CDA JOURNAL, VOL 40, Nº2

Neurology of Sleep and Sleep-Related Breathing Disorders and Their Relationships to Sleep Bruxism

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ABSTRACT Conditions that affect sleep can impact overall health. More than 70 million Americans suffer from problems with sleep. The purpose of this article is to provide the basic science of sleep physiology and how it relates to disorders that are pertinent to dentistry. Concepts are presented that explain airway dynamics and how the jaw and tongue influence airway obstruction. Additionally, explanation is given on an association between temporomandibular jaw dysfunction and bruxism during sleep.

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Relative to other disciplines, sleep medicine is in its infancy. For years the delay in development of this field stemmed primarily from the limited technology available to assess the sleeping process.[1] As technology developed and improved, the ability to understand the governing mechanisms of sleep in a meaningful fashion became possible. Over the past 50 years, tremendous strides have occurred that led to the current knowledge and establishment of the field of sleep disorders medicine. Unlike other areas of the medical field, sleep medicine focuses on a physiologic process and not an organ system, and it integrates many disciplines under a single umbrella. The American Academy of Dental Sleep Medicine (AADSM) was originally founded as the Sleep Disorders Dental Society in 1991 and has grown extensively in the past 20 years. Now dentists can become involved, and, to assure they properly address sleep medicine, there now exists a dental sleep medicine board that provides guidelines for dentists who wish to credential in this area of medicine.[2,3]

There is a full spectrum of conditions that affect sleep that have a far-reaching impact on patients with a variety of conditions. The purpose of this article is to provide the basic science of sleep physiology and how it relates to disorders that are pertinent to dentistry. Concepts are presented and build toward a knowledge base that will bring the reader to understand not only how modification of the jaw relationship can enhance breathing but also explains how abnormal conditions during sleep can be associated with sleep bruxism (SB) and TMJ dysfunction (TMD).

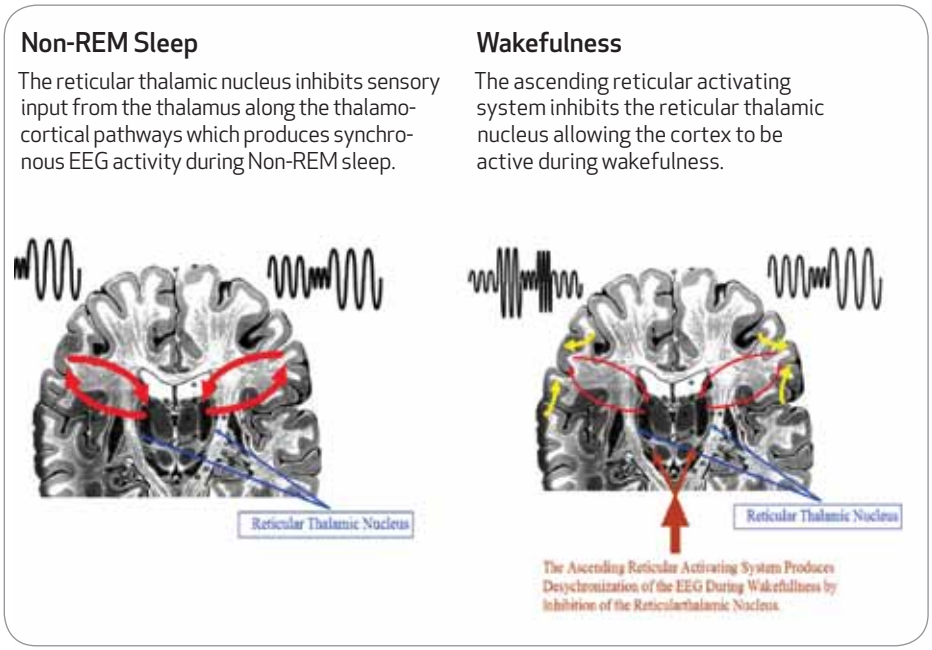

FIGURE 1. In non-REM sleep the cortex is not processing information from sensory input and the two hemispheres display EEG that is synchronized, meaning that the activity is similar on both sides of the head. This synchronization is governed by the reticular thalamic nucleus and is a key feature of non-REM sleep. During wakefulness the ascending reticular activating system stimulates the thalamus in regions that turn down the filtering mechanism of the reticular thalamic nucleus. This allows sensory input to pass through the thalamus, onto the cortex. In so doing each cortical region performs specific processing and this result in changes in changes in the EEG that are different in each region, thus desynchronizing the EEG signal.

Basic Sleep Physiology and the Stages of Sleep

Sleep is divided into two main categories or stages: REM and non-REM. REM refers to rapid-eye movement sleep, but first it would be appropriate to describe non-REM sleep, since it constitutes the majority of the sleeping process. Non-REM sleep is divided into three progressively deeper stages, referred to as N1, N2, and N3.[4] N3 is considered the deepest, most restorative level of sleep and is also referred to as slow wave sleep.[5,6] During non-REM sleep, the brain utilizes a filtering mechanism within the deep brain structures in a region known as the reticular thalamic nucleus that blocks the sensory input coming from throughout the body from reaching the cerebral cortex. This filtering mechanism involves gamma aminobuteric acid (GABA), an inhibitory neurotransmitter. During non-REM sleep there is a global filtering of the incoming sensory signals from throughout the body at the thalamic level.[7] The filtering is most robust during stage N3 and it is during this portion of sleep that certain hormonal changes take place. For example, growth hormone secretion achieves its highest level during stage N3 sleep. It is now recognized that the greatest restoration of the body occurs during non-REM sleep, and most specifically during stage N3[5,6] (**FIGURE 1**).

REM sleep has very different characteristics. During REM, an individual is actively dreaming or hallucinating and the cortex acts almost as though it is awake. There are three neurotransmitters that play an important role in REM sleep. These are acetylcholine (Ach), norepinephrine (NE) and serotonin (5HT). In the brainstem, the dorsal raphe nucleus, which utilizes 5HT and the locus coeruleus that utilizes NE, are actively firing while awake and during non-REM sleep. These two nuclei, through 5HT and NE, suppress a region of the brainstem known as the pedunculopontine tegmental (PPT) nucleus which, when not suppressed, sends output to both the lower brainstem and to the thalamus with fibers that release Ach as its transmitter.

REM sleep occurs when the locus coeruleus and the dorsal raphie nucleus stop their inhibitory activity over the PPT nucleus. In effect, this results in activation of the two Ach pathways of the PPT nucleus. Pathway 1 ascends into the thalamus as part of the ascending reticular activating system having an activating affect on the cortex creating hallucinatory phenomena during REM sleep. Pathway 2 descends to the bottom of the brainstem (medulla) ending at the reticulo-spinal track nucleus. The reticulo-spinal track consists of neurons that descend down into the spinal cord and release glycine as a neurotransmitter. This causes post-synaptic inhibition on the motor neurons that leave the spinal cord, resulting in paralysis of the body during REM sleep. Therefore, during REM sleep the body becomes paralyzed while the cortex is very actively hallucinating. This muscle paralysis prevents the body from acting out on the impulses generated from the brain's cortex during REM sleep.[6,8-10] This change of muscle tone activity that occurs during REM sleep has significant ramifications on aspects of breathing and influences the degree of obstructive breathing as will be outlined later below[11] (**FIGURE 2**).

During the day, a person maintains a focused attention that shifts from one object or thought to another. This capability of shifting our concentration in a focused fashion is provided by the brain's ability to filter out irrelevant stimuli.[12-14] As the brain fatigues there is a breakdown in the brain's ability to filter out irrelevant stimuli. This results in inattention and distractibility and occurs with sleep deprivation.[15] The part of the brain that provides the filtration and focusing of attention is also within the thalamus.

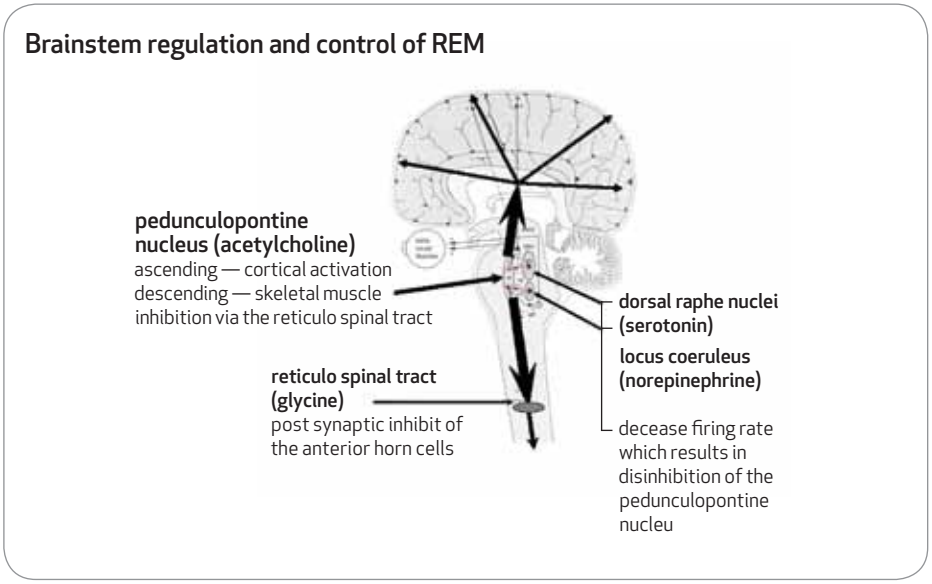

FIGURE 2. Diagram shows the brainstem regions responsible for the regulation of REM sleep.

As described above, during non-REM sleep, the thalamus provides a global filtration of incoming sensory stimuli. As we become more sleep deprived this filtering effect is enhanced in order to achieve sufficient filtration of sensory input necessary for us to fall off to sleep.

While wide awake maintaining a focused attention on a complex task the brain is functioning in a completely opposite fashion as to what occurs during non-REM sleep, during which there is no specific cortical processing taking place and the cortex is globally under inhibition by the reticular thalamic nucleus. Trying to maintain focused attention while sleep deprived may prove challenging because of a globally enhanced degree of filtering exhibited by the reticular thalamic nucleus. Frequently, to maintain wakefulness when sleep deprived a person increases their stimulation level to stay awake. This is achieved by changes in behavior such as fidgeting, shifting in a chair, getting up to walk around etc. In essence they become hyperactive as a compensation to override the enhanced sleep drive of the reticular thalamic nucleus.[16-18]

The brain cycles through stages of N1, N2, N3, and REM over a 90-minute period, with REM sleep occurring approximately every 90 minutes throughout the night. Each of these 90-minute segments is referred to as a sleep cycle. As the brain progresses through these sleep cycles there is a decreased amount of N3 sleep and an increase in the amount of REM sleep.[4,19] As a result, the last portion of the night typically has no N3 or slow wave sleep and the largest portion of REM sleep of the night. Therefore, most of REM sleep occurs during the second-half of the night (**FIGURE 3**).

Fragmentation of Sleep and Daytime Consequences

Repetitive disruptions in sleep continuity lowers the restorative properties that sleep is intended to provide. Increased sleepiness throughout the day results from a heightened level of filtering from the reticular thalamic nucleus as it attempts to put the brain to sleep.[7] There are several common conditions that disrupt the continuity of sleep and are important to understand in order to identify common sleep disturbances. One common condition referred to as periodic limb movements of sleep or PLMS. This phenomena frequently occurs in patients who have restless leg syndrome (RLS) but can also be seen in people without RLS.[6,20] A more elaborate discussion of this condition is outside the scope of this article. Another common condition that is important for dentists to recognize relates to obstructive breathing during sleep. The hallmark condition in this category is referred to as obstructive sleep apnea (OSA).[6,21,22]

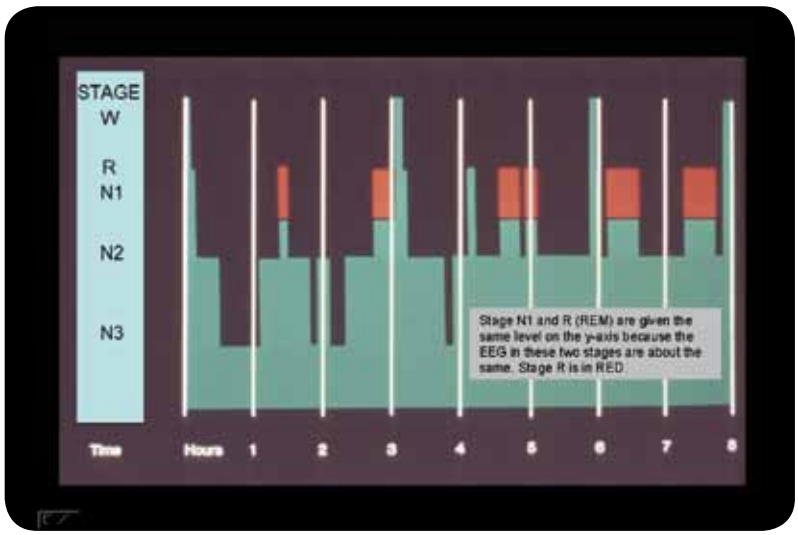

FIGURE 3. Diagram shows a typical hypnogram of the different sleep stages that occur throughout the night.

The Principles of Obstructive Breathing During Sleep

To understand obstructive breathing, first refer to a principle of the physics known as the Bernoulli Effect. As flow goes through a space, there is a negative pressure or vacuum that develops within that space. If the space becomes narrower and the flow volume is maintained constant then there is an increase in the degree of negative pressure or vacuum. An example of this phenomena is demonstrated by flow of water in the shower where the shower curtain is pulled in toward the flow of water if the water stream is brisk. If the shower curtain is pushed away from the water, expanding the space, this lowers the amount of negative pressure within the space. This same principle occurs in the back of the throat. The tongue attaches to the inner aspect of the mandible and then travels posterior toward the pharyngeal wall and also curves upward and forward into the oral cavity. As the mandibular muscles relax and the mandible retrudes, the base of the tongue moves with it and encroaches on the posterior pharyngeal wall. As this occurs the posterior pharynx narrows and this results in an increase in negative pressure during breathing. When an individual goes to sleep it is normal for there to be an increase in the magnitude of negative pressure of the posterior pharyngeal airway. Typically, an awake person generates approximately –2 to –5 cm H_2O pressure in this region. When they fall off to sleep, generally it increases to –5 to –8 cm H_2O. Under abnormal conditions, such as with patients who have OSA, when the individual falls asleep, the pharyngeal space narrows to a degree where the negative pressures can range from –15 to –30 cm H_2O and, when severe, can even generate levels beyond -100 cm H_2O.[6,23-28]

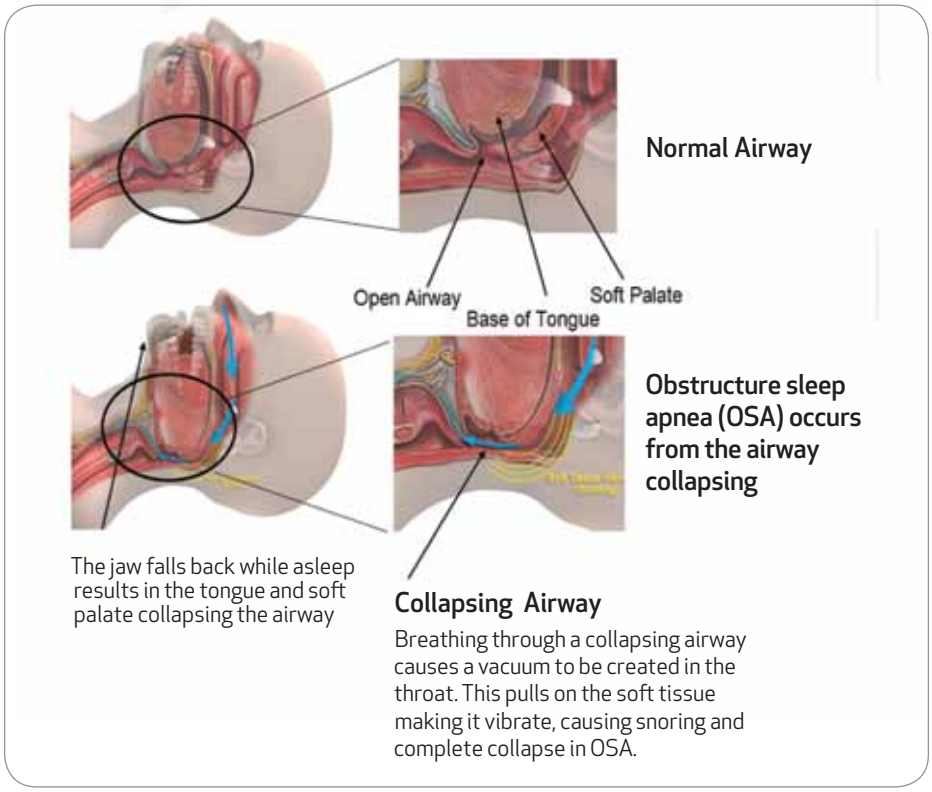

FIGURE 4. Diagram demonstrating the changes in the airway structure during sleep and the resulting effects of breathing with the development of negative prssure within the pharynx.

Abnormal degrees of negative pressure in the posterior pharynx causes the tissue of the posterior pharynx to vibrate as it is pulled downward. This vibration causes snoring. Depending on the muscle tone of the entire pharyngeal region, which is influenced also by the various sleep stages and other factors, the airway may collapse from this negative pressure vacuum. In some patients the pharyngeal walls have more resistance toward collapse where –50 cm H_2O does not cause complete collapse of the airway. In other cases, the airway may be very collapsible and completely collapse at lower magnitudes of negative pressure.[6,25,26]

OSA is classically described when the airway completely collapses, cutting off flow while the chest muscles continue to attempt respirations. This type of phenomena can clearly be seen on a sleep study. From a practical standpoint, there is a spectrum of the degree of obstructive events with complete obstruction on one end and normal breathing on the other with various degrees of partial blockage in between. With partial blockage, airflow continues to be maintained but may be decreased without pauses in breathing. Frequently when this occurs there may even be a reduction in the blood oxygen level resulting from the decreased amount of air flow. However, many experts contend that a reduction in the oxygen level is not necessary for an event to be significant if there is a disruption in sleep continuity from the partial obstruction. These partial obstructions lasting 10 seconds or longer are known as hypopneas. Respiratory events that occur during an individual's sleep, including both apneas and hypopneas are reported using the apnea hypopnea index (AHI) that refers to the averaged, hourly frequency of these types of events. These events are reported together in the AHI because research has not demonstrated a distinction in the detrimental effects produced by severe hypopneas versus severe apneas (**FIGURE 4**).

The Upper Airway Resistance Syndrome

Many subtle respiratory events from partial airway obstruction occur and are demonstrated with an increased magnitude of negative pressure with each breath, leading to disrupted sleep. Frequently, these subtle events go untabulated by routine PSG testing methods because the breathing efforts recording belt technology, airflow monitors, and oxymetry monitoring used do not always demonstrate changes associated in breathing leading to arousals in sleep. These subtle events trigger the muscles of the throat, tongue, and mandible, increasing muscle tone and opening the airway to normalize respirations. More subtle respiratory events known as respiratory effort-related arousals of sleep (RERAS) are best identified and tabulated when the sleep study is performed with the addition of a small, soft catheter placed through an individual's nose and swallowed by the patient such that the tip of the catheter resides in the esophagus within the midthoracic level. This probe, measuring internal negative pressures during sleep, allows for the proper tabulation of these RERAs that would otherwise go undetected and is called esophageal pressure (Pes) testing. (Pes denotes pressure within the esophagus.)

When an individual has fragmented sleep and sleepiness primarily resulting from these more subtle respiratory events the diagnosis of upper airway resistance syndrome (UARS) is given.[29-31] Another method for picking up RERAs using a pressure cannula at the nostrils show patterns of flow restriction, known as flow limitation, do not show the degree of effort being exerted and are not the gold standard of measuring RERAs.

The UARS, consisting of subtle respiratory events, is a very common disorder but its occurrence is unknown in part due to the fact that most sleep disorders centers do not employ Pes testing to objectively establish the diagnosis.[29-31]

Factors That Change in the Collapsibility of the Upper Airway Throughout the Night

The likelihood of airway collapse fluctuates during the night based on sleep stage and body position, in addition to other factors. When someone is lying in a supine position, gravity plays a role in the collapsibility of the pharynx.[11] Gravity can pull the tongue and mandible downward, increasing the degree of obstructive breathing as compared to lying in the lateral position. Frequently, individuals with severe obstructive respirations during sleep tend to prefer sleeping in the lateral or prone position. This preference in body position is probably a subconscious preference to improve respiration. Sleep stage also influences the collapsibility of the airway primarily as a function of muscle tone. For example, during REM sleep, muscle paralysis is manifested throughout the body as it decreases the muscle tone of the upper airway muscles contributing to its collapsibility. As a result, it is frequently demonstrated that patients with severe sleep apnea have their worst obstructive respiratory episodes during REM sleep. Since REM sleep occurs more toward the last portion of the night there would be a minimal amount of REM sleep measured with a split night study (in which only half the night is done in a diagnostic fashion). As a result, the full severity of the person's obstructive respirations may go unrecognized. For this reason, it is important to not rely on sleep studies conducted with a split night protocol when establishing a severity rating of obstructive breathing for an individual, unless, however, the half night demonstrates severe OSA. Frequently, partial night studies show individuals to have a lesser severity of obstructive respiration than would have otherwise been recognized had the diagnostic study been performed the entire night. Rendering a designation of mild or moderate OSA to a patient who has had a split night study is not appropriate for this reason.

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For reasons not yet fully understood, the upper airway is more resistant to collapse during slow wave or N3 sleep.[32-34] Frequently, individuals exert large degrees of negative pressure during slow wave sleep to breathe against a partially collapsed airway and do not develop complete collapse of the upper airway. The same individual during other stages of sleep, such as N1 or N2, will have complete airway collapse at much lower degrees of negative pressure.

Various medications such as those that cause muscle relaxation are known to enhance the degree of collapsibility of the upper airway by decreasing the airway muscle tone. Opiate medications can also blunt the ability to increase airway tone in the presence of increased negative pressure and thus enhance obstructive respirations. Alcohol has a similar effect to that of muscle relaxants medications.[34,35] Sleep deprivation also has the effect of blunting the ability to increase upper airway tone in the presence of obstruction, and, as a result, obstructive respirations become worse when a person is sleep deprived. This clearly is observed in individuals who provide a history of snoring to a greater degree after having been awake for 24 hours.[6]

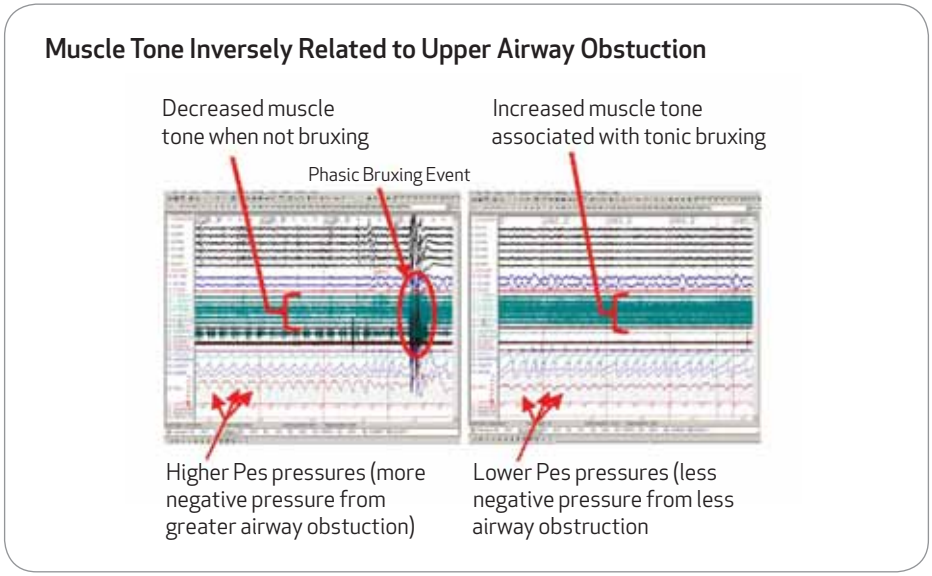

FIGURE 5. The two tracings above are from the same patient at different points during the night. The tracing on the left demonstrates less EMG activity in the muscles influencing the mandible, when compared to the tracing on the right. As a result, there is a greater degree of obstruction and negative pressure on the left tracing compared to the right. This is a demonstration of how tonic bruxism opens up the airway and reduces the obstruction. The phasic increase in EMG seen the left tracing as part of the arousal is a result of the obstructive hypopnea.

Treatment of Obstructive Respirations During Sleep

There are a variety of methods that can be implemented to treat OSA. Surgical methods are provided elsewhere. For patients in whom adipose tissue is a significant factor, weight reduction is beneficial. The most reliable method for treating this condition is through the administration of continuous positive airway pressure (CPAP), which was initially studied by Colin Sullivan, MD, and published in 1981.[37] Over the past 30-plus years, significant improvements in CPAP treatment have been implemented. CPAP works by providing positive pressure to nullify the Bernoulli Effect, described earlier, and neutralizing the vacuum of the upper airway, preventing upper airway collapse. This allows an individual to maintain respirations without repetitive arousals, improving sleep so individuals awaken refreshed.

Various types of masks have been developed to accommodate the variety of facial structures and personal preferences that exist within a general population. CPAP therapy has been successfully implemented in patients as young as under the age of 1 and in the elderly beyond 90 years old. In some, special accommodations are necessary to overcome pitfalls in CPAP therapy. Sometimes simple measures such as the addition of an elastic chin strap that holds the mandible closed can provide the difference between treatment failure and treatment success. The mouth falling open may result in CPAP pressure leakage that nullifies the effect.

Recently there have been innovative techniques that utilize a combination between dental appliances and CPAP such that the CPAP mask is anchored onto an appliance resulting in a stable mask without straps around the head.[38] Also made are full-face masks moulded from an impression of the patient's face that attach to a dental appliance. This results in a mask covering the nose and mouth without any straps around the head. The advantage of using a dental appliance in conjunction with a CPAP mask is that it helps stabilize the mandible in a more anterior or at least in a neutral position, enhancing the treatment from CPAP alone by opening the posterior pharyngeal airway space. Standard full-face masks have a tendency to push the mandible back, particularly if the straps are tightened, and this has the effect of increasing airway obstruction and making the administration of CPAP therapy more difficult. Stabilizing the mandible with a dental appliance has demonstrated to improve the use of full-face masks when this problem occurs.[39]

Properly administering CPAP requires a PSG study that carefully assesses the changes in breathing with the different CPAP levels, and the technologist titrates the pressure to identify the optimal settings for each patient. Many patients require very specific settings.

Bruxism During Sleep as a Protective Mechanism of Upper Airway Collapse

The brain has inherent mechanisms utilized to decrease or eliminate the obstruction of the upper airway during sleep. The preference of body position mentioned above is one subconscious technique frequently utilized. Another method may be sleep clenching or bruxism. SB brings the mandible into occlusion, with or without tongue thrusting, and may reduce obstruction of the upper airway that would otherwise occur in the absence of such mechanical maneuvers.

Historically, it has been recognized that SB and TMD have been associated with obstructive sleep apnea patients.[40-43] The cause for this association has not been previously well-established and assumed to be brought on by the arousals triggered by OSA. Thus, bruxing or clenching phenomena were considered part of the arousal process.[44-46] Recent research by Simmons and Prehn has demonstrated that SB or clenching may occur as a mechanism to prevent airway collapse. Their studies have demonstrated that during the

SB process there is a reduction of negative pressures of the upper airway, measured by pressure catheter Pes monitoring.[47-49] When the SB process is not present, there is an increase in airway obstruction. Studying this population is difficult because SB throughout the night minimizes the obstruction and, as a result, the degree of obstruction referenced by the AHI may not meet the threshold necessary to be considered abnormal by most sleep disorder centers not utilizing Pes monitoring, and not tabulating RERAs properly. Treatment of obstructive breathing with CPAP alone reduces the bruxism/clenching and improves TMD symptoms in a high percentage of patients.[50-52] This sheds additional light on the importance of airway dynamics and dentistry (**FIGURE 5**).

Establishing the sleep-disorder diagnoses early could result in a significant improvement in overall health. This is significant for cardiovascular and cerebral vascular disease since these conditions are now known to be worsened by the ongoing process of obstructive breathing during sleep.[50-55] Establishing that a patient's airway is vulnerable to collapse at a point in the pathogenesis when they are attempting to compensate for the collapse by bruxing may prevent the escalation of this disturbance into OSA when compensation either no longer is being exerted or no longer provides adequate airway protection.

As our heath care system matures, opportunities for collaboration between physicians and dentists are clearly evolving. Changes in the upper airway, particularly the mandibular anatomy and position, affect airway dynamics and can influence sleep. Only through greater initiative within dentistry will the dentist's impact in our overall health care system be fully recognized. The aspects of sleep and sleep physiology covered in this article can help the dentist work closer with physicians to improve their patient's health. ■■■■

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