

A New Look At an Old Device

Tongue retaining devices make a comeback and prove their validity through a plethora of scientific research.

By Allen J. Moses, DDS, and R. Michael Alvarez, DDS

The tongue retaining device (TRD) was invented by Charles Samelson, MD, an analytically trained psychiatrist motivated by his own snoring problem. His wife, an accomplished pianist, complained that the loud snoring was affecting her “musical ear.” She was also experiencing such secondary effects of his snoring as sleep fragmentation and excessive daytime sleepiness (EDS) due to the numerous arousals.

Samelson could mimic his snoring by allowing the base of his tongue to fall against the back of his throat, and reasoned that snoring was a partial airway obstruction. To keep his airway open during sleep, he needed to keep his tongue forward. Initially, he molded tongue sleeves out of beeswax. By creating suction in the sleeve and extending the tip of the sleeve anterior to the lips, the goal was accomplished; however, the beeswax was fragile and broke apart easily. Eventually, latex prototypes were developed and a patent was applied for.

Samelson presented his unique idea to Rosalind Cartwright, MD, director of the Sleep Disorder Center at Rush University Medical School. Cartwright was impressed with the idea of a nonsurgical approach to keep the airway open during sleep. They initiated many objective scientific studies that validated the efficacy of the TRD. The first article was published in *The Journal of the American Medical Association* in 1982. They presented their initial findings at the same scientific meeting where Dr Colin Sullivan introduced continuous positive airway pressure (CPAP). A good deal of the research on the TRD was conducted at Rush under the leadership of Cartwright.

Scientific Background

Sleep-disordered breathing (SDB) has received a lot of attention in the scientific literature in the past decade. SDB is commonly divided into four subtypes: obstructive sleep breathing, central apnea, Cheyne-Stokes respiration, and nocturnal hypoventilation. Obstructive sleep apnea (OSA) is by far the most common type of SDB. Apnea literally means “want of breath.” Apnea is defined in adults as cessation of breathing for a period of at least 10 seconds.¹

OSA is usually caused by various combinations of the following contributory factors causing blockage of the pharyngeal airway. These factors include flaccidity of tongue musculature; hyperplastic or redundant tissue; inflammation of tissues; large tonsils; obesity; nasal obstructions (such as allergic rhinitis, hypertrophied adenoids, deviated septum, broken nose, enlarged turbinates, and nasal polyps); long soft palate; large uvula; retrognathic mandible; large tongue;

incoordination of genioglossus muscle; edentulous collapse; and micrognathia due to four bicuspid extraction orthodontics.²

A diagnosis of OSA is based on the co-existence of EDS and at least two other major symptoms, such as snoring, witnessed apneas, frequent nocturnal arousals, and at least five obstructed breathing events per hour of sleep on polysomnographic study.³

Snoring refers to the sounds emanating from partial obstruction of the airway during sleep. Hypopnea defines an event of at least 10 seconds where there is breathing, but ventilation is reduced in depth of breaths by at least 50% from the previous baseline during sleep. The term upper airways resistance syndrome (UARS) is used to describe recurrent episodes of airflow limitation during sleep without accompanying apnea episodes.

The consequences of snoring, UARS, and OSA are arousals from restorative sleep, EDS, and a high statistical correlation with automobile and workplace accidents and such comorbidity factors as hypertension, coronary artery disease, congestive heart failure, transient ischemic attack, and stroke.⁴⁻⁶ There seems to be a continuum from snoring to UARS and OSA. Patients with UARS and snoring suffer from many of the same comorbidity factors as OSA patients.⁷⁻¹¹ Successful treatment or prevention of SDB is of critical importance to people diagnosed with snoring, UARS, and OSA.

Intraoral Devices

Four distinct types of intraoral devices can be identified that treat snoring and apnea. They are as follows:

Type I—Devices that open the bite vertically. Typically, these are mandibular orthotics. Placed in the mouth to curtail the effects of bruxism, they create more room in the oral cavity for the tongue by increasing the vertical dimension. Dentists using flat plane mandibular appliances for bruxism commonly encounter patients who serendipitously report decrease or cessation of snoring with their Type I device. Devices in categories II, III, and IV by their nature also open the bite vertically. A Type I device does not actively advance the mandible forward, but it also does not prevent it from advancing forward if the muscles so dictate.



Figure 1. An example of a Type II device that is adjustable both anteriorly and vertically.

Type II—Devices that actively advance the mandible forward and support this position. The tongue, by the nature of human anatomy, must advance with the mandible, but its advancement is passive. Figure 1 (page 36) shows a Type II device that is adjustable both anteriorly and vertically.



Figure 2. Examples of a Type III and IV device—positioners constructed with an interior tongue bulb.

Type III—Devices that actively pull or hold the tongue in a position more anterior than the normal resting tongue position. They are constructed with a bulb protruding anterior to and between the incisor teeth and lips, which by means of negative pressure holds the tongue forward during sleep. The mandible by nature must also come forward with the tongue, but the mandibular advancement is passive. Figure 2 shows an example of a Type III device.

Type IV—Devices that actively advance the mandible forward and support it in a forward position, and also have the anterior bulb protruding between the teeth and beyond the lips to actively hold the tongue in a protruded position. An example would be a positioner with adequate vertical opening for a tongue bulb to be built in. Figure 2 is an example of a Type IV device—a positioner constructed with an anterior tongue bulb.

TRDs can be either Type III or Type IV. Because TRDs are based on suction or negative pressure to keep the tongue in the forward posture, a lip seal around the device is essential. Therefore, nasal patency is requisite for success of TRDs.¹²

Anatomy

The soft palate consists of five striated muscles including the palatoglossus, palatopharyngeus, levator palatini, tensor palatini, and musculus uvulae. They work in complex group actions. Palatoglossus, palatopharyngeus, and levator palatini are true respiratory muscles, as evidenced by increased electromyographic (EMG) activity during inspiration. Increased activity of palatoglossus and palatopharyngeus and decreased activity of levator palatini are associated with nasal breathing. In the supine position, increased activity has been documented in palatoglossus and palatopharyngeus during nasal breathing to promote nasal airflow and counteract the effects of gravity on the soft palate and tongue.¹³⁻¹⁵

The major tongue muscle, genioglossus, protrudes the tongue. With nasal breathing, the genioglossus tenses with each inspiration.¹⁶ Genioglossus contracts on lying down supine,¹⁷ thus increasing or maintaining the retroglossal airway in awake patients with OSA.¹⁸

Genioglossus strength, however, decreases with age.¹⁹ Sleep generally produces a hypotonia of striated muscle.²⁰ Respiratory problems can occur because the pharynx can collapse when intraluminal pressure falls with inspiration. Patients with OSA have smaller upper airways than normal subjects when assessed in the supine position by computed tomography scan.²¹⁻²²

Pharyngeal size in awake normal subjects decreases with obesity and decreases with age in both men and women.²³

Control of tongue posture is central to preventing OSA, UARS, and snoring. Control of tongue posture is also an extremely complex issue. The collapse of the tongue on the pharynx has been shown by Remmers et al²⁴ to be associated with decreased EMG tone in genioglossus. Genioglossus EMG activity does not alter from the wakeful state to non-rapid eye movement (NREM) sleep²⁵ but does decrease in eye-movement-dense REM sleep.²⁶

Initiation of the apneic airway occlusion occurs on inspiration. The factors essential to the closure are the decreased EMG activity of genioglossus and insufficient opposing activity by the upper airway dilating muscles. Negative pharyngeal pressure on inspiration created by such airway obstructors as tonsils, uvula, nasopharyngeal polyps, and deviated septum facilitates sucking the airway closed when genioglossus tone is reduced during sleep. Arousal from sleep by the brain is a requisite for pharyngeal reopening and reactivation of genioglossus.

TRD Effectiveness

Numerous studies published in the scientific literature have objectively measured the effectiveness of TRDs. Higurashi et al²⁷ measured eight patients with pulse oximetry, baseline before and after treatment with a TRD. They found TRDs to be effective at increasing nocturnal O₂ levels in OSA patients—in some cases, severe OSA.

Ono et al²⁸ demonstrated in PSG studies on seven patients that a TRD reduced the apnea hypopnea index (AHI) and counteracted fluctuating genioglossus EMG activity.

Adachi et al²⁹ observed in a study of 10 OSA adult patients and four symptom-free controls that the timing relationship between genioglossus activity and inspiratory effort is an important contributory factor in the pathogenesis of OSA. Further, this relationship explains the effectiveness of TRDs in the resolution of OSA.

Cartwright et al³⁰ conducted a study using polysomnography (PSG) to measure the effects of position on AHI and found that by training patients to sleep in a lateral position, they could reduce AHI. Patients wearing a TRD, however, had the reduction in AHI even in the supine position.

In 1985, Cartwright³¹ performed a discriminant function analysis on 16 male subjects that entered age, obesity, and ratio of apnea events per hour in a side vs back sleep posture, which correctly classified the success of the patient's response to TRD treatment. The strongest predictor of success of a TRD is

increased AHI severity in supine vs lateral sleep position; 68% (11 out of 16) patients got successful results with TRD treatment.

Ono et al³² demonstrated that a TRD has different effects on the awake genioglossus muscle activity in control subjects than patients with OSA. The change in anatomic configuration caused by a TRD enlarges the volume of the airway and reduces upper airway resistance to airflow.

Cartwright and Samelson³³ conducted a PSG study of 20 middle-aged nonobese males with a diagnosis of OSA who had significant reduction in the number of apneic episodes wearing a TRD. The results with TRD were comparable to the rate reported for patients treated surgically by either tracheostomy or uvulopalatopharyngoplasty (UPPP). The sleep of patients with OSA is characteristically light, lacking in Stage 3, Stage 4, and REM sleep, and has numerous and significant O₂ desaturations. Use of a TRD resulted in an increase in Stage 3 and REM sleep, and a decrease in light (Stage 1) sleep.

Caldarelli et al³⁴ reported a PSG study of 40 OSA patients on whom surgery was performed. The surgical procedure was submucous resection alone or with palatopharyngoplasty (PPP). Of the 40 subjects, 18 cases were deemed successful using an American Academy of Sleep Medicine criterion of 50% reduction in AHI. Some of the 22 whose results did not fulfill the success criterion were given supplemental treatment with a TRD. Five out of 10 less-than-successful submucous resection cases then met the success criterion. In a less-than-successful group of six patients receiving both submucous resection and PPP and then receiving supplemental treatment with a TRD, four out of the six were deemed successfully treated on follow-up PSG studies.

Dort and Hussein³⁵ performed a block randomized control study of 39 patients comparing a one-size-fits-all TRD to a nonsuction control device. The suction device reduced snoring by more than 70%, significantly reduced the respiratory distress index, and increased the quality of life scale. These results were cited as being comparable to those reported for custom-made devices.

Cartwright et al³⁶ reported a study of 24 males suffering from OSA and using a TRD as the initial treatment, and developed a prediction model of factors associated with successful use of a TRD. Post-treatment severity of OSA, as measured by AHI, was significantly lower than pre-treatment or post-treatment on non-TRD nights in patients with a wide range of apnea severity. The factors most indicative of TRD success include AHI severity at least two times worse supine than nonsupine and body weight not greater than 50% above the ideal.

Cartwright et al³⁷ compared somnoplasty, a surgical procedure, to a noninvasive oral appliance, a brand-new TRD. No significant difference was noted between the two groups in percentage of time spent in loud snoring while treated; 70% of patients in both groups met improvement criteria for reduced loud snoring.

Discussion

The success of the TRD when treating OSA has been reported in numerous scientific papers using objective measurement. The low compliance rate noted for CPAP use³⁸⁻⁴⁰ suggests that all patients do not tolerate it very well. CPAP can certainly be uncomfortable. Patients needing high CPAP pressure often cannot keep their mouth closed, necessitating use of the head strap. Perhaps the use of a TRD in conjunction with CPAP could significantly lessen the pressure necessary to interrupt the pharyngeal obstruction of the tongue in sleeping OSA patients. A TRD, even with a large tongue bulb and mandibular advancement (Type IV), would allow a patent lip seal around the device.

Clinicians must use their best clinical judgment, having examined their patient, reviewed the history and the PSG data, consulted with other treating or referring doctors, and evaluated the relevant literature.

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