

Feature Article

Should the Dentist Independently Assess and Treat Sleep-Disordered Breathing?

With sufficient training and knowledge, the dentist can and should manage the mild snorer and moderate sleep apneic.

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abstract

Sleep-disordered breathing is a chronic problem of the inappropriate mechanical collapse of the upper airway. Symptoms range from mild occasional snoring to severe obstructive sleep apnea. The standard of care for the diagnosis and treatment of sleep-disordered breathing by sleep medicine has been the use of the polysomnogram and continuous positive airway pressure. This approach is burdensome, costly, and ineffective due to lack of compliance with or rejection of treatment. Oral appliances are highly effective in managing the mild snorer to the moderate sleep apneic and are approaching the efficacy of continuous positive airway pressure with the severe apneic. The dentist can and should manage these patients. However, the dental practitioner must acquire sufficient training and knowledge to appropriately treat these patients.

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Dentists have been criticized by sleep specialists and in professional literature for independently assessing and treating sleep-disordered breathing, ranging from snoring to sleep apnea. For instance, Rosalind Cartwright, PhD, a sleep specialist, said that dentists were Ademedicalizing@ sleep-disordered breathing.¹ In addition, she called into question the motivation of otolaryngologists and pulmonary physicians for using home monitoring equipment and self-titrating continuous positive airway pressure devices, thereby "despecializing" sleep-disordered breathing. She assumes that only a multidisciplinary team of otolaryngologists and pulmonologists headed by a sleep specialist can properly provide therapy. Today, many dentists in the United States, Canada, England, and Scotland are successfully treating patients with sleep-disordered breathing. A discussion of whether dentists should treat these patients should be based on what is best for the patient, patient preference, and outcomes. To reach a conclusion, several factors must be analyzed and evaluated. These are:

* The anatomy and physiology of the upper airway and the pathophysiology of sleep-

disordered breathing;

* The diagnosis and treatment of sleep-disordered breathing; and

* A proposed standard of care.

The Anatomy and Physiology of the Upper Airway and the Pathophysiology of Sleep-Disordered Breathing

Sleep-disordered breathing is the inappropriate mechanical collapse of the upper airway during sleep. This ranges from slight or infrequent partial collapse to total collapse resulting in a continuum of symptoms from mild snoring to obstructive sleep apnea.

Understanding normal physiology and anatomy of the upper airway from the nose to the glottis is critical to managing the mechanical collapse of the airway. The pharynx acts as a collapsible tube to control breathing, swallowing, and phonation. Because the airway and alimentary canal cross in the pharynx, a very sophisticated mechanism causes this flexible tube to collapse during swallowing,² protecting the airway. As swallowing begins, the mandible retrudes, generally into centric occlusion, forming a stable bony base for muscular contraction of the tongue, elevation of the hyoid bone, and contraction of the muscles of the soft palate and constrictor muscles of the pharynx. Once swallowing is complete, the airway is returned to normal by the relaxation of the constrictor muscles, the contraction of the dilator muscles (stylopharyngeal and palatopharyngeal muscles), and a posturing of the mandible straight forward in a "physiologic rest position" or breathing position. The mandibular plane at rest is parallel to the maxillary plane. A forward movement of the mandible brings the condyles down the eminence of the fossa, causing the posterior aspect of the mandible to move away from the maxilla (Christensen's phenomenon). The chin point moves an equal distance down and forward, creating a space between the maxillary and mandibular teeth known as "freeway space" in dentistry.

This position of the mandible has three distinct functions. First, the movement away from the maxilla stretches and tightens the lateral walls of the pharynx and the superior constrictor muscle through the attachment to the medial pterygoid plate, the pterygoid mandibular raphe, the floor of the mouth, and the tongue.³ Schwab and colleagues⁴ point out the importance of the lateral collapse of the pharynx in obstructive sleep apnea patients. Secondly, the volume of the oral cavity increases, allowing room for the tongue. Thirdly, the forward chin point brings the genial tubercles forward to provide a fixed bony base. The tongue then rests behind the upper incisors and independently forms an air seal with the soft and hard palate. A simple test to confirm the mandible/tongue position is to try breathing only through the nose with the jaw rotated back and open. Another test is to part the lips while the tongue is in the proper position, confirming the ability of the tongue to form an air seal. The description of the neuromuscular activity of the tongue in both the nonapneic and apneic individual has been well-described by Mezzanote and colleagues.⁵ Thus, the function of the mandible and the temporomandibular joint is similar to that of a tent pole, holding the airway open in a protrusive position and allowing it to collapse in a retruded and/or rotated position. A test to confirm this is to try to swallow or snore in a protruded position.

Virtually every health care professional and many lay individuals have a clear understanding of the role the mandible plays to reverse the collapse of the airway when they are trained in

the "ABCs" of cardiopulmonary resuscitation. The "A" represents airway management, clearing the airway of any foreign object, followed by performing a "jaw thrust" maneuver. Only when the mandible is placed in the proper position to open the airway can ventilation, or breathing, "B," be performed easily and successfully. These same techniques apply to managing a patient's airway during conscious sedation or to ventilating a patient with a bag in general anesthesia.⁶ The pathophysiology of sleep-disordered breathing and treatment relate directly to this mechanism of airway management. Sleep-disordered breathing occurs because of the increased narrowing of the airway (flow-resistive load), which can be from two basic causes. The first and primary cause is the position of the mandible and tongue. A sleep-disordered breathing patient's decrease in baseline neuromuscular activity during sleep results in rotation of the mandible into a functionally retruded craniofacial position, partial mouth breathing, and the collapse of the tongue into the pharynx.⁷ The reactivation of the neuromuscular control reverses the collapse as indicated by the chin point electromyogram. The position of the mandible can be affected by underlying craniofacial anatomy, soft tissue anatomy, increasing age, pathological changes, medications, alcohol, food, fatigue, and sleep stage (atonia during rapid eye movement sleep).⁸⁻¹² A supine sleep position also negatively affects the position of the mandible by allowing it to rotate back and open.¹³ The second major cause of airway narrowing is obesity. Fatty deposition in the neck, pharyngeal tissues, and tongue tend to narrow the lumen of the airway and decrease the likelihood of the neuromuscular mechanism and jaw holding the airway open enough to prevent obstruction.¹⁴ Obesity also requires more effort for the patient to ventilate (mass load), although this is separate from the mechanical collapse of the airway. Other causes of airway narrowing, such as a deviated septum or allergies, must be evaluated and may be contributing factors but are not the primary causes of the underlying problem.

Diagnosis and Treatment of Sleep-Disordered Breathing

Traditional Medical Approaches

Sleep-disordered breathing is a continuum from mild, occasional snoring to severe obstructive sleep apnea. Yet, the vast amount of time, effort, and money is spent on the differentiation between the apneic and the nonapneic patient using the polysomnogram for diagnosis. The result is that the diagnosed obstructive sleep apnea patient is treated with continuous positive airway pressure with there being little or no treatment for the failed continuous positive airway pressure patient or the non-obstructive sleep apnea patient. Historically, this treatment paradigm developed in the United States for three reasons. The first was the discovery of sleep apnea by psychiatrists and neurologists during routine experimentation on sleep in the sleep lab setting.¹⁵ The second was the discovery of a way to manage the disorder via the use of continuous positive airway pressure.¹⁶ The third was the high prevalence and the serious consequences of the disorder.⁹ Both the diagnosis and treatment of obstructive sleep apnea required the use of the polysomnogram, causing the proliferation of sleep laboratories for clinical purposes. This occurred during a time of adequate funds for new therapies and created a new specialty, sleep medicine, supported by a billion-dollar sleep industry. The focus of this sleep medical-industrial complex has been

the improvement of the systems surrounding the polysomnogram-continuous positive airway pressure approach. Millions are spent on mask and continuous positive airway pressure technology to improve compliance. A paucity of resources is allocated to other promising technology. Dr. Cartwright was an early advocate of oral appliance therapy. Her 1988 paper on the Tongue Retaining Device invented by psychiatrist Charles Samuelson cited great promise for oral appliances.¹⁷ The results of treatment of 24 patients were that only five of the 24 remained unimproved (the five included the most obese cases). Her conclusions were that "treatment can proceed in a logical fashion starting with the less invasive treatments for both mild and severe cases and with careful clinical management, most patients will reach acceptable levels of control. Only a few will require the more cumbersome CPAP [continuous positive airway pressure] or more invasive surgical treatments." These therapies would be appropriate "for those who do not respond to a trial of habit change and TRD [Tongue Retaining Device] treatment."

"An appliance ... has an early place in the treatment of these [apneic] patients either alone or as an adjunct to other measures."

However, to date little has been done within the sleep community to further oral appliance technology.

Comparison of Treatment Alternatives

Blinded, crossover, outcome studies on all approaches should be the basis for any discussion of the proper management of sleep-disordered breathing. Since these are not available, continuous positive airway pressure therapy and oral appliance therapy will be compared with published data. Surgical treatment is excluded from this discussion since surgery is usually recommended for sleep-disordered breathing only after the more conservative therapies have been tried. Conservative therapies such as weight loss and positional therapy are also excluded since these should be instituted no matter what other approaches are tried. To date, most of the published studies relate to polysomnograms and continuous positive airway pressure. As explained previously, for lack of access to sleep labs and funding, much less has been done with oral appliances. Any analysis of treatment should include efficacy, effectiveness, cost, availability of medical resources, quality of life, and prevention.

Efficacy is the capacity to produce a desired effect. Continuous positive airway pressure is currently the gold standard for efficacy in normalizing an abnormal polysomnogram, not necessarily the gold standard for managing sleep-disordered breathing. Continuous positive airway pressure has two distinct functions. The first is airway management and the second is ventilation. Less has been written describing the ventilatory effects such as increase in vital capacity or an increase in the pressure gradient across the alveoli. The new autopaps appear to be sophisticated ventilators. However, the effect of continuous positive airway pressure to act as a pneumatic splint preventing the mechanical collapse of the pharynx has been well-described by Sullivan.¹⁶ Two facts reveal that it is doubtful that this is the most efficacious way to manage an airway. The first is that continuous positive airway pressure does not work with the jaw rotated back and open, which is the usual sleep position for most

individuals with sleep-disordered breathing. According to Mark Forester, chief technologist at Presbyterian Sleep Institute in Dallas, more than 90 percent of the institute's patients leak air through the mouth, thereby requiring chin straps for continuous positive airway pressure to be effective. Therefore, a significant part of the efficacy of continuous positive airway pressure is the position of the mandible, whether this is accomplished by an external jaw positioning device and/or an increase in the baseline neuromuscular activity of the jaw positioning muscles and tongue to close the jaw and effect an air seal between the tongue and soft palate. The second fact is the difficulty in ventilating a patient with air pressure alone while managing a patient in anesthesia or CPR. Until the jaw and head are in the proper position, ventilation, even with great pressure, is difficult.

The efficacy of continuous positive airway pressure to normalize polysomnograms is well-documented. Although the results of most of the published studies on oral appliances have shown that they are not as efficacious in normalizing polysomnograms, the results of studies on the newer, adjustable appliances are approaching that of continuous positive airway pressure. In an abstract of preliminary results based on 38 patients by Pancer and Hoffstein,¹⁸ the average respiratory disturbance index was 42 before oral appliance therapy. After therapy, most of the symptoms were eliminated, and the average respiratory disturbance index was 11, with virtually all events being mild hypopneas. An abstract by Roberts, Jamieson, and Becker¹⁹ cites similar results with continued improvement beyond the maximum range of protrusion of the mandible. Loube has shown normalization of polysomnographic parameters with and without an oral appliance in a patient with upper airway resistance syndrome.²⁰ More-definitive studies are now under way on oral appliance therapy with upper airway resistance syndrome.

Effectiveness is not the same as efficacy. Effectiveness is efficacy over time to obtain the desired results. Based on this definition, continuous positive airway pressure is highly ineffective for the treatment of sleep-disordered breathing, even obstructive sleep apnea, due to cost, initial rejection of treatment, and compliance. Guilleminault found a 2 percent compliance with continuous positive airway pressure in a large series of patients with upper airway resistance syndrome.¹⁶ Similar results have been shown with the nonapneic snorer. Therefore, continuous positive airway pressure is virtually excluded as a viable therapy in more than 70 percent of the patients with sleep-disordered breathing. A number of studies have also shown the lack of compliance with continuous positive airway pressure in the sleep apnea patient, even those with severe apnea.²¹ A seminal study, done by Kribbs and Pack, utilized covert monitoring to determine continuous positive airway pressure use.²² Of 35 patients, only 16 (46 percent) met the minimum criteria of four hours of use on 70 percent of days monitored. If a conservative criterion of seven hours of sleep based on normative data of middle-aged adults is used, only two of 35 achieved this result at least five of seven days. Their conclusion is that "frequent, long-duration, quality sleep is a relatively rare occurrence in OSAS [obstructive sleep apnea syndrome] patients treated with CPAP [continuous positive airway pressure] ... that actual CPAP use by OSAS patients falls short of providing quality sleep all night, every night."

Another study reviews the diagnostic and treatment process for all patients seen in a major sleep center during an 11-year period.²³ An assumption was made that all the patients who were diagnosed by history to have obstructive sleep apnea were patients with symptoms of