Myofunctional Therapy
Role in Pediatric OSA

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KEYWORDS

• Obstructive sleep apnea syndrome • Myofunctional therapy • Functional oral appliance
• Lateral cephalometric • Polysomnography

KEY POINTS

- Myofunctional therapy (MFT) has been reported to be an alternative treatment to obstructive sleep apnea (OSA), but compliance and long-term outcome in the children were considered as an issue.
- Compliance is a major problem of MFT, and MFT will have to take into consideration the absolute need to have continuous parental involvement in the procedure for pediatric OSA.
- Passive MFT gives more improved measurements and better compliance, but potential negative effects of device on other jaw will have to be continuously evaluated.

INTRODUCTION

Obstructive sleep apnea (OSA) is a common and serious disorder that affects the health of the general public.[1] The pathophysiology of OSA is complex. Most cyclical and cessations in sleep-disordered breathing events are driven by anomalies in both anatomic and neurochemical control of upper airway and/or chest wall respiratory musculature.[2] Therefore, several medical and surgical treatment modalities exist as treatment for OSA.[3–5] The causes of OSA are different between adult and children. Guilleminault and colleagues[6,7] published a review of 50 pediatric patients, which demonstrated that the clinical features of pediatric OSA were different from that of adults in 1981. Among the types of pediatric sleep-disordered breathing (SDB), OSA has the highest prevalence[8] but its pathophysiology continues to be unclear. Such as adenotonsillar

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hypertrophy, obesity, anatomic and neuromuscular factors, and hypotonic neuromuscular diseases are also involved. Currently, the main cause of pediatric OSA is most commonly hypothesized to be a result of an anatomically or functionally narrowed upper airway. Related research in recent years showed that anatomic features of children with craniofacial anomalies are highly related to pediatric OSA.[9,10] Therefore, the established first-line treatment, adenotonsillectomy (AT), is unable to eradicate pediatric OSA completely, and high levels of relapse continue to be a point of contention.[11] There are very high rates of pediatric OSA in the preterm birth infant.[12] It means the initial oral facial anatomic build-up of an individual has a clear impact on the risk of collapsibility of the upper airway during sleep,[9,12] because there is a continuous interaction between orofacial functions and orofacial growth after birth. The dysfunctions identified to date affect orofacial development leading to SDB through changes in the orofacial growth.[9,10,12,13] Recently myofunctional therapy (MFT) has been suggested as an adjunct treatment of SDB in children.[9] Results of studies done on children with orthodontic problems have shown that isolated extensive and well-controlled MFT can lead to return to normal orofacial anatomy.[14] In adults, there is reported improvement of OSA and snoring also.[15–17] But the use of MFT alone when dealing with pediatric SDB has not been widely investigated and the long-term effects of MFT on SDB are still unknown. Problems with MFT have been reported: (1) current forms of MFT are difficult for children younger than 4 years, (2) the poor compliance with daily exercises and the absence of continuous parental involvement with the training exercises of the child are major causes of failure of treatment. Preliminary studies indicated that usage of a functional oral appliance thought to induce some extra muscle activity while asleep (also called "passive myofunctional therapy" [PMFT]) may decrease mouth breathing and have an impact on the position of the mandible.[16,17] Such changes may lead to improvement of the narrow upper-airway during sleep and may improve the snoring and pediatric OSA. The authors' study explored the effectiveness of MFT and PMFT on pediatric OSA and the long-term effects of these approaches on OSA and craniofacial growth.

METHODS

The study had the following design:

A. Patient recruitment
   1. Children between 4 and 16 years of age, diagnosed with OSA based on clinical evaluation and nocturnal, in-laboratory polysomnography (PSG) with an apnea–hypopnea index (AHI) greater than 1 event/h or a respiratory disturbance index (RDI) greater than 5event/h; and
   2. Either children previously diagnosed with pediatric OSA who had undergone adenotonsillectomy, but with residual AHI greater than 1 event/h or RDI greater than 5 event/h at 6 months after tonsillectomy and adenoidectomy (T&A) or children diagnosed with pediatric OSA but without evidence of adenotonsillar hypertrophy after ear, nose, and throat evaluation.
   3. Recruited children were age matched and were treated (1) either with MFT (total 20 min/d) for 0.5 year (b) or with a previously described and specifically designed oral appliance with a tongue bead[18] during sleep (passive MFT) for 0.5 year. The appliance is a one-piece, custom-made adjustable oral device for advancing the mandible. A bead is mounted on the lower part of the frame for the tip of the tongue to roll, which in turn places the tongue in a forward position so as to open the airway.(c) The authors also recruited one age-match control group that did not receive any treatment and agreed to follow-up.

B. Procedures

   Evaluation of the 3 groups was planned to be at baseline and subsequently at 3 and 6 months after MFT or PMFT treatment, with clinical evaluation, PSG, and lateral cephalometric films evaluating bone structure development at baseline, and 6 months.

C. PSG during sleep

   A Neurovirtual BWIII PSG Plus sleep systemTM (Fort-Lauderdale. Fl. USA) was used. The following variables were recorded: electroencephalography (F3-M2, F4-M1, C3-M2, C4-M1, O1-M2, O2-M1), electro-oculogram (EOG), chin and leg electromyography (EMG), electrocardiography (ECG) with a modified V2 lead, body-position sensor, nasal cannula/pressure transducer, mouth thermistor, thoracic and abdominal plethysmography bands, neck microphone, and finger pulse oximetry.

   Scoring was performed by an individual not involved in the study and blind to all conditions.

D. MFT

   MFT is composed of isotonic and isometric exercises that target oral (lip, tongue) and oropharyngeal structures (soft palate,

lateral pharyngeal wall). MFT[14,17–19] aims to obtain appropriate head posture and positioning of the tongue on the palate against the upper teeth, appropriate swallowing and mastication using both sides and posterior chewing, appropriate breathing through the nose while keeping the mouth closed, and appropriate speech and articulation.

1. For soft palate exercises, patients pronounce oral vowel sounds either continuously (isometric exercises) or intermittently (isotonic exercises).[15]
2. Tongue exercises include moving the tongue along the superior and lateral surfaces of the teeth, positioning the tongue tip against the anterior aspect of the hard palate, pressing the entire tongue against the hard and soft palate, and forcing the tongue onto the floor of the mouth.[15]
3. Facial exercises address the lip (ie, contraction and relaxation of the orbicularis oris), buccinators (ie, suction movements and application of intraoral finger pressure against the buccinator muscles), and jaw muscles (ie, lateral jaw movements).[15]
4. Stomatognathic functions are addressed by instructing patients to inhale nasally and exhale orally without and then with balloon inflation and performing specific swallowing and chewing exercises (ie, swallowing with the teeth clenched together, tongue positioned in the palate, and without contraction of perioral muscles; alternating chewing sides).[15]

For children, MFT requires active parental involvement to obtain good results. Parents and children had initial training with a specialist and parents were asked to supervise a minimum of 20 minutes of exercise daily. The MFT specialist was available to parents and regular clinical follow-up was scheduled. This regular daily activity was called "active MFT."[18]

E. Oral appliance

The "passive MFT" short term used was reported recently.[18] The amount of mandibular advancement associated with the wearing of the device at rest-supine awake was 50% of the maximum mandibular advancement. Patients were instructed to wear their appliance nightly, the appliance been placed just at bedtime and to use their tongue to roll the bead (ie, passive MFT) just before falling asleep. Parents kept sleep logs to record the nightly wear by all children for 12 months.

F. Statistical analysis

General descriptive statistics, ANOVA, Wilcoxon signed-rank test, and Mann-Whitney analyses of the treatment results from the lateral cephalometric X ray, and PSG reports were performed using SPSS-18.

RESULTS

Total 121 pediatric OSA were enrolled. Fifty-four children were received MFT (Group A). But only 23 children completed the 6-month PSG study. Out of these 23, only 10 children had good compliance (compliance rate >80%) and completed pre- and postcephalometric test. Group B enrolled 56 pediatric OSA who received oral device (PMFT) treatment and 50 children completed 6 months of research with good compliance (>80%). The data showed no significant differences in age (groupA:7.02 $\pm$ 2.44; group B:7.97 $\pm$ 3.08, P = .338), sex (50% boy; 64.6% boy; P = .37), and BMI (16.61 $\pm$ 1.74; 17.82 $\pm$ 4.45, P = .10) distributions between the 2 groups. According to the questionnaires administered with oral device, children accepted nightly use of the device without complaint or objection. No side effect was reported by children or parents.

Considering the very important "drop-out" rate of subjects in Group A, the authors firstly performed analyses on Group A subjects. They considered each subject as its own control and compared baseline with posttreatment. PSG results are presented in **Table 1**. At 6-month follow-up, significant differences were already noted involving the respiratory disturbance index (P = .032), respiratory effort–related arousal (P = .048), and sleep latency (P = .036) that showed significant improvement after 0.5 year of treatment.

Interestingly the patients who received MFT and who had good compliance (n = 10) showed also significant improvement in AHI, PNS-NPhp (distance between PNS and posterior side of nasopharynx), and PNS-AD2 (distance from PNS to the nearest adenoid tissue measured along the line perpendicular to S-BA) measurements. Compared with no-treatment control group, the good compliance children in the Group A showed significant improvement in AHI (P = .037) (**Table 2**).

Table 3 shows the PSG outcome of 0.5 year of treatment with PMFT. Many significant findings were noted: significant decrease in the AHI during total sleep (P = .003), the hypopnea index (P = .003), the arousal index (P = .035), and the hypopnea count (P = .011).

Table 1
Polysomnography results of treatment with MFT

	PSG Pre (n = 23) Mean ± SD	PSG-0.5 y (n = 23) Mean ± SD	*P*-Value
BMI	16.61 ± 1.74	16.76 ± 2.12	.589
AHI in sleep (event/h)	2.47 ± 1.31	2.26 ± 1.84	.492
AHI in REM (event/h)	5.27 ± 4.15	4.99 ± 6.18	.799
AI (event/h)	1.43 ± 1.04	1.03 ± 02	.082
HI (event/h)	1.05 ± 0.92	1.23 ± 1.25	.518
ODI	1.64 ± 1.12	1.71 ± 2.22	.877
RDI	3.53 ± 2.01	2.57 ± 1.76	0.032*
Efficiency %	89.37 ± 5.01	90.41 ± 78.32	.312
Awake %	6.67 ± 4.58	7.04 ± 5.17	.759
REM %	24.31 ± 4.66	20.13 ± 7.0	.022*
Stage 1%	8.12 ± 4.43	12.15 ± 6.83	.041*
Stage 2%	38.82 ± 7.71	39.47 ± 9.87	.736
Stage 3%	28.75 ± 6.63	27.38 ± 7.53	.485
Total sleep time (min)	394.43 ± 22.73	398.33 ± 21.72	.504
Awake Count	12.70 ± 4.36	14.35 ± 5.52	.170
Sleep Latency (min)	16.20 ± 12.83	10.78 ± 9.49	.036*
REM Latency (min)	64.5 ± 27.16	94.5 ± 56.34	.023*
Arousal Count	43.83 ± 14.27	40.52 ± 11.15	.321
Arousal Index	7.96 ± 2.74	7.2 ± 1.97	.219
Mean Heart Rate	82.14 ± 14.04	78.23 ± 10.67	.098
PLM Index (event/h)	0.30 ± 1.00	1.14 ± 2.66	.124
Snore Index (event/h)	136.32 ± 144.80	122.38 ± 123.58	.683
Obstructive Apnea	0.39 ± 0.78	0.48 ± 0.99	.648
Central Apnea	6.87 ± 5.50	4.91 ± 5.53	.161
Mixed Apnea	0.74 ± 1.86	0.48 ± 0.85	.503
Hypopnea	5.87 ± 5.11	6.91 ± 7.17	.515
RERA	5.87 ± 8.96	1.74 ± 2.78	.048*
Mean SaO2%	97.74 ± 0.62	97.83 ± 0.39	.539

t-test was performed to analyze the 0.5 year of treatment results.

Abbreviations: AI, apnea index; HI, hypopnea index; mean SaO2, mean oxygen saturation; ODI, oxygen desaturation index; PLMS index, periodic leg movements in sleep index; RDI, respiratory disturbance index; REM, rapid eye movement; RERA, respiratory effort–related arousal.

* *P*<.05.

The authors could compare only the children with good compliances for MFT (ie, n = 10) and the subjects who used the device regularly (ie, n = 48). It is obvious that the improvement of AHI and upper airway of group B (passive MFT) is more than group A (MFT) (see **Tables 1** and **3**).

DISCUSSION

In adults, there is reported improvement of OSA and snoring under MFT, but MFT alone when dealing with pediatric SDB has not been widely investigated.[15–17] Moreover, the long-term effect on SDB is unknown. Prior publications looking at role of MFT in improving sleep-related breathing had emphasized the very large amount of nonusable data.[15,17,19–21] Compliance with treatment is a major issue. In prior studies the authors learned that it is very important, particularly at the beginning of treatment, to see child and parents several times a week and to involve in depth one of the parents who will perform the exercise at the same time as the child. Such involvement may be costly depending of the social security system and the reimbursement provided for the specialist involvement. The daily involvement of at least one parent with performance of the

Table 2
Comparison of the difference of cephalometric outcome between the treatment with myofunctional therapy (good compliance) and control group

	MFT (Good Compliance) (n = 10)			Control (n = 11)					
	Ceph-Pre Mean ± SD	Ceph-0.5 y Mean ± SD	*P* Value	Ceph-Pre Mean ± SD	Ceph-0.5 y Mean ± SD	*P* Value	MFT 0.5y-Pre Mean ± SD	Control 0.5y-Pre Mean ± SD	P_T Value
Age	6.8 ± 1.99	—	—	8.46 ± 3.00	—	—	—	—	0.443
Gender (Male %)	40.0%	—	—	72.7%	—	—	—	—	0.096
AHI (event/h)	2.46 ± 1.52	1.46 ± 1.41	.015*	2.42 ± 2.83	2.59 ± 2.11	.766	−1.00 ± 1.18	0.17 ± 2.09	0.037*
AI	1.11 ± 0.94	0.60 ± 0.61	.249	0.76 ± 0.78	0.93 ± 0.55	.483	−0.51 ± 1.08	0.23 ± 0.94	0.246
ODI	1.60 ± 1.04	1.04 ± 1.49	.208	2.13 ± 3.07	2.04 ± 2.37	.859	−0.56 ± 1.82	0.23 ± 3.00	0.248
HI	0.78 ± 0.54	0.49 ± 0.45	.058	2.32 ± 3.03	1.40 ± 1.96	.483	0.29 ± 0.46	−0.83 ± 3.21	0.595
ANB	6.67 ± 2.16	6.60 ± 2.02	.779	4.92 ± 2.19	4.78 ± 2.46	.441	−0.07 ± 0.82	−0.14 ± 1.37	0.620
PNS-NPhp	11.64 ± 3.81	13.66 ± 3.81	.028*	12.55 ± 4.12	14.26 ± 4.16	.131	2.02 ± 2.45	1.70 ± 4.40	0.526
PMm-NPh	6.40 ± 3.30	7.55 ± 3.00	.103	9.75 ± 2.90	9.78 ± 3.76	.859	1.15 ± 1.82	0.02 ± 2.88	0.439
OPha-OPhp	11.87 ± 5.67	9.63 ± 2.07	.241	13.44 ± 3.96	11.18 ± 1.71	.041*	−2.25 ± 5.51	−2.26 ± 3.17	0.647
MinRGA	12.87 ± 3.33	11.99 ± 2.20	.508	14.31 ± 3.49	12.72 ± 2.78	.213	−0.88 ± 3.25	−1.59 ± 3.35	0.673
PNS-AD2	11.15 ± 2.58	13.23 ± 2.99	.028*	12.82 ± 3.97	14.12 ± 3.07	.026*	2.09 ± 2.23	1.30 ± 1.47	0.324

Abbreviations: AI, apnea index; ANB, A point-nasion-B point angle (normal range is about 2–4); HI, hypopnea index; MinRGA, minimal retroglossal airway; ODI, oxygen desaturation index; OPha-OPhp, distance anterior side-posterior side of oropharynx; P, Intragroup comparison: Wilcoxon signed-rank test; PMm-NPh, distance soft palate-posterior side of nasopharynx; PNS-AD2, distance from PNS to the nearest adenoid tissue measured along the line perpendicular to S-BA; PNS- NPhp, distance between PNS and posterior side of nasopharynx; P_T, intergroup comparison: Mann-Whitney test for difference between groups.

* $P<.05$.

Table 3
Polysomnography results of treatment with oral device (passive myofunctional therapy)

	PSG Pre(n = 50) Mean ± SD	PSG 0.5 y(n = 50) Mean ± SD	*P*-Value
BMI	17.82 ± 4.45	18.26 ± .50	.124
AHI in sleep	5.56 ± 6.65	2.85 ± 2.45	.003**
AHI in REM	9.64 ± 12.32	7.59 ± 9.29	.126
AI	1.47 ± 2.25	1.01 ± 1.12	.131
HI	3.46 ± 4.45	1.84 ± 1.95	.003**
ODI	4.03 ± 6.72	2.71 ± 3.17	.133
RDI	7.41 ± 10.85	5.34 ± 4.11	.237
Efficiency %	86.01 ± 11.44	87.85 ± 9.77	.328
Awake %	10.57 ± 11.11	7.89 ± 7.06	.130
REM %	20.44 ± 4.99	19.35 ± 5.29	.054
Stage 1%	9.29 ± 7.38	9.47 ± 6.52	.867
Stage 2%	43.36 ± 8.91	44.87 ± 8.40	.336
Stage 3%	25.89 ± 9.14	26.29 ± 8.04	.780
Total Sleep Time	389.37 ± 59.76	390.16 ± 60.73	.947
Awake Count	17.67 ± 10.13	17.71 ± 11.41	.984
Sleep Latency	14.08 ± 12.07	17.44 ± 20.30	.312
REM Latency	108.49 ± 74.83	95.74 ± 57.51	.280
Arousal Index	11.02 ± 8.50	9.02 ± 5.17	.035*
Mean Heart Rate	74.24 ± 13.19	69.94 ± 14.81	.058
PLM Index	0.99 ± 2.74	0.56 ± 1.58	.267
Snore Index	142.65 ± 205.37	123.78 ± 155.01	.477
Obstructive Apnea	3.02 ± 7.83	1.42 ± 3.68	.127
Central Apnea	4.20 ± 6.01	4.02 ± 4.85	.841
Mixed Apnea	1.34 ± 3.93	0.50 ± 1.18	.166
Hypopnea	17.96 ± 23.14	10.54 ± 11.18	.011*
RERA	17.79 ± 20.74	15.33 ± 18.95	.464
Mean SaO2%	97.38 ± 0.67	96.93 ± 4.24	.467
WASO	32.36 ± 4.76	28.93 ± 28.19	.631

PSG-0.5 y: PSG was performed with usage of oral device at the time of recording.
ANOVA. Post hoc: Bonferroni test.
Abbreviations: AI, apnea index; HI, hypopnea index; Mean SaO2, mean oxygen saturation; ODI, oxygen desaturation index; PLMS index, periodic leg movements in sleep index; RDI, respiratory disturbance index; RERA, respiratory effort–related arousal.
* $P<.05$.
** $P<.01$.

exercises at the same time in the morning and/or evening with the child is a critical issue. Social factors are important in MFT—often both parents work outside; families may have several children; work and school schedules—but, particularly in Far East Asian countries, the very large demands from schools and very demanding home-work time are clear handicap to parental involvement in daily reeducation programs. Even in the 10 families where good compliance was noted at 6 months, the authors were unable to pursue their long-term follow-up. MFT is one of the rehabilitations, and there is no way to get the immediate results; therefore many parents or patients easily give up after 3 months of treatment. The "passive MFT" requires very little involvement of parents, and children are very quickly used of having the device in their mouth, so compliance is not a problem. But placing any dental device in a mouth of a child is always an issue: any effect induced on one of the jaw will lead quickly to a compensatory effect on the other one. Many

devices are advertised for children, but very little, if any, study has been performed to appreciate the long-term changes induces on upper and lower jaws when an appliance is used for several months, despite the fact that such changes are always noted to some degree after months of usage. The authors kept their device for 0.5 year and could not see any change at their clinical and imaging evaluations. But they used cephalometric X rays and probably in the future 3-dimensional dental computed tomography will be a better tool. Usage of such devices must be done with clear follow-up and usage must be with clear time limit. Also long-term follow-up without any of these devices is an important issue.

How the device really works has not been completely worked out. It is a type of "functional appliance," and functional appliances have been "advertised" for more than 70 years,[22] as having effects on orofacial growth, but the secondary effects noted when usage is too long and the unavoidable impact on the other jaw has been clear limitation in their usage.

One crucial finding with the PSG at 0.5 year postrecording compared with baseline is that they breathed through their nose during sleep, while mouth breathing was the rule at baseline including in our post-T&A children. All-night nasal breathing is the only demonstration of successful treatment of the upper airway.[23]

Finally, the authors' study is the first prospective study performed on children with pediatric OSA that demonstrate that MFT can lead to nasal breathing during sleep. It also outlined the very important difficulties associated with such treatment. The limitations are the high drop-out rate of MFT group and the authors only followed 6 months side effect of oral device treatment (PMFT). This study indicates that "passive MFT" may be a valid alternative, but very careful attention to the potential negative secondary impact of the device, that may not be immediately appreciated, is a clear requirement.

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