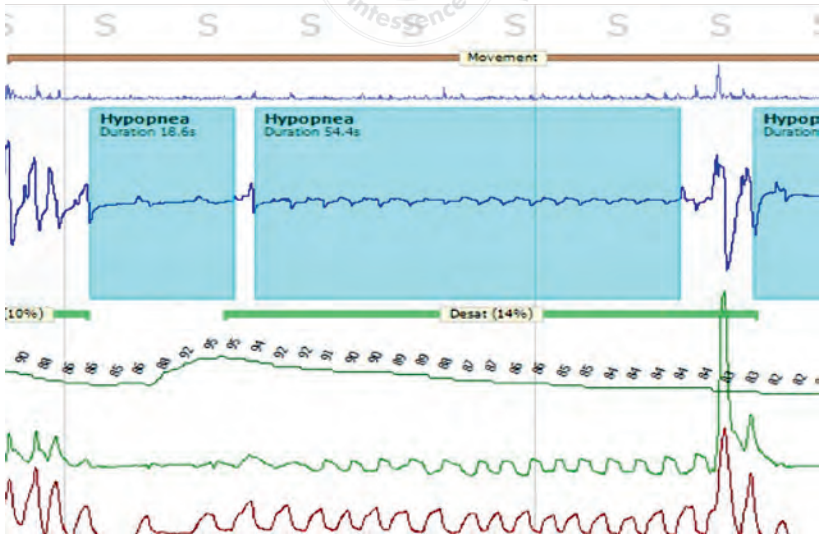

Fig 1-3 Screenshot of HSAT indicating hypopneas. Notice that the patient's airflow is not a flat line. Air is entering the lungs, but the volume is reduced to the point that the patient's blood SaO_2 is not maintained. The decreased airflow results in a 14% desaturation. At all times during the event, the patient is attempting to breathe but is unable to adequately maintain oxygen levels.

sleep that helps to identify and diagnose various sleep disorders (Fig 1-4). Typically, a PSG records the following parameters:

- Brainwaves (via EEG)
- Blood oxygen levels
- Eye movements (via electrooculogram [EOG])
- Muscle tone (via electromyogram [EMG])
- Respiratory activity, breathing patterns, and chest motion (via nasal flow sensor and effort belts around the chest)
- Heart rate (via electrocardiogram [ECG])
- Leg movements (via EMG)
- Snoring
- Sleep position

Respiratory disturbance index

The respiratory disturbance index (RDI) is calculated as the number of respiratory events (apneas, hypopneas, and respiratory effort–related arousals [RERAs]) divided by the number of hours of sleep documented during a PSG study.

Fig 1-4 Patient during a PSG.

Respiratory effort–related arousal

Respiratory effort–related arousal (RERA) is not always scored during a sleep test. It is defined as a sequence of breaths lasting at least 10 seconds and characterized by increasing respiratory effort or by flattening of the inspiratory portion of the flow signal, leading to an arousal from sleep. The sequence of breaths does not include apneas or hypopneas. Because the definition of a hypopnea has never been consistent and is more frequently revised than other sleep breathing parameters, RERA is not typically used to describe SRBDs but may be useful for informing clinicians about sleep fragmentation.

Snoring

Snoring is traditionally defined as breathing during sleep with a rough, hoarse noise due to vibration of the soft palate and tongue. It is helpful to separate patients who simply make a snoring noise from patients whose snoring results in a RERA. For the purposes of this text, the authors refer to snoring as any respiratory-related noise made during sleep that does not result in an arousal. Respiratory noises and events that result in an arousal, but do not qualify as an apnea or hypopnea, are referred to as a RERA. It should be noted that loud snoring is the most common symptom of a patient with OSA.

Overview of Sleep-Related Breathing Disorders

SRBD, also known as *sleep-disordered breathing* (SDB), is a collection of conditions or disorders where patients suffer from the consequences of restricted breathing and/or reduced oxygenation during sleep. Although OSA is generally referred to as SRBD, SRBD is a much broader term that includes a spectrum of breathing anomalies, ranging from chronic or habitual snoring (resulting in airflow limitations) to upper airway resistance syndrome (UARS), OSA, central and complex apnea, and in some cases, Cheyne-Stokes respiration and obesity hypoventilation syndrome. Although OHS is not a true sleep disorder, patients with very severe OSA who are morbidly obese frequently suffer from daytime hypoventilation, which can be mitigated with the use of a single-arch temporomandibular joint (TMJ) splint that encourages the patient to function with their mandible protruded during the daytime. The amount of protrusion built into the daytime splint varies with the severity of the patient's condition. Patients with severe OHS use continuous positive airway pressure (CPAP) and a MAD during sleep.

SRBD symptoms routinely become more severe and complex with age, and the consequences of the resultant sleep fragmentation and oxygen desaturation can be deadly. SRBDs make maintaining oxygenation during sleep difficult as a result of repeated upper airway restriction and resistance during sleep that causes recurrent nocturnal asphyxia, fragmented sleep, fatigue, daytime sleepiness, major fluctuations in blood pressure, and increased sympathetic nervous system activity.[7] An aging population and skyrocketing obesity further contribute to SRBDs. As we age, the airway often becomes smaller, due to increased fat deposits, and more collapsible, secondary to hormonal changes. A patient who has been diagnosed with OSA is at increased risk for hypertension, stroke, diabetes mellitus, and heart attack, as well as early mortality. The physiologic changes associated with SRBDs can be devastating to a patient's ability to stay oxygenated during sleep. This is particularly impactful when a patient has a compromised immune system.

The various airway conditions associated with SRBDs include, but are not limited to, respiratory noise, snoring, flow limitations, RERAs, UARS, hypopneas, OSA, OSA syndrome (OSAS), mixed sleep apnea, and central sleep apnea. The risk factors and underlying pathophysiologic mechanisms of these disorders have both overlapping and unique features.

Figure 1-5 shows the continuum of sleep disorders, progressing from a baby making a "cute noise" when sleeping all the way to apneas, where the airway is totally obstructed. Each level represents a higher degree of collapse with increasingly serious consequences. Although SRBDs cannot be accurately represented as a true progression (snoring does not inevitably result in OSA), it is important to see that every diagnosis lies within a larger scope of poor airway function.

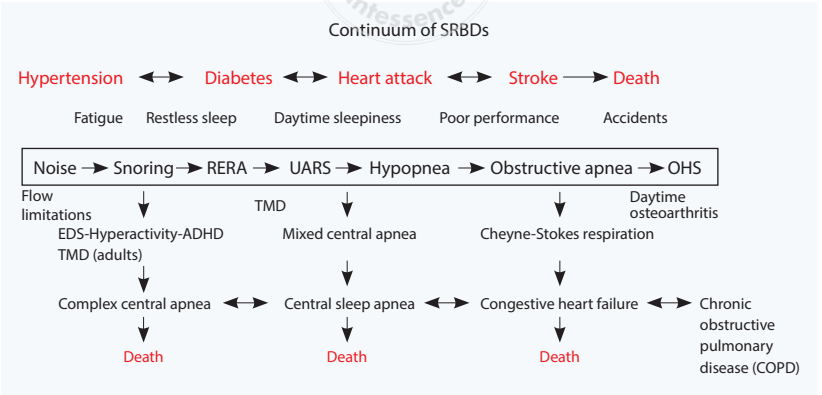

Fig 1-5 Continuum of sleep disorders and related conditions.

Once a patient starts experiencing hypopneas and apneas, medical insurance may provide benefits. The last stage of SRBDs is when patients have difficulty maintaining an open airway during the daytime. OHS is common in morbidly obese patients with BMIs greater than 45. Therapies for OHS may include a TMJ mandibular advancement (Gelb) splint, portable oxygen, and weight loss therapy. A daytime advancement splint is simply one of the many tools which can help these patients stay adequately oxygenated during the day. The ultimate goal is weight loss, which will likely include bariatric surgery. OHS involves autonomic nervous system dysfunction and is not well managed in the dental office. Patients with OHS are commonly treated with multiple therapies by collaborating health care providers.

Obstructive sleep apnea

Common symptoms of OSA include the following[8]:

- Unexplained daytime sleepiness
- Loud snoring with periods of silence followed by gasps as breathing resumes
- Attention deficit and/or hyperactivity in children
- Trouble concentrating; mood changes such as irritability, anxiety, treatment-resistant depression, and forgetfulness
- Temporomandibular disorder symptoms, sleep bruxism
- Decreased sex drive, sexual dysfunction, and heavy night sweats
- Restless leg syndrome/periodic limb movements
- Restless or fragmented sleep and lack of dreams

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