

## Review Article

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## ORAL STRUCTURES AND SLEEP DISORDERS: A LITERATURE REVIEW

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### ABSTRACT

In recent years, a wealth of studies revealed the many physiological and histological changes the oropharyngeal muscles go through as a result of sleep disorders, in particular, as a result of Obstructive Sleep Apnea (OSA). This article presents a literature review of the most recent findings regarding sleep disorders and their effect on oropharyngeal structures. The article includes classifications and definitions of various sleep disorders; explains the negative implications of sleep disorders in children, as well as in adults; reviews the viable options to treat sleep disorders; suggests steps in which the orofacial myologists could be instrumental in identifying and referring patients with possible sleep disorders; and suggests further studies on the subject conducted by orofacial myologists.

### INTRODUCTION

For centuries, science deconstructed and reconstructed the functions of the human body during daytime, but nighttime functions received very little, if any, attention. Why do people sleep for several hours at night? Why do people (and animals alike) have the need to sleep at all? What happens when we sleep? These are some of the questions that have not been answered adequately. However, in the last couple of decades, scientists from different disciplines started to inquire about nighttime functions and their importance. Currently, in fields such as dentistry, otolaryngology-head and neck surgery, neuropsychology, or physiology, an incredible wealth of information is being uncovered as to why we have sleep disorders and how to solve the problem. Orofacial myologists can benefit from this research and make it useful in their everyday practice by helping in the identification of at-risk subjects, providing referrals when indicated, and maybe even treating selected cases of sleep disorders. Orofacial myologists have the in-depth knowledge of anatomy, physiology and pathology of oropharyngeal musculature that help them understand the role of those muscles in sleep disorders. Also, there is a new and exciting field of research that can be done on the efficacy of myofunctional

therapy (MFT) to treat, directly or indirectly, selected sleep disorders. For those orofacial myologists who see children for MFT, it would be interesting to record information on sleep habits before and after therapy, to see whether or not selected sleep disorders decreased or even disappeared.

### BACKGROUND

Sleep disorders affect millions of Americans each year, regardless of age, gender and economic status. The overall damage to the economy, stemming from sleep disorders, is easily established in the billions. Current scientific literature agrees that the negative effects of sleep disturbances increases cardiovascular pathologies, while decreasing the recovery rate of cardiovascular conditions such as a stroke or heart attack. According to Roux, D'Ambrosio, & Mohsenin (2000), sleep disorders are independent risk factors for cardiovascular diseases, secondary to hypercapnia, arousal, hypoxia, increased sympathetic tone, and altered baroreflex control during sleep. Obstructive Sleep Apnea (OSA), alone, is an independent risk factor for stroke (Partinen, 1995). Also, sleep disorders are correlated with reduced working performance, car and job-related accidents (Hodoba, 1999), excessive daytime sleepiness, impaired memory,

depression, increased attention deficit in children, increased learning difficulties, and relational problems (Lavigne, Goulet, Zuconni, Morisson, Lobbezoo 1999; Lyle, 1999; Smith, Ronald, Delaive, Walld, Manfreda, Kryger, 2002).

The International Classification of Sleep Disorders (cited in Thorpy, 1990) defines the following three major categories:

**Dyssomnias:** insomnia, narcolepsy, "restless leg syndrome", nocturnal eating, and circadian sleep disorders related to jet lag or work shifts. Dyssomnias are characterized by difficulty in initiating or maintaining sleep, or excessive sleepiness during the day;

**Parasomnias:** sleepwalking, nightmares, Rapid Eye Movement (REM) stage disorders, limb movement disorder, sleep bruxism, sleep related swallow disorders, snoring, and sleep apnea. Parasomnias are defined as disorders of arousal and sleep stage transition.

**Sleep disorders associated with psychiatric or medical disorders:** anxiety, overuse of alcohol and drugs, headaches, Parkinsonism, epilepsy, Gastro Esophageal Reflux Disorder (GERD), fibromyalgia, severe sleep bruxism (fragmentary myoclonus), and sleep choking syndrome. These are not primarily sleep disorders but symptoms of other pathologies that induce sleep disturbance or excessive sleepiness.

Within their practice, orofacial myologists can be instrumental in identifying parasomnias, in particular, snoring and sleep apnea. However, not all snoring is pathological and not all sleep apneas are the same. The classification by Lavigne et al. (1999) gives a frame of reference for definitions of sleep disorders (Table I). Orofacial myologists might be involved mainly, directly or indirectly, with Obstructive Sleep Apnea and Snoring.

Table I. Characteristics of Sleep Disorders (Modified from Lavigne et.al., 1999)

Snoring	Obstructive Sleep Apnea (OSA)	Central Sleep Apnea (CSA)
Loud noise	>5 episodes and >10 seconds duration per hour	Apnea >10 sec. each
No arousal	Accompanied respiratory effort	No accompanied respiratory effort
Normal sleep and respiration patterns	Frequent arousals	Respiratory efforts after 10-30 sec. of apnea-hypopnea
No O2 desaturation or cardiac arrhythmia	Bradycardia	10-60 sec. hyperventilation after apneic episode
Worse in supine position	Arterial O2 desaturation	Frequent arousals
Complaints from bedroom partners	Short sleep latency (<10 min., with 10-20 min. normal)	Bradycardia
Dry mouth at wake time	Happens during daytime sleep as well	O2 desaturation
	Not due to GERD, asthma, choking etc.	Not due to physical obstruction as in OSA, GERD, asthma, choking etc.
	Excessive sleepiness by self and others report	Complaints of insomnia or hypersomnia by self and other report
	Morning headache	Frequent napping during the day
	Dry mouth	Driving somnolence
	Sexual dysfunction	Headache at wake time
	Intellectual performance deterioration	Depressive reactions
		Reports of hypertension
		Sexual dysfunction

Sleep disorders are influenced by sleep stages. Usually there are five stages sequentially running throughout the night (Kalat, 1996):

**Light sleep** or Stage 1, characterized by muscle relaxation, fragmented images, a drop in body temperature and unsynchronized neural activity;

**Transition** or Stage 2, characterized by a generalized slowing of heart rate, breathing and brain wave activity;

**Deep sleep** or stages 3 and 4, characterized by release of neurotransmitters that induce diffuse body relaxation, inhibits perception of external stimuli, and produce slow and large brain waves, which indicate a reduction of brain activity. These two stages are more common in the first half of the night;

**REM** or Stage 5, characterized by paradoxical rapid eye movements (REM) in opposition to postural muscles in maximum relaxation state. The dreams, during stage 5, are more vivid and the body is most difficult to waken. This stage is shorter in the evening and longer in the morning hours.

Males and females present OSA in different sleep stages (O'Connor, Thornley, & Hanly, 2000; Shea, Edwards, & White, 1999), with women having a significantly higher REM OSA than men. Since REM stages are reduced in time as compared to non-REM stages, women present less severe cases of OSA. Also, the generalized muscular relaxation in both REM and non-REM puts men who sleep in a supine position disproportionately at risk for OSA, compared to women.

## DIAGNOSIS/TREATMENT

Sleep disorders are usually diagnosed in sleep clinics, through an overnight polysomnography, where the patient is attached to a complex set of instruments designed to measure heart beat, breathing

amplitude and frequency, and especially blood oxygenation level. It is the hourly fluctuation of the content of oxygen in the blood that reveals episodes of sleep apnea. If the patient cannot stay overnight at the clinic, for whatever reason, it is possible to have a sufficient assessment of Respiratory Disturbance Index (RDI) by using portable pulse oxymetry, a minimally invasive procedure already used by physical therapists to monitor their patients during rehabilitation (Brouillette et al, 2000).

The severity of sleep apnea is measured by the RDI or by the Apnea-Hypopnea Index (AHI). Both measure the number of episodes of apnea of more than 10 seconds each, per hour. Less than 5 episodes of apnea per hour are considered normal, 10-20 episodes indicate a mild sleep apnea, 21-40 indicate a moderate sleep apnea and over 40 episodes of sleep apnea per hour is considered to be a severe disorder.

Many factors contribute to sleep disorders:

**gravity** (Elliott, Shea, Dijk, Wyatt, Riel, Neri, Czeisler, West, Prisk, 2001),

**age** (Ivanhoe, Cibirka, Lefebvre, Parr, 1999; Mortimore, Fiddes, Stephens, Douglas, 1999),

**gender** (Mortimore et al., 1999; O'Connor, Thornley, & Hanly, 2000),

**sleep position** (Hellsing, 1989; Jan, Marshall, & Douglas, 1994),

**body weight and body mass** (Partinen, 1995; Redline, Tishler, Schluchter, Aylor, Clark, Graham, 1999),

**small retrognathic mandible** (Victor, 1999; Watanabe, Isono, Tanaka, Tanzawa, Nishino, 2002),

**reduced upper airway space** (Nelson & Kulnis, 2001),

**elongated uvula or hypotonic soft palate**

(Series, Cote, Simoneau, Gelinas, StPierre, Leclerc, Ferland, Marc, 1995; Series, Simoneau, StPierre, Marc, 1996),

**craniofacial dysmorphism** (Victor, 1999),

**debilitating illnesses** (Riley, Benson, Gremillion, Myers, Robinson, Smith, Waxenberg, 2001; Smith et al, 2002),

**strokes and TBI** (Sandbergh, Franklin, Bucht, Gustafson, 2001),

**drugs, alcohol and tobacco** (Ivanhoe et al., 1999),

**discoordination between breathing and swallowing patterns**

(Smith, Wolkove, Colacone, Kreiman, 1989; Teramoto, Sudo, Matsuse, Ohga, Ishii, Ouchi, Fukuchi, 1999).

The impact of sleep disorders can be identified, quantified and monitored in orofacial changes visible through cephalometric analysis (Caprioglio, Zucconi, Calori, Troiani, 1998; Kulnis 2000).

Existing studies have indicated that individuals at risk for OSA are likely to be:

-children with enlarged tonsils and adenoid pads, coupled with nasal allergies or arched palate (Nelson & Culnis, 2001; Nieminen, Tolonen, Lopponen, Lopponene, Luotonen, Jokinen, 1997; Nieminen, Tolonen, & Lopponen, 2000; Seto, Gotsopoulos, & Cistulli, 2001);

-middle-age males, or post menopausal women, obese or overweight, often with short and thick neck or small mandibles, who sleep on their backs (Ivanhoe et al., 1999; Lavigne et al., 1999; Lyle, 1999; Odeh, Shnall, Gavriely, Oliven, 1995 ).

Stroke and Traumatic Brain Injury (TBI) patients may be at risk for Central Sleep Apnea (CSA) or a combination of OSA and CSA. According to Beetar, Guilmette, & Sparadeo (1996), TBI patients present more

insomnia and more complaints of pain than non-TBI patients. Also, mild TBI patients present significantly more pain complaints and more complaints of sleep problems than moderate to severe TBI patients. Finally, the study suggests that pain is strongly associated with sleep disorders.

Sleep disorders are common and associated with morbidity and mortality secondary to hemodynamic, metabolic, and hematologic changes during sleep-related breathing disorders. Patients with a Transient Ischemic Attack (TIA) should be evaluated for sleep disorders (Mohsenin, 2001). Sleep apnea is common in Cardio Vascular Accidents (CVA) patients and is associated with delirium, depression, delayed response to verbal stimuli, and impaired activities of daily living (Sandberg et al., 2001). The incidence of sleep disorders and strokes increases with age, therefore a sleep screening protocol needs to be included in assessing elderly CVA or TIA patients.

Most muscles of the oropharyngeal structures are impacted by sleep disorders, especially those which are directly involved with keeping the airways patent (Choi, Kee, Lee, Ye, 2001; Fogel, Malhotra, Pillar, Edwards, Beauregard, Shea, White, 2001; Malhotra, Pillar, Fogel, Edwards, Ayas, Akahoshi, Hess, White, 2002; Mathur, Mortimore, Jan, Douglas, 1995; Mortimore et al., 1999; Olofsson, Mattsson, Hammarstrom, Hellstrom, 1999; Popovich & White, 1995; Scardella, Krawciw, Petrozzino, Santiago, Edelman, 1993; Schotland, Insko, & Schwab, 1999; Smirne, Innaccone, Ferini-Strambi, Comola, Colombo, Nemni, 1991; Stauffer, Buick, Bixler, Sharkley, Abt, Manders, Kales, Cadieux, Barry, Zwillich, 1989; Woodson, Garancis, & Toohill, 1991; Yoshida, 1998). As a group, the muscles responsible for airway patency are called Pharyngeal Dilator Muscles (PDMs), which are antagonistic muscles of the upper, medial and inferior pharyngeal constrictors (Table II). In a study by Kuna & Smickley (1997),

**Table II. Most Important Pharyngeal Dilator Muscles**

**Genioglossus:** Maintains the tongue in a forward position and it is the only muscle active in supine position;

**Geniohyoid:** Contributes to a forward tongue position by pulling the root of the tongue towards the mandible;

**Musculus Uvulae:** Follows the palatoglossus in a downward position, sealing against the tongue, therefore incrementing nasopharyngeal airways space;

**Palatoglossus:** Also called nasopharyngeal dilator, pulls the soft palate down, toward the tongue, increasing the nasopharyngeal airways space;

**Sternothyroid:** Contributes to pharyngeal airways stability, by pulling the larynx down;

**Sternohyoid:** Contributes to pharyngeal airways stability, by pulling the hyoid bone downward;

**Digastric:** With the anterior and posterior belly, contributes to pharyngeal airways by stabilizing the hyoid;

**Mylohyoid:** Contributes to pharyngeal air space opening by pulling the hyoid upward and forward;

**Stylohyoid:** By pulling the hyoid backward and upward, it contributes to pharyngeal airways stability.

#### **Muscles Indirectly Involved in Pharyngeal Dilation**

**Orbicularis Oris:** Contributes to mouth closure and therefore stability of the mandible, facilitating a forward position of the Genioglossus;

**Masseter:** Contributes to stability of the mandible, therefore facilitating a forward position of the Genioglossus.

during non-REM sleep, the Superior Pharyngeal Constrictor was not activated in subjects with OSA nor in the control group, except for airway reopening, indicating the involvement of other muscles in obstruction of airflow such as the PDMs.

The most important PDM is the genioglossus (GG), which is the main muscle of the tongue and is responsible for its forward and upward movement. Many studies suggest that the GG goes through physiological and histological changes as a result of sleep disorders. According to Smirne et al. (1991), the GG muscles, in subjects with OSA, present an increase in fibers Type II (fast twitch) and a reduction of fibers Type I (slow twitch). The GG and geniohyoid, in subjects with OSA, present an adaptation and muscle injuries significantly higher than in the sternohyoid and sternothyroid muscles in subjects with OSA and a control group, suggesting that not all PDMs are involved and damaged, in sleep disorders, to the same extent. (Schotland, Insko, & Schwab, 1999). The metabolic and histochemical characteristics (glycolytic, glycogenolytic, and anaerobic enzymes, Type I and Type II fibers) in the musculus uvula and GG muscles are different in sleep apnea subjects, compared to snorers and a control group, and are not present in all PDMs (Series et al., 1996). These findings suggest a structural change, mostly for the soft palate and the tongue, in

response to OSA.

Shea, Edwards, & White, (1999) suggest that the GG responds to local mechanisms of upper airway negative pressure. The intrapharyngeal negative pressure itself modulates the GG activity, independent from central control, and monitors the activity during and between breaths. The GG reflex is more active during wakefulness and reduced during REM sleep, therefore it is more vulnerable to collapse during REM sleep. The palatoglossus muscle is a nasopharyngeal dilator muscle and is activated during negative upper airway pressure in inspiration, but only the GG is activated in a supine position (Mathur et al., 1995). Mortimore et al. (1999) indicated that the GG strength is greater in males than females and decreases with age. However, fatigability of GG is equal across genders. This finding may explain why sleep disorders increase with age but does not explain the gender difference. Wiltfang, Klotz, Wiltfang, Jordan, Cohrs, Engelbe, and Hajak (1999), found that daytime electro-stimulation of suprahyoidal muscles may prevent episodes of obstructive sleep apnea suggesting that muscle training may be helpful in treating of sleep disorders.

During sleep, in healthy subjects, masseters present activity characterized by low intensity and short duration of muscle



contraction. Such intensity and duration of muscle activity is greater in men than women. Conversely, facial pain may be associated with increased activity of masseters during sleep (Gallo, Gross, & Palla, 1999). Masseters are involved in sleep bruxism, and there are indications that bruxism is mostly regulated by central cerebral stimuli and not by occlusal or muscular imbalance, as old literature indicated (Lavigne, 1999; Lavigne et al. 1995). Electromyogram (EMG) of GG, masseters, and lateral pterygoid muscles showed that there is a decrease in EMG amplitude, corresponding to hypotonia, during episodes of OSA, compared to before the episodes, and a greater amplitude after the episodes of OSA. These findings suggest that the OSA episode puts a strain on the muscles as the body tries to remove the obstruction. A decrease in EMG amplitude was not observed in Central Sleep Apnea (Yoshida, 1998), confirming that in CSA the brain is somehow disconnected from the muscular receptors that trigger an inspiratory act when the oxygen level drops.

The importance of muscle tone and balance in muscle activity across the sleep and wake cycles, suggests that when orofacial myologists perform their therapy for lip seal, masseter balance, tongue repositioning and velopharyngeal competency, the benefit might extend from daytime muscle activities to nighttime muscle activities. It would be interesting to document the effect of myofunctional therapy on sleep disorders.

Dentists and orthodontists already have a history of using appliances to treat sleep disorders (Strauss, 2001), with over 35 dental devices currently available. These devices exert their action on mastication and tongue muscles. The devices are mainly divided in two categories: the Tongue Retaining Devices (TRD) and the Mandibular Advancement Devices (MAD). The TRD can be worn by edentulous patients because they fit like upper dentures. The TRD feature a suction cup

built toward the anterior alveolar area of the palate, which holds the tongue and keeps it in a forward position, increasing the retroglossal space. The MAD devices open the airways by keeping the mandible in a slightly forward position. They look like sport mouth guards. MADs are also called Mandibular Repositioning Devices (MRD). Oral appliances are more effective in cases of mild to moderate OSA than severe OSA (Fransson, Isacson, Leissner, Nasman, Alton, 2001; Nakazawa, Sakamoro, Yasutake, Yamaga, Kotorii, Myiarata, Ariyoshi, Kameyama, 1992).

The MAD are contraindicated for individual with TMJ disorders and it may cause temporary TMJ pain, vertical changes in occlusion and horizontal occlusion shifts. However, most studies agree that when the patients are motivated, compliance for using the devices every night is superior to using Continuous Positive Air Pressure devices (CPAP). The TDR and the MAD also have a greater overall result in the long run when compared to surgery options (Rose, Barthlen, Staats, Jonas, 2002; Walker-Engstrom, Tegelberg, Wilhelmsson, Ringqvist, 2002). A study by Bondemark (1999), on a MAD device over a 2-year period, suggests that prolonged use of these devices did not change the inclination of upper and lower incisors, but it significantly changes the position of the mandible, to a more forward and downward position. He also found that, as a result of the mandibular shift, there was a significant decrease in overjet and overbite in his subjects. Functional orthodontic devices, still used in Europe, such as Balther's Bionator, are an example of MAD (Hiyama, Kuribayashi, Ishiwata, Kuroda, 2002). A new field of research is studying more flexible and comfortable sleep appliances to decrease facial pain and increase compliance and improve results.

A popular and non-invasive treatment for sleep disorders is the nasal CPAP. The CPAP device is a calibrated pump which delivers room air through a mask with a

gentle pressure, strong enough to overcome any stricture or obstruction. Although the CPAP is recognized to be effective in treating sleep disorders, patients' compliance is rather low, mostly because of the need for a cumbersome mask, its impact on intimacy, and morning dryness of the mucosa (Ivanhoe et al., 1999).

Sometimes the use of the nasal mask, as an alternative to an oral mask, improves the compliance (Mortimore et al., 1999). Problems with portability of the device hinders compliance as well. Aside from increasing blood oxygenation, CPAP may correct the function of GG muscles, because the structure and fatigability of those muscles are different in subjects with OSA, compared to a control group. In the hospital setting, sleep disorders, which are common in TBI patients, may go undetected and untreated, when CPAP could provide a viable treatment option (Castriotto & Lai, 2001).

In more severe cases of OSA, a surgical approach may be considered. The American Sleep Disorders Association considers several types of surgery as viable procedures to treat OSA (Table III).

The most popular procedures have been the Laser Assisted Uvulopalatoplasty (LAUP) and the Uvulopalatopharyngoplasty (UPPP) for adults and tonsillectomy and adenoidectomy in children. For LAUP and UPPP, the short term results are good but

the long term efficacy, indicated by the presence of OSA at the 2-year mark, ranges from 46% to 73% (Ivanhoe et al. 1999; Littlefield & Mair, 1999). Also, LAUP is more effective with mild to moderate OSA and less with severe OSA (Finkelstein, Stein, Ophir, Berger, Berger, 2002). However, the general consensus is that more aggressive types of surgeries are more effective in cases of mild or moderate OSA and not as effective in cases of severe OSA, thus making surgery a less appealing alternative, reserved for those patients with severe craniofacial malformations or other conditions (Walker-Engstrom et al, 2002;

Vilaseca, Morello, Montserrat, Santamaria, Iranzo, 2002).

The role of the orofacial myologist is to identify some of the risk factors indicated for sleep disorders and refer the patient for further evaluation. Ivanhoe et al. (1999) suggests the use of different self-administered questionnaires for the patient to fill out, which could help in better identifying the at-risk patients. Kump, Whalen, Tishler, Browner, Ferrette, Strohl, Rosenberg, Redline, (1994) found questionnaires to be statistically useful, and the best predictors of accuracy were questions about the intensity of snoring as judged by a roommate, episodes of nocturnal choking observed by a roommate and episodes of falling asleep while driving. Victor (1999) and Ivanhoe et al. (1999) also offer simple questionnaires that can be used to screen patients with sleep disorders.

While waiting for the results of a polysomnography, to determine the objective presence of sleep disorders, the orofacial myologist might be able to give some suggestions regarding behavioral changes that can help the patient improve sleep. Changes in sleeping position, reduction or elimination of drugs and alcohol consumption, including sleeping pills, sensible weight loss programs are all changes that the patients may be able to make on their own (Ivanhoe et al., 1999). Also, the use of some low-tech devices might help. Nasal strips may increase oxygenation. Chewing on latex tubes may increase the masseters function and provide more stability to the mandible during sleep. Chewing gum during the day may reduce drowsiness and increase attention and concentration (Hodoba, 1999). Earplugs are an excellent and underestimated device to reduce sleep disorders in debilitated patients in Intensive Care Units (ICU) or for those sleeping in noisy rooms (Wallace, Robins, Walker, 1998). Earplugs are also advisable for snorers' bed partners. However, ear plugs may reduce or eliminate sounds and noises that are necessary to



**Table III. Types of Surgery Available for Sleep Disorders.**


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<b>Laser-assisted uvulopalatoplasty (LAUP):</b> Removal of part of the uvula and creation of vertical trenches in the soft palate, performed in local anesthesia over a few weeks;
<b>Uvulopalatopharyngoplasty (UPPP):</b> Removal of tonsils followed by partial removal of pharyngeal arches, uvula and soft palate, requires general anesthesia;
<b>Tonsillectomy and adenoidectomy:</b> Removal of tonsils and adenoids, mostly in children to increase airways space, is performed usually in general anesthesia;
<b>Nasal septal surgery:</b> Usually it includes correction of nasal septum deviation and remodeling of the turbinates to increase nasal airflow, requires general anesthesia;
<b>Radio Frequency Ablation (RFA):</b> Scars the palatal muscles but not the mucosa, creating some stiffening of the soft palate, performed in local anesthesia;
<b>Cautery Assisted Palatal Stiffening (CAPS):</b> Use of electrocautery to remove strips of mucosa from the soft palate and uvula, causing a stiffening of the palate, performed in local anesthesia;
<b>Mandibular surgery:</b> Is reserved for patients with severe retrognathism who did not benefit from less invasive options;
<b>Inferior mandibular osteotomy:</b> Increases the airways space by moving the mandible forward, and is often performed in conjunction with remodeling or repositioning of other structures;
<b>Maxillo-mandibular advancement osteotomy (MMO):</b> Is reserved for bariatric patients with severe OSA who present a retrognathic mandible;
<b>Hyoid bone suspension:</b> Consists in repositioning the hyoid over the thyroid, securing the tongue in a more downward and forward position, thus creating more postlingual space;
<b>Genioglossal advancement with hyoid myotomy and suspension (MOHM):</b> Complex and lengthy surgical operation reserved for selected patients;
<b>Partial tongue resection:</b> Indicated for selected patients due to its impact on swallowing and speech;
<b>Linguoplasty:</b> Reduction and remodeling of the tongue performed in subjects with enlarged tongue, often secondary to genetic disorders;
<b>Tracheostomy:</b> reserved for severe cases of sleep apnea, usually secondary to other medical conditions requiring drastic approaches.

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detect, such as crying babies, barking dogs, ringing telephone, or alarms. Another low-tech device could be a wide band of tape applied to the chin and cheeks to help the mandible and the mouth to stay closed. Some patients have probably already come up with customized solutions to their problems.

The role of applied orofacial myology in treating sleep disorders is still anecdotal. It makes sense that training the tongue to be at rest in an anterosuperior position promotes better airways patency, although at the present moment there is no study that supports such a hypothesis. It's clear that orofacial myologists have the expertise to apply non-invasive treatments for sleep disorders. Such treatments can be judged and quantified just like any other. Sleep disorders are a serious problem that can be reduced by retraining the muscles.

## CONCLUSIONS/RECOMMENDATIONS

The studies presented in this article indicate

a direct involvement of orofacial muscles in the genesis and persistence of sleep disorders. Although no known study has yet addressed the impact of muscle conditioning on sleep disorders a few theories can be postulated. The Genioglossus muscle (GG) is the most important muscle for airway patency at night and suffers the greatest physiologic damage during OSA. The GG is also the main muscle trained by orofacial myologists in tongue repositioning (tongue on the spot). Theoretically, by repositioning the tongue, the tone would increase and maintain during sleep. Also, if it is possible to increase tonus in muscles by training them, such as lip pulls, tongue push against tongue depressor, masseters contraction during chewing, soft palate/tongue posterior contact during gargling/blowing exercises, the newly acquired tonus should theoretically prevent the muscles from nighttime collapse. If we accept the theory that all orofacial muscles should be in a balanced relationship with one another to prevent OSA, then, by strengthening

hypotonic muscles (lips, masseters, soft palate, tongue) and weakening hypertonic ones (mentalis, lips, masseters) the concerted movement and tone of the orofacial muscles should continue during the sleep hours. Kuehn (1997), who researched the effectiveness of exercises for the soft palate, pointed out that while it is easy to train orofacial muscles against resistance, such as a button or a tongue depressor, traditional exercises for the soft palate such as gargling and blowing do not increase soft palate tonus. Theoretically, by using the CPAP as exerciser, the soft palate would work against resistance (the air blown by the CPAP) and increase its tonus, hopefully carrying it over during nighttime. This theory deserves further study.

All these theories could be verified by a multidisciplinary approach. Only sleep specialists can diagnose sleep disorders and, for the time being, only universities have the possibility to carry on research on the effectiveness of orofacial myology on sleep disorders. There is the need for grants

to support research on the application of OFM in treating sleep disorders, since polysomnography is quite expensive. There is the need for orofacial myologists to let the sleep specialists know that there is something worth investigating, such as muscle conditioning to treat sleep disorders. Finally, sleep studies could provide the scientific basis to the claim of habituation continuing during sleep. Clinicians have plenty of anecdotal observations but only a sleep study could indeed establish the accuracy of such a statement.

Orofacial myologists have much to gain from research on muscle behavior during sleep. It could open the door for an interesting and fruitful collaboration, similar to the one already existing between myofunctional therapists and orthodontists.

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