



The role of oral myofunctional therapy in managing patients with mild to moderate obstructive sleep apnea

Hemmat Baz,¹ Mohsen Elshafey,² Shawky Elmorsy,³ Mohamed Abu-Samra³

¹Phoniatrics Unit, ²Thoracic Medicine Department ³ENT Department. Faculty of Medicine, Mansoura University, Egypt

Correspondence to: Shawky Elmorsy, Email: shawky_morsy2003@yahoo.com

Introduction: *Oral Myofunctional therapy could be considered as a new therapy for snoring and obstructive sleep apnea syndrome (OSAS), because of its direct action on oral motility.*

Aim of this work: *to evaluate the effect of Oral Myofunctional therapy as a simple method for treatment of patients with mild to moderate Obstructive Sleep Apnea Syndrome (OSAS).*

Materials and methods: *30 patients with mild to moderate OSAS were subjected to the following: 1-Thorough history taking with stress on symptoms of OSAS (snoring, fragmented sleep, witnessed apneas, morning headache and daytime sleepiness) and calculation of Epworth sleepiness scale. 2-Physical examination with stress on neck circumference, body mass Index (BMI), and vocal tract examination to exclude space occupying lesions in the nose, mouth and larynx. 3-Full night polysomnography for objective diagnosis of OSAS 4- All patients were treated by Oral myofunctional therapy. 5- Full night polysomnography repeated 3 month after the myofunctional therapy.*

Results: *There was significant decrease of apnea hypopnea index (AHI), arousal index after myofunctional therapy as compared to before myofunctional therapy ($p < 0.001$ for all). Also there were significant decrease in desaturation parameters (desaturation index, average duration SaO₂ < 90%, % total sleep time SaO₂ < 90%) after myofunctional therapy ($p < 0.001$).*

Conclusion: *myofunctional therapy can achieve subjective improvement in OSAS symptoms, as well as polysomnographic abnormalities in patients with mild to moderate OSAS and so can be considered as an alternative method of treatment.*

Keywords: *Myofunctional therapy, Obstructive Sleep Apnea Syndrome, Snoring.*

INTRODUCTION

Obstructive sleep apnea Syndrome (OSAS) is characterized by recurrent episodes of upper airway collapse and obstruction during sleep. These episodes of obstruction are associated with recurrent oxyhemoglobin desaturations and arousals from sleep.⁽¹⁾ The upper airway is a compliant tube and, therefore, is subjected to collapse.⁽²⁾

Most patients with obstructive sleep apnea (OSAS) demonstrate upper air way obstruction at either the level of the soft palate, or the level of the tongue. Research indicates that both anatomic factors (e.g., enlarged tonsils; volume of the tongue, soft tissue, or lateral pharyngeal walls; length of the soft palate; abnormal positioning of the maxilla and mandible) and/ or associated neuromuscular insults are the main etiological factors predisposing to OSAS.^(3,4)

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Many treatment modalities had been implicated in managing OSAS including; Continuous positive airway pressure which is considered the most effective line of treatment in cases of OSAS patients, mainly those who present expressive drop of the oxygen saturation (SaO₂), moderate OSAS and severe OSAS.⁽⁵⁾

Intra-oral devices, lingual retainers, and jaw (mandible) positioners are indicated in OSAS patients from mild to moderate, and retrognathic OSAS patients who are not above the ideal weight, and have not severe oxemoglobin desaturations.⁽⁶⁾

Surgical techniques that vary from otorhinolaryngologic surgeries (as Laser-assisted uvulopalatoplasty (LAUP), or Radiofrequency-assisted uvulopalatoplasty (RAUP)), to maxillary functional orthopedic surgeries present variable results.⁽⁷⁾ The most common procedures reach between 40% and 50% of efficiency and many times more than one technique must be combined, as one- stage or two- stage procedure.

Another form of the treatment modalities is the loss of corporal weight through diet regimen or surgery, ponderal reductions of 10% of corporal weight may lead to the reduction of up to 50% of AHI, and with 20% of corporal weight loss, the patient may become asymptomatic. However, the long term success rates are discouraging with the regaining of weight and reappearance or aggravation of the OSAS.⁽⁸⁾

The treatment modalities aforementioned may act in a palliative way, since they may not effectively treat the factor that precipitates the installation of the OSAS, or they are of difficult acceptance by the patients (high cost, long term difficult maintenance, etc.). Thus, in the pursuit of searching for other therapeutic methods, the hypothesis of the phoniatics intervention in managing OSAS through the myofunctional therapy was raised, to bring benefits for these patients. As in patients with OSAS there was a significant reduction of the muscular tonus and increase of the resistance of the upper airway during sleep.⁽⁵⁾ It is also believed that the dilating force of the upper airway muscles is the only force responsible for counterbalance the forces which promotes the collapse, represented by the negative pharyngeal transmural pressure and for the weight of the structures which form the upper airway, these facts justify the rehabilitation of the orofacial and pharyngeal musculature of these individuals.⁽⁵⁾

Aim of the work: The aim of this work was to evaluate the effect of oral myofunctional therapy as a simple method for treatment of patients with mild to moderate obstructive sleep apnea syndrome.

PATIENTS AND METHODS

This prospective study was conducted upon 40 patients diagnosed as having mild to moderate OSAS. Ten patients were excluded from the study because they were reluctant in performing the oral myofunctional therapy (2008-2011). Approval of ethic committee and informed consent were obtained. The study excluded patients with Body mass index > 40, craniofacial malformation, using hypnotics, hypothyroidism, previous stroke, neuromuscular disease, heart failure, coronary artery disease, severe OSAS, physical obstruction in nose or throat, abnormally large tonsils, uncorrected deviated septum, drug/alcohol abuse, depression, and previous treatment for snoring (surgical or non-surgical). All participants were underwent the following:

1. Thorough history taking with stress on symptoms of OSAS (snoring, fragmented sleep, witnessed apneas, morning headache and daytime sleepiness) and calculation of Epworth sleepiness scale.
2. Physical examination with stress on neck circumference, body mass index (BMI), vocal tract examination to exclude space occupying lesions in the nose and mouth, and dental examination (teeth and gum).
3. Full night polysomnography (Jaeger sleep screen) for objective diagnosis of OSAS, performed in a sleep center in the presence of specially trained technicians. During polysomnography, multiple body functions are monitored. Sleep stages are recorded via an electroencephalogram, electrooculogram, and chin electromyogram.

Breathing is monitored, including airflow at the nose and mouth (using both a thermal sensors and a nasal pressure transducer), effort (using inductance plethysmography), and oxygen saturation.

The breathing pattern is analyzed for the presence of apneas and hypopneas, (definitions have been standardized by the American Academy of Sleep Medicine (AASM)), this breathing pattern analysis is repeated later on 3 months after the oral myofunctional therapy.

4. Oral myofunctional therapy including variety of training strategies according to Galye Burditt 9 and Cuimaraes et al. 10 from these strategies we focused on certain training strategies to the tongue, soft palate, and the pharynx, aiming at increasing the tone and endurance of the targeted muscles. The therapy were given to patients on three months period, twice sessions weekly in a hierarchal manner, providing to teach the patients to practice the therapy regularly at home by a rate of three to five times per day with minimum 10 minutes for each time.

Oral myofunctional therapy can be divided into:**A) Non articulatory oral myofunctional therapy:**

1. Tongue stabilization:
 - Push tongue tip forward just in front of lips without touching teeth or lips for about 30 seconds.
 - Spread centre of the tongue, so the sides of the tongue touch bottom of upper teeth for about 30 seconds.
2. Tongue protrusion outside the mouth (tip forward, tip lift and tip down).
3. Tongue lateralization: push tongue to right/left corner of the mouth keep it pointed
4. Tongue elevation:
 - Place tongue tip as far as possible on the roof of the mouth.
 - Place tongue on the roof of the mouth with tip against upper front teeth while sucking it against roof of the mouth.
5. Holding the tongue tip between teeth anteriorly while trying to swallow
6. Resistive therapy in which tongue press against palate and against hand resistance applied to the cheek on both sides.
7. Palatal elevation with and without yawn (to feel the soft palatal lift).

B) Articulatory therapy:

1. Production of Lingovelar sounds (produced by contacting the dorsum of the tongue and the velum) G, K, separately several times each.
2. Production of Uvular sounds Y, X, and Q (produced by contraction of the uvula) separately several times each.

Statistics: Data were analyzed using SPSS (Statistical Package for Social Sciences) version 10. Qualitative data was presented as number and percent. Comparison between groups was done by Chi-square test. Normally distributed data was presented as mean \pm SD. Pearson's correlation coefficient was used to test correlation between variables. $P < 0.05$ was considered to be statistically significant.

RESULTS

The mean age of the studied cases was 44.07 ± 7.54 years, 73.3% were males and 26.7% were females, mean BMI was 33.59 ± 1.98 mean neck circumference was 42.77 ± 1.67 cm (Table 1). There was significant improvement of OSAS symptoms (snoring, excessive daytime sleepiness, morning headache) after oral myofunctional therapy as compared to before the therapy ($p=0.008, 0.003, 0.014$ respectively) while there were lower percentages of nocturnal choking and witnessed apnea after oral myofunctional therapy as compared to before myofunctional therapy but without statistical significance ($p=0.083$ and 0.083 respectively) (Table 2). There were significant decrease in neck circumference and ESS after oral myofunctional therapy as compared to before the therapy ($p < 0.001$ for both) while there were no significant change in BMI ($p=0.232$) (Table 3). There were significant decrease of AHI, arousal index and % total sleep time in snoring after myofunctional therapy as compared to before myofunctional therapy ($p < 0.001$ for all). Also there were significant decrease in desaturation parameters (desaturation index, average duration $SaO_2 < 90\%$, % total sleep time $SaO_2 < 90\%$) after myofunctional therapy as compared to before myofunctional therapy ($p < 0.001$ for all), while there was significant increase in minimum SaO_2 % ($p=0.006$) after myofunctional therapy as compared to before myofunctional therapy (Table 4). There were significant positive correlation between changes of AHI and changes of neck circumference ($r=0.561$ $p < 0.001$) while no significant correlation between changes of AHI and changes of BMI ($r = 0.418$ $p=0.121$) during the period of the oral myofunctional therapy (Table 5).

Table 1. Demographic data of the studied cases.

	Mean \pm SD
Age (years)	44.07 \pm 7.54
BMI (kg/m²)	33.59 \pm 1.98
Neck circumference(cm)	42.77 \pm 1.67
Sex [No (%)]	
Male	22 (73.3%)
Female	8 (26.7%)

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Table 2. Symptoms of studied cases with OSA before and after upper airway exercises.

Symptoms	Before upper airway exercises		After upper airway exercises		P value
	No	%	No	%	
Snoring	30	100	16	53.3	0.008
Excessive day time sleepiness	30	100	12	40	0.003
Morning headache	18	60	6	20	0.014
Nocturnal choking	12	40	6	20	0.083
Witnessed apnea	10	33.3	4	13.3	0.083

Table 3. Anthropometric variables and ESS before and after upper airway exercises.

	Before upper airway exercises Mean \pm SD	After upper airway exercises Mean \pm SD	P value
Neck circumference(cm)	42.77 \pm 1.67	42.01 \pm 1.96	< 0.001
BMI(kg/m ²)	33.59 \pm 1.98	33.50 \pm 2.04	0.232
ESS	16.40 \pm 1.96	9.27 \pm 2.89	< 0.001

Table 4. Polysomnographic variables before and after upper airway exercises.

	Before upper airway exercises Mean \pm SD	After upper airway exercises Mean \pm SD	P value
AHI	22.27 \pm 4.51	11.53 \pm 5.38	< 0.001
Desaturation index	14.53 \pm 5.04	9.27 \pm 4.27	< 0.001
Average duration SaO ₂ < 90%	18.27 \pm 6.79	9.40 \pm 3.29	< 0.001
% total sleep time SaO ₂ < 90%	2.01 \pm 1.22	1.09 \pm 0.72	< 0.001
Minimum Sao ₂ %	84 \pm 4	87 \pm 5	= 0.006
Arousal index	28.87 \pm 8.41	15.33 \pm 6.11	< 0.001
% total sleep time snoring	14.05 \pm 4.89	3.87 \pm 4.12	< 0.001

Table 5. Correlation of changes of AHI with changes of neck circumference and BMI.

	Changes in AHI	
	R	P
Changes in neck circumference (cm)	0.561	< 0.001
Changes in BMI(kg/m ²)	0.418	=0.121

DISCUSSION

Maintenance of pharyngeal patency during breathing requires the coordinated activity of pharyngeal and thoracic respiratory muscles. During inspiration, subatmospheric pressures are produced in the upper airway as a result of inspiratory muscle contraction. The tendency for the pharyngeal lumen to collapse is opposed by the activation and contraction of the upper airway muscles including dilators, such as the sternohyoid and the omohyoid, and pharyngeal lumen regulators, such as the genioglossus and digastric muscles.⁽¹¹⁾ Genesis of OSAS is multifactorial and includes anatomical and physiological factors. Upper airway dilator muscles are crucial to the maintenance of pharyngeal patency and may contribute to the genesis of OSAS.⁽¹²⁾ the aim of this work was to evaluate the effect of myofunctional therapy as a simple method for treatment of patients with mild to moderate obstructive sleep apnea syndrome. Out of 40 cases, 30 cases with mild to moderate OSAS were eligible for the study and completed the course of oral myofunctional therapy (3 months). The symptoms of OSAS (snoring excessive daytime sleepiness and morning headache) showed significant decrease after myofunctional therapy as compared to before myofunctional therapy ($p=0.008$, 0.003 , and 0.014 respectively) also the significant decrease in % total sleep time of snoring ($p<0.001$) and in Epworth sleepiness scale ($p<0.001$) confirmed the subjective significant improvement in snoring and excessive daytime sleepiness. These were in accordance to Cuimaraes et al.⁽¹⁰⁾ who reported significant improvement in snoring frequency and intensity (by using visual analogue scale) and in excessive daytime sleepiness (by Epworth sleepiness scale) $p=0.001$, 0.001 and 0.006 respectively after use of myofunctional therapy for 3 months. Also this was in accordance to Puhan et al.⁽¹³⁾ who reported improvement in snoring and daytime sleepiness after 4 months training of upper airway muscles by didgeridoo playing (a wooden wind instrument that is may be from 3-10 feet in length which is common among the indigenous people of northern Australia). Ojay and Ernest⁽¹⁴⁾ by using singing therapy reported some improvement in the mean value of recorded snoring per hour slept (pretreatment 6.1 ± 1.8 minutes versus post treatment 5.1 ± 2.6 minutes, mean reduction 17.6%) post therapy ($p=0.04$). This can be explained by the existence of elongated and floppy soft palate and uvula, enlarged tongue and inferior displacement of hyoid bone in OSAS.⁽¹⁵⁾ Specific therapy were developed targeting tongue repositioning. The facial muscles are also recruited during chewing and were also trained with intention of training muscles that promote mandibular elevation, avoid mouth opening and so may affect the propensity to myofunctional edema and collapsibility.⁽¹⁶⁾ There was significant decrease in neck circumference after oral myofunctional therapy in comparison to before myofunctional therapy ($p<0.001$) and this decrease

correlate positively with the decrease in AHI ($p=0.029$) while BMI does not change significantly after myofunctional therapy ($p=0.232$) and did not correlate significantly with changes in AHI ($p=0.121$). This was in accordance to Cuimaraes et al. 10 who reported significant decrease in neck circumference ($p = 0.01$) but no significant change in BMI ($p=0.65$) after myofunctional therapy. This illustrate that the changes in neck circumference cannot be attributed to changes in BMI during this period of therapy as there were no significant changes in BMI and so these changes in neck circumference can be attributed to myofunctional remodeling. Carrera et al.⁽¹⁷⁾ reported that snoring and OSAS patients have a prevalence of type 11 muscle fiber, probably because of inflammatory trauma promoted by vibration, affecting and decreasing the myofunction of upper airway. Blottner et al.⁽¹⁸⁾ reported that improvement of muscle tone by physical training was associated with increase in the proportion of type I muscle fibers and in the size of type 11 muscle fibers as demonstrated by muscle biopsy (type 1 having endurance and type 11 having speed capability). Methods to increase muscle tone of the upper airway are based on gain of endurance and strength properties. So increase in type 1 muscle fibers by therapy resulting in improvement in OSAS manifestation. There was significant decrease in AHI and arousal index after myofunctional therapy ($p<0.001$ for both). This was in accordance to Cuimaraes et al.⁽¹⁰⁾ who reported significant decrease in apnea index and hypopnea index ($p=0.004$, 0.007 respectively). The decrease in AHI in our study was from 22.27 to 12.93 events/ hour which represent 41.9% decrease while in study of Cuimaraes et al.⁽¹⁰⁾ it was from 22.4 to 13.7 events/ hour which represent 39.3% decrease. this decrease in AHI approach what reported by review about mandibular advancement by Hoffstein¹⁹ which was 42%. Pitta et al.⁽⁵⁾ reported improvement in two patients with severe OSAS by application of oral myofunctional therapy for a period of 16 week (a decrease in AHI, ESS, snoring and an improvement in oxygen desaturation). So this gives the potential use of this treatment in patients with severe OSAS especially if CPAP cannot be tolerated by the patients. Puhan et al.⁽¹³⁾ reported marginal improvement in AHI ($p=0.05$) by using didgeridoo playing for 4 months. This marginal improvement can be explained by the non-specific myofunctional therapy applied by didgeridoo playing in comparison to specified oral myofunctional therapy applied in our study and study of Cuimaraes et al.⁽¹⁰⁾ (myofunctional therapy, tongue therapy and pronounced voice for soft palate). Four cases showed normalization of AHI (AHI <5 events/hour) after oral myofunctional therapy and 14 cases showed $>50\%$ decrease in AHI but without normalization of AHI while 12 cases showed less than 50 % decrease in AHI. The BMI of cases that showed response ($>50\%$ decrease in AHI) ranged from 30-32 while of those that showed no response ($<50\%$ decrease in AHI) ranged from 35-36.7. This illustrate that patients selection is critical for potential

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benefits from therapy (those with low BMI) because obstruction is caused not only by weak and collapsing muscles but also by bulk formed by deposits around the tongue and throat in obese patients. The desaturation index, average duration SaO₂ <90%, % total sleep time SaO₂ <90% were significantly decreased after myofunctional therapy (p<0.001 for all) while the minimum SaO₂ < 90% was significantly increased (from 84 ± 4 to 87± 5, p=0.006). This was in accordance to Cuimaraes et al.⁽¹⁰⁾ who reported significant increase in minimum SaO₂ from 83 ± 6 to 85 ± 7% (p<0.001). This illustrates that only slight improvement occurred in SaO₂ (about 3%) and so this method can be applied to cases of OSAS with slight decrease in SaO₂. The limitation of this study is the application on small number of cases together with the dependence of the results on the compliance of the patients on regular application of the therapy. Another limitation was absence of standardization of the maneuvers and duration of the therapy that can achieve maximum effect. It is not clear how long the therapeutic effects in the responders persist and whether a longer duration of training beyond 3 months or repetition of training after an interval might be beneficial.

CONCLUSION

- Oral myofunctional therapy can achieve subjective and objective improvement in OSAS symptoms and their polysomnographic abnormalities in patients with mild to moderate OSAS.
- Oral myofunctional therapy can be considered as alternative method of treatment of mild to moderate OSAS.
- Future studies will be needed to determine optimal treatment elements (i.e., load/intensity, frequency, and duration) and to confirm the hypothesized need for ongoing practice to maintain beneficial treatment effects.

REFERENCES

1. Young T, Evans L, Finn L, Palta M. Estimation of the clinically diagnosed proportion of sleep apnea syndrome in middle-aged men and women. *Sleep*. 1997;20:705-6.
2. Patil SP, Schneider H, Schwartz AR, Smith PL. Adult obstructive sleep apnea: pathophysiology and diagnosis. *Chest*. 2007;132:325-37.
3. Savage CR, Steward DL. Snoring: a critical analysis of current treatment modalities. Does anything really work? *Curr Opin Otolaryngol Head Neck Surg*. 2007;15:177-9.
4. Schwab RJ, Pasirstein M, Pierson R, et al. Identification of myofunctional anatomic risk factors for obstructive sleep apnea with volumetric magnetic resonance imaging. *Am J Respir Crit Care Med*. 2003;168:522-30.
5. Pitta DB, Pessoa AF, Sampaio AL, et al. Oral myofunctional therapy applied on two cases of severe obstructive sleep apnea syndrome. *Intr Arch Otorhinolaryngology*. 2007;ii:350-4.
6. Nowara S, Lowe et al. Oral appliances for treatment of snoring and sleep apnea: a review. *Sleep*. 1995;18:501-10.
7. Lim DJ, Kang SH, Kim BH, Kim HG. Treatment of primary snoring using radiofrequency-assisted uvulopalatoplasty. *Eur Arch Otorhinolaryngol*. 2007;264:761-7. Epub. 2007.
8. Charuzi I, Ovnat A, Peiser J, et al. The effect of surgical weight reduction in sleep apnea quality in obesity-related sleep apnea syndrome. *Surgery*. 1985;95:535.
9. Galye Burditt. Oral sensory and motor treatment. *Visual health information*. 1996. Tacoma, W A. USA.
10. Cuimaraes KC, Drager LF, Genta PR, et al. Effect of oropharyngeal therapy on patients with moderate obstructive sleep apnea syndrome. *Am J Respir Crit Care Med*. 2009;179:962-6.
11. Van Lunteren E, Vafaie H, Salomone RJ. Comparative effects of aging on pharyngeal and diaphragm muscles. *Respir Physiol*. 1995;99:113-25.
12. Schwartz AR, Patil L, Laffan AM, et al. Obesity and obstructive sleep apnea: pathogenic mechanism and therapeutic approaches. *Proc Am Thorac Soc*. 2008;5:185-92.
13. Puhan MA, Suarez A, Cascio CI, et al. Didgeridoo playing as alternative treatment for obstructive sleep apnea syndrome. *BMJ*. 2005;332:266-70.
14. Ojay A, Ernest E. Can singing therapy reduce snoring? A pilot study. *Complement Ther Med*. 2000;8:151-6.
15. Arens R, Marcus CL. Pathophysiology of myofunctional obstruction, a developmental perspective. *Sleep*. 2003;27:997-1019.
16. Chiu KL, Ryan CM, Shiota S, ET AL. Fluid shift by lower body positive pressure increases pharyngeal resistance in healthy subjects. *Am J Respir Crit Care Med*. 2006;174:1378-83.
17. Carrera M, Babe E, Sauleda J. Effect of obesity upon genioglossus structure and function in obstructive sleep apnea. *Eur Respir J*. 2004;23:425-9.
18. Blottner D, Salavnova A, Butmarin B. Human skeletal muscle structure and function preserved by vibration muscle therapy following 55 days of bed rest. *Eur J Appl Physiol*. 2006;97:261-71.
19. Hoffstein V. Review of oral appliances for treatment of sleep-disordered breathing. *Sleep Breath*. 2007;11:1-22.