Sys Rev Pharm 2020; 11(6): 522 – 528
A multifaceted review journal in the field of pharmacy
E-ISSN 0976-2779 P-ISSN 0975-8453

A Systematic Review of Oral Myofunctional Therapy for Future Treatment in Pediatric Obstructive Sleep Apnea (OSA)

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Article History: Submitted: 05.04.2020 Revised: 11.05.2020 Accepted: 23.06.2020

ABSTRACT
Obstructive sleep apnea (OSA) is a sleep-related respiratory disorder with a reduction or obstruction of total air flow despite continuous efforts with patents to breathe. The prevalence of OSA is around 2-4% in men and 1-2% in women. The American Academy of Pediatric Dentistry (AAPD) recognizes that obstructive sleep apnea (OSA) occurs in a pediatric population. Oral myofunctional therapy has emerged as an alternative treatment that allows for obstructive sleep apnea (OSA). The aim of this study is to systematically review the literature on oral myofunctional therapy for future care in children with obstructive sleep apnea (OSA). Data source of Web of Science, Scopus, and The Cochrane Library. Studies published from 2011 to 2020 that evaluate treatments with isolated oral myofunctional therapy in subjects with OSA. Sixty articles were assessed, including 60 articles from an electronic database, 50 records were filtered, 30 records were issued, 22 full-text articles were assessed for eligibility and 10 full-text articles were included. All of these articles say that myofunctional therapy has the potential to be an option for OSA treatment. Therefore a systematic literature review shows that oral myofunctional therapy for future treatment of obstructive sleep apnea in children (OSA).
Keywords: Obstructive Sleep Apnea; Myofunctional Therapy; Pediatric
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DOI: 10.31838/srp.2020.6.80

INTRODUCTION

Obstructive sleep apnea (OSA) is a respiratory disorder associated with sleep with obstruction of complete airflow to breathe. Usually occurs during sleep, the couch that causes blockage of the upper airway.[1]Consequently, there are several parts and total breathing pause which lasts at least 10 seconds during sleep.[2]Then, blood oxygen saturation, with oxygen levels dropping, suddenly and drops 50% or more in severe cases. The brain shows a compilation response of little oxygen and reminds the body that causes a brief arousal from sleep. This restores normal breathing patterns.[1] This pattern can occur several times in one night. This results in fragmentation in the quality of sleep and produces excessive daytime sleepiness.[1,2]

Sleep-related disorders breathing encompass a broad spectrum of conditions, including obstructive sleep apnea (OSA), where recurrent partial or complete obstruction of the upper airway occurs. The prevalence of OSA is approximately 2–4% in men and 1–2% in women.[3-7]It has been established that OSA is an independent risk factor for cerebrovascular and cardiovascular disease and can consequently lead to significant morbidity and mortality. Alongside the health-related impacts, OSA can also confer a considerable social and economic burden for those affected,and it has been suggested that the daytime sleepiness and resultant impaired cognitive function may contribute to job-related and motor vehicle accidents. Sleep-related respiratory disorders cover a wide spectrum of conditions, including obstructive sleep apnea (OSA), where partial or complete obstruction of the upper airway occurs. The prevalence of OSA is around 2-4% in men and 1-2% in women.[3-7]It has been established that OSA is an independent risk factor for cerebrovascular and cardiovascular disease and consequently can cause significant morbidity and mortality. In addition to health-related impacts, OSA can also impose significant social and economic burdens on those affected, and it has been suggested that daytime sleepiness and the resulting cognitive function can contribute to work-related accidents and motor vehicles. Separately, moderate to severe OSA has been reported to independently increase the risk of death of all causes. The burden of disease associated with OSA only tends to increase because worldwide obesity rates in the population increase.[1]

The American Academy of Pediatric Dentistry (AAPD) recognizes that obstructive sleep apnea (OSA) occurs in a pediatric population. Undiagnosed and / or untreated OSA is associated with learning problems, cardiovascular complications, growth disorders (including failure to develop), and / or behavioral problems.[8] To reduce these complications, AAPD encourages health professionals to routinely screen their patients for increased risk. for OSA and to facilitate medical referral when indicated.

The pathophysiology of pediatric OSA is often multifactorial, with significant contributions from adenotonsillar hypertrophy, obesity, and genetics. Polysomnography is currently the most accepted diagnostic modality for OSA, but limited availability and high cost limits are routine.[9] At present, there is no consensus among anesthesiologists regarding the best and safest anesthetic techniques for children with significant OSA; there is also a lack of agreement between anesthesiologists, surgeons and institutions on specific criteria for identifying children with OSA who will benefit from hospital admission and aggressive postoperative monitoring after surgery. The purpose of this review is for an overview of oral myofunctional therapies for future treatment in children with obstructive sleep apnea[10]

Diagnosis of OSA in children can be a challenge due to various symptoms that arise. The evaluation of children with suspected sleep disorders begins and, for the most part, ends with a thorough history.[11] OSA clinical presentations include behavioral and neurocognitive disorders, enuresis, cardiovascular sequelae, poor school performance, and headaches, , including systemic and pulmonary hypertension. It is important to note that parental reports alone do not distinguish OSA from simple primary snoring.[12]The accuracy of clinical evaluation of pediatric OSA in predicting poor sleep studies is poor, ranging from 30% to 85% .[13]

Further diagnostic evaluation of a child with a clinical history suggestive of OSA includes polysomnographic performance, which is considered a 'gold standard' for diagnosis and quantitative description of OSA.[14] Children with snoring primary do not have other night and daytime symptoms and have normal sleep polysomnographicstudies.Polysomnography continuously monitors physiological variables during different sleep phases and can distinguish primary snoring from OSA and also provides a more complete description of obstructive events that occur during sleep.[15] Central sleep apnea is characterized by polysomnography in the absence of airflow and respiratory effort. Some patients, especially those with neuromuscular conditions, can display a mixture of centers and OSA[16]

Daytime polysomnography has been used to evaluate children with suspected Sleep Disorders, although normal nap studies are not enough to exclude the diagnosis of OSA in patients with manifestationsclinical that suggest OSA.[17] While the validity of portable monitoring modalities in the diagnosis of OSA in children is still unknown, there is increasing interest in their use in polysomnographic sites to increase access and reduce costs. One example of a portable monitoring modality is nocturnal oximetry, which assesses the severity of OSA by calculating the amount and severity of oxyhemoglobin desaturation during sleep. Isolated heavy desaturation (80%) or desaturation group (more than three episodes, 90%) are considered abnormal. The positive predictive value for oximetry is 97% .[18]

Table 1: McGill Oximetry Assessment System. The severity of OSA is determined by the presence of SpO2 and the number of episodes during nocturnal oximetry. OSA, obstructive sleep apnea[2]

Oximetry Score	OSA Classification	Number of events of SpO2 <90%	Number of events of SpO2 <85%	Number of events of SpO2 <80%
1	Normal/Inconclusive for OSA	<3	None	None
2	Mild	≥3	≤3	None
3	Moderate	≥3	>3	≤3
4	Severe	≥3	>3	>3

Table 2: The severity of OSA by polysomnography (PSG) in children and adults as defined by the American Society of Anesthesiologists Task Force on Perioperative Management of Patients with Obstructive Sleep Apnea. OSA, obstructive sleep apnea; AHI, apnea / hypopnea index.[2]

OSA severity	AHI Children	AHI Adults
None	0	0-5
Mild	1-5	6-20
Moderate	6-10	21-40
Severe	>10	>40

Risk factors for the development of OSA in children include a family history of snoring or OSA, physical abnormalities, cerebral palsy, muscular dystrophy,[19] Down syndrome, sickle cell disease, oral breathing, and any conditions that can cause narrowing in OSA. upper airway. An important common risk factor for OSA is obesity.[20] According to Tauman and Gozal, the recent increase in obesity has led to an increase in OSA prevalence among children, because this condition has been shown to be positively correlated with body mass index.[21]

Symptoms of Pediatric OSA include loud snoring 3 or more nights per week, episodes of respiratory arrest witnessed by others,[22] sudden wakes accompanied by shortness of breath, mouth breathing including symptoms of dry mouth or sore throat, difficulty sleeping with several sleepless nights. day,[23] anxiety, sweating, waking up in the morning feeling not refreshed, and finally, frequent headaches in the morning.[24] Daytime consequences of Sleep Disorder Breathing can affect the nervous system, cardiovascular system and/or the inflammatory system.[25]

Physiologically, the pharynx is a very easily narrowed area in its overall expansion.[26]The activity of widening pharyngeal muscles, especially the genioglossus and palatini tensor muscles that oppose the pharyngeal tendency to collapse as a protective mechanism.[27]Changes in the mechanism are associated with obstructive sleep apnea disorder syndrome.

Treatment options for OSA include weight loss,position therapy, oral appliances , positive continuousairway pressure (CPAP), and surgery. Operations offer a viable alternative to nasal CPAP in patients who areintolerant of nasal CPAP.In that case, oral appliances such asmyofunctional therapy applied to OSA[28] has the aim of strengthening the nasopharyngeal and oropharyngeal muscles which contribute to reducing airway collapse during sleep. the exact mechanism by which Oral

Myofunctional Therapy helps with Sleep Respiratory Disorders is still unclear to this day.[30]

At this stage, a systematic review is needed to prove the level of scientific evidenceOral myofunctional therapy as a future treatment for Obstructive Sleep Apnea in children. The aim of this study is to systematically review the literature for articles evaluating Oral Myofunctional Therapy exercises as a treatment for OSA in children.

METHODS

Search Strategy

A search was performed on Web of Science, Scopus, and The Cochrane Library.Studies published from 2010to 2020 thatthe treatment with isolated Oral Myofunctional Therapy in subjects with OSA were included.MeSH terms and keywordsused for the search included various combinations of the following: "myofascial reeducation," "myofunctional therapy,""obstructive sleep apnea," "orofacialmyotherapy," "oral myotherapy, "sleep," and "sleep apneasyndromes. One example of search is: ("Myofunctional Therapy' AND "SleepApnea Syndromes") OR ("sleep" AND ("myofascialreeducation" OR"myofunctional therapy" OR "rapid maxillary expansion")or each of the searches, the titles and abstracts werescreened and the full text versions of articles that met criteriawere downloaded. Full texts were reviewed and any referenced articles that were not already obtained were ordered andobtained. "Related citations" were also reviewed during thesearches, and the "cited by" function on Google Scholar wasalso used to identify any additional studies

The flowchart in Figure 2 identifies the included andexcluded articles at each stage. Sixty were assessed, including 60 articles from the electronic databases, 0 from the manual hand search. 50records screened, 30 records excluded, 22 full-text articles assessed for eligibility and 10 full text articles included

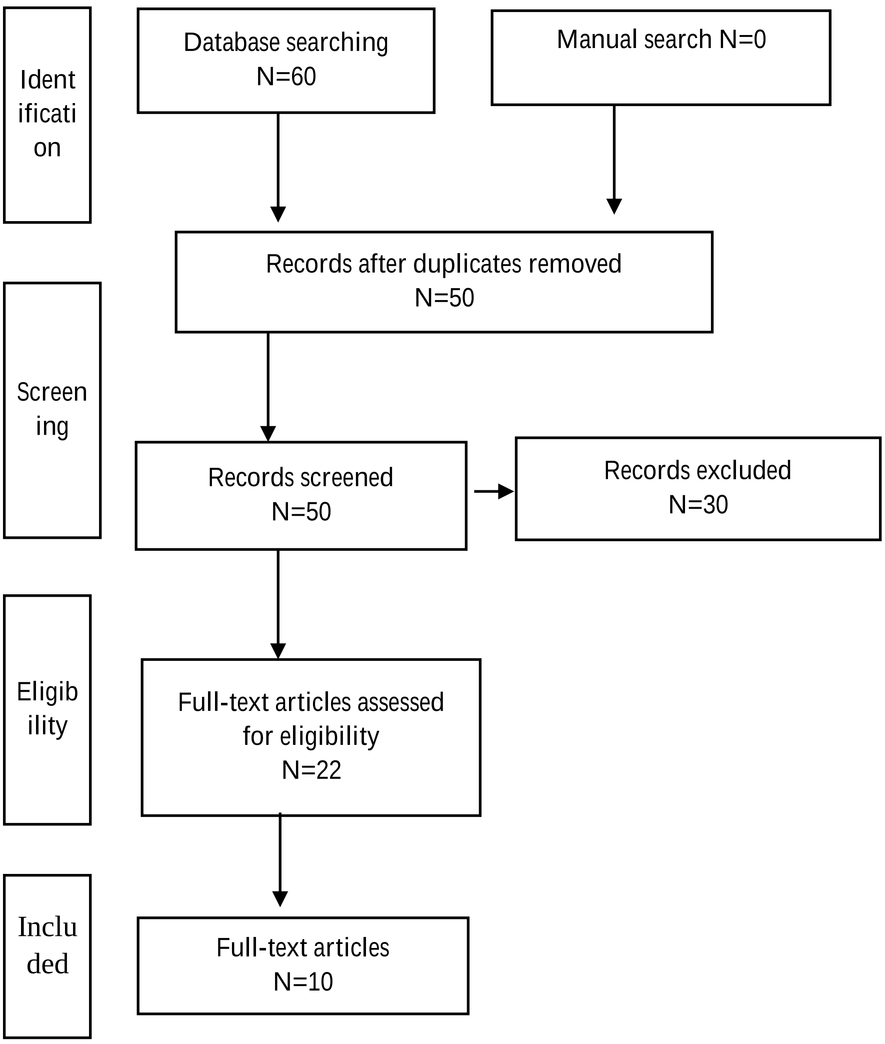

Figure 2: A flow chart describing the search methodology and numbers ofarticles included/excluded at each stage

LITERATURE REVIEW

In this systematic review there are study selection: inclusion criteria for this review were: 1) children (<18 years old) with OSA, 2) all languages, 3) all study designs and publication types were considered, 4) Article 2011-2020 yeaar, and 5) both published and unpublished data were sought out. Exclusion criteria were: 1) studies that are not about RME as treatment for OSA, and 2) adult.

Table 3: Study design and treatment protocol

Author (year)	Study Type	N	f/m (mean age)	Variables analyzed	Treatment	Conclusion
Caprioglio et al., (2014)[31]	Quasi-experimental, not randomized, no control	14	(7.1 ± 0.6)	AHI SpO2	Rapid maxillary expansion (6months)	Treatment improve SAHS (Sleep Apnea-Hypopnea Syndrome)
Rabasco et al., (2014)[32]	Quasi-experimental	40	(4-8)	AHI	Rapid maxillary expansion (6 months)	Data confirm theefficacy of Orthodontictreatment for children with slightor moderate SAHS
Villa et al., (2014)[33]	Quasi-experimental control group, not randomized	52	15/37 (5.03)	AHI SpO2	Rapid maxillary expansion (6 months)	Treatments improve SAHS.
Fastuca et al., (2015)[34]	Quasi-experimental no control group, not randomized	15	11/4 (7.5 ± 0.3)	AHI SpO2	Rapid maxillary expansion	RME improves oxygen saturation for patientswith posterior crossbite
Villa et al., (2015)[35]	Quasi-experimental no control group, not randomized	40	17/23 (6.3 ± 1.6)	AHI SpO2	Rapid maxillary expansion	It is important to start orthodontic treatment early to improve treatment efficiency
Pireleli et al., (2015)[36]	Clinical evaluation	31	(8,68 years ole)	AHI SpO2	Rapid maxillary expansion (6 months)	RME treatment for pediatric OSA
Villa et al., (2017)[37]	Case control study	54	Sleep-disordered breathing (7.1 ± 2.5), Healthy children (7.8 ± 2.2)	AHI	Myofunctional therapy (6 months)	Myofunctional therapy can be used to integrate medical and surgical treatments for OSA and helprestore the resting posture of the tongue; appropriate oral, lingual and facial muscle patterns; nasal breathing; normal lip posture; and correct swallowing patterns
Klauer (2018)[38]	Case study	1	9 years old	AHI	Myofunctional therapy (3 months)	Myofunctional therapy is a greet tool in our armamentarium for treating children and adults with maloclussion or OSA
Hsu et al. (2018)	Clinical evaluation	54	4-16 years old	AHI	Passive myofuntional therapy (3 months)	Passive myofunctional therapy is a valid alternative treatment for pediatric OSA
Shim et al., (2019)[39]	Case reports	2	7 years old	AHI SpO2	Passive myofunctional therapy (6months)	Sleep Disorder Breathing in early childhood can have adverse effects on myofunctional balance and is associated with malocclusion. Orthodontic treatment, alone does not guarantee that these issues will be resolved. Myofunctional therapy may facilitate successful orthodontic treatment and maintenance

Table 4: Cephalometric mesurements before and after treatment Myofunctional Tools in Children with Sleep Disorders: Two Case Reports[39]

Case Reports	SNA (81)	SNB(77)	ANB (3)	APDI (81)	FMA (26)	U1 to SN (105)	IMPA (95)	Interincisal angle (122)	Incisal overjet (3,3)	Incisal overbite (2,3)	U-nasolabial angle	L-Nasolabial angle	Nasolabial angle (100)
Shim et al(2019)													

39											(20)	(80)	
Before treatment (1)	79,9	73,9	6	74,7	25,5	110,2	94,4	122,5	7,2	3,4	12,1	68,8	80,9
After treatment (1)	79,7	74,3	5,4	75,3	25,9	101	96,8	128,7	3,7	2,3	19,8	71,3	91,1
Before treatment (2)	71,9	70,6	1,4	73,3	39,3	99,4	85,1	131,1	3,8	-1,4	37,6	83,2	120,7
After treatment (2)	72	71,5	0,5	77,5	37	93,7	87,2	136,2	0,5	-1,2	36,1	81,8	117,9

Based on (Table 4) Caprioglioet al.[31] reported not randomixed quasi-experimental with no control of 14 children who received myofunctional therapy using rapid maxillary expansion. The exercise group was followed up for 6 months. The exercise is repeated several times a day in 6 months. Further in table 3 with other studies. In contrast to (Table 4) the case report shows cephalometricmesurements before and after the treatment of myofunctional devices in children with respiratory disorders during sleep. The different with Klauer[38] study design case study with 1 children and theexercise using myofunctional device is only 3 monthsthatcan be a greet tool in our armamentarium for treating children and adults with maloclussion or OSA.

Case report 1 (Table 4) Onextraoral examination, lateral convex profile and lip disability were observed. The incisors of proklin (U1 to SN 110.2 °), substantial overjet (7.2 mm) and normal overbite (3.4 mm) were recorded in cephalometric analysis. Large ANB (6.0 °) and reduced APDI (74.7 °) were also recorded. FMA is 25.5 °, which means normal vertical growth. Furthermore, the tendency of the class II framework with the mandible being reexamined with respect to the maxilla, was observed. As for soft tissue analysis, the upper lip is protruding (Nasolabial angle 80.9°) and the lower lip is eluted again (L-nasolabial angle 68.8 °). In the analysis of the cast model, the molar relationship is the terminal flush plane and the canine relationship is class I. The upper and lower arches are narrow. Patients were diagnosed with class II division 1 as a consequence of Sleep Disorder Breathing,[39]

After 6 months of treatment, U1 to SN decreased from 110.2 ° to 101.1° and IMPA increased from 94.4° to 96.8° (Table 4). Overjet was reduced from 7.2 mm to 3.7 mm, and overbite from 3.4 mm to 2.3 mm. The nasolabial angle and the L-nasolabial angle increased from 80.9° to 91.1° and 68.8° to 71.3°, respectively. AHI has decreased from 1.3 to 0.6, and the average SpO2 has increased from 96.6% to 97.2%. Oral breathing is returned to nasal breathing, and moderate lip competence with decreased bruxism is noted.[39]

Case report 2 (Table 4) Onextraoral examination, convex lateral profile, and poor lip competence were observed. On intraoral examination, the tongue offered with an anterior openbite was found. The cephalometric analysis showed retrocline upper incisors (U1 to SN 99.4°) and lower incisors (IMPA 85.1°). A reduced ANB (1.4°) and APDI (73.3°) were also recorded. FMA is 39.3°, which means vertical growth patterns. The pattern of the malocclusion framework is ambiguous because ANB and APDI are conflicting. The nasolabial angle and the nasolabial angle are both reduced. In the cast model analysis, the molar relationship is the terminal flush plane and 3 deciduous canines (# 53, 73, 83) are lost prematurely. The diameter of the mandible is shifted about 2.6 mm to the right. Overbite -1.4 mm and overjet 3.8 mm. The upper and lower curved shapes are tapered and ovoid respectively. The patient was diagnosed with Angle 2 malnutrition class 2 division 2 and had symptoms of Sleep Disorder Breathing[39]

After 6 months of treatment, there was an increase in APDI from 73.3° to 77.5°, which is within the normal range (Table 4). Clinically, convex lateral facial profile is also found to decrease. The bimolar width between the dento-gingival junction center of the first primary molar has increased from 21.4 mm to 22.9 mm in the maxilla, and from 22.0 mm to 23.3 mm in the lower jaw. In contrast, the bimolar width between the dento-gingival junction center of the first permanent molar has decreased from 29.5 mm to 28.3 mm in the maxilla, and from 31.3 mm to 31.1 mm in the mandible. AHI decreased from 1.8 to 1.0 after 6 months of treatment, and the mean SpO2 increased from 96.2% to 97.3%. Respiratory Sleep Disorders are improved with Myofunctional therapy.[39]

DISCUSSION

Myofunctional therapy has the potential to be an option for the treatment of OSA.[40]This is defined as a treatment for facial and mouth muscles, which is very important for the maintenance of craniofacial integrity to achieve normal nasal breathing. Re-education of myofunctional therapy trains normal and strong sucking, good mastication using both sides of the jaw, normal swallowing, normal tongue position, and nasal breathing with lips in good contact at rest. Nasal breathing upon awakening and sleeping is a demonstration of normal respiratory function, and persistence of mouth breathing is an indicator of abnormal respiratory function.

Myofunctional therapy requires the involvement of at least one parent and child for a minimum of 10 minutes of active exercise both in the morning and at night. Maintaining this routine has proven challenging with young children and compliance has been limited, especially if there is no routine contact with myofunctional specialists who can provide support to parents and children.[41]

Recently, myofunctional therapy (MFT) has been suggestedas additional treatment of Sleep Disorder Breathing (SDB) in children.[42] ResultsStudies conducted in children with orthodontic problems have shown thatextensive, well-controlled isolated myofunctional therapy can lead to a return to normal orofacial anatomy. In adults, there is an increase in OSA and snoring as well.[43,44,45] But usagemyofunctional therapy itself when dealing with pediatric SDB havehas not been widely investigated and the long-term effects of myofunctional therapy on SDB are still unknown. Problemwhere myofunctional therapy has been reported: (1) current formMFT is difficult for children under 4 years,(2) poor compliance with daily exercise andthe absence of ongoing parental involvement withexercises for children are the main causetreatment failure. Preliminary studies are indicatedthat the use of functional oral tools is thought ofinduces some extra muscle activity during sleep(also called "passive myofunctional therapy"[PMFT]) can reduce mouth breathing and haveimpact on the position of the mandible

CONCLUSION

Therefore a systematic literature review shows that oral myofunctional therapy for future treatment of obstructive sleep apnea in children (OSA).

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