Review Article

Pediatric Obstructive Sleep Apnea: The Role of Orthodontic Management - Review Article

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Pediatric obstructive sleep apnea (OSA) is a common breathing-related sleep disorder affecting 1% to 5% of children. It often presents with less recognized signs and symptoms compared to adult OSA. Consequently, many patients with pediatric OSA remain undiagnosed. Risk factors include adenotonsillar hypertrophy, craniofacial anomalies, retrognathia, nasal obstruction, macroglossia, nasal septal deviation, and obesity. Orthodontist as a healthcare provider could have a significant role in screening, diagnostic referral, and treatment. The management approach for pediatric OSA requires multidisciplinary collaboration to obtain an optimal treatment outcome. Currently, adenotonsillectomy is recommended as first-line therapy. However, the treatment success varies considerably among patients. Children with OSA are often found to have narrow and constricted maxilla, mandibular retrognathia, and posterior rotation of mandible. Therefore, orthodontic treatment such as rapid maxillary expansion (RME), functional jaw orthopedic appliances, and protraction facemask could improve pediatric OSA with proper case selections. Additional maxillary expansion can also be performed in conjunction with adenotonsillectomy regardless of treatment sequence. The present article reviewed the currently available literature on the efficacy of various orthodontic treatments on pediatric OSA. Additional high-quality evidence is required to further substantiate the effectiveness of these orthodontic therapy.

Keywords: Pediatric OSA, Orthodontic treatment, Rapid Maxillary Expansion (RME), Functional jaw orthopedic appliances, Protraction facemask

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Obstructive sleep apnea (OSA) is a common chronic disorder that is characterized by repetitive episodes of complete or partial collapse of the upper airway during sleep, with a consequent cessation or reduction of the airflow[1]. The prevalence of OSA in children has been reported to be between 1% to 5%[2]. Pediatric OSA often presents with less recognized signs and symptoms. The percentages of undiagnosed pediatric OSA are likely to be far more than that of adults[3]. Risk factors include obesity, craniofacial anomalies, retrognathia, nasal obstruction, macroglossia, and enlarged adenotonsillar tissues[1,4].

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Etiology

The multifactorial etiology of OSA consists of complex anatomic and neuromuscular factors, as well as an underlying genetic predisposition[1,4-6]. An increase in upper airway collapsibility due to structural changes such as adeno-tonsillar hypertrophy, mandibular retrognathia, and variations in craniofacial structures leading to upper airway restriction have been shown to increase the risk of developing OSA.

Adenotonsillar hypertrophy

For pediatric OSA, the obstruction of the upper airway caused by hypertrophy of tonsils and adenoids is the principal cause. Children with OSAs have larger adenoids, tonsils, and soft palates as measured by MRI when compared to children without OSA[6]. The peak incidence of childhood OSA at three to six years of age is closely related to the ages at which the tonsils and adenoids are also at their largest[7]. However, several studies demonstrate no significant correlation between adenotonsillar size and severity of pediatric OSA[8-10]. Guilleminault et al demonstrated that OSA could recur during adolescence despite of

tonsillectomy performed in preadolescence[11].

Dentofacial abnormalities

The dentofacial abnormalities include mandibular retrognathia, midfacial hypoplasia, narrow and constricted maxilla with high-arched palate, and posteriorly rotated mandible. Chronic mouth breathing, which is seen in children with upper airway obstruction also results in similar features such as the high-arched palate, narrow maxilla, retrognathic mandible, and increased lower facial heights[12].

Neuromuscular factors

Both genetic and environmental impairment of orofacial muscle activity have been shown to impact the development of the airway. An unstable central respiratory control may lead to fluctuations in ventilatory drive that could destabilize and collapse the airway[13].

Obesity

Previously not seen to be a contributing factor to OSA in children unlike in adults, the increasing prevalence of obesity in children has become an important risk factor, with an estimated OSA prevalence of 36% in obese children[14,15]. Adipose tissue deposition surrounding the airway, reduced lung volume, and decreased central ventilatory drive are the main mechanisms for OSA due to obesity[16].

Clinical presentations

OSA should be suspected when nocturnal symptoms of habitual snoring, pausing, gasping or choking, cyanosis, witnessed apneas or mouth breathing, increased work of breathing or paradoxical breathing, restless sleep, and sleeping with a hyperextended neck are reported.

Fragmented sleep from OSA results in hypersomnolence or daytime sleepiness, the most reported symptom of adult patients with OSA. In children, the daytime symptoms are more related to attention and concentration problems, hyperactivity, behavioral changes, daytime sleepiness, fatigue or extreme tiredness, headaches in the morning, poor school performance, and mood changes such as irritability and depression. Hence OSA in children can be categorized based on symptoms into two phenotypes. Type I phenotype constitutes what frequently described as behavioral alterations such as hyperactivity and inattention without significant daytime hypersomnolence, whereas type II phenotype is more associated with diurnal symptoms more similar to those of the adults[17]. It is essential to include OSA in the differential diagnosis when encounter children with behavioral problems since there can be some symptoms that overlap among these conditions. Behavioral dysfunctions like attention deficit hyperactivity disorder (ADHD), hypersomnolence, somatization, depression, aggression, and abnormal social behaviors are the most frequently reported behavioral abnormalities associated with OSA in children[18,19].

The signs and symptoms of pediatric OSA vary considerably along their developmental stages. In pre-school age, snoring or loud breathing is the most common symptom, along with oral breathing, disturbed sleep with frequent changes of position, unusual sleep positions such as hyperextension neck, nightmares, and growth retardation. In addition, behavioral disorders such as hyperactivity and inattention are more frequently reported in pre-school age. In school-age, excessive sweating during sleep is a common manifestation, as well as sleep terror and sleep walking, chronic cough, hyporexia, learning disabilities, daytime sleepiness, emotional instability, difficulty in morning awakening, bruxism, and diurnal headaches. Children with OSA are at high risk for enuresis that often resolves when respiratory sleep disorders are adequately treated[17].

On physical examination, findings of tonsillar hypertrophy, nasal septal deviation, obesity, midface deficiency, macroglossia, or mandibular hypoplasia may strengthen the suspicion of OSA[20]. Delayed growth and impaired weight gain have been frequently reported in children with OSA. In severe cases, evidence of pulmonary hypertension manifested by a loud second pulmonary heart sound and systemic hypertension has been reported. An appearance of neurologic impairment, genetic diseases and complex medical disorders are risk factors for pediatric OSA such as uncontrolled epilepsy, neuromuscular disorders, Prader-Willi syndrome, achondroplasia, Chiari malformation, Ehlers-Danlos syndrome, mucopolysaccharidoses, and trisomy 21[21,22].

Consequences of untreated OSA in children

Untreated OSA is associated with cardiovascular complications, impaired growth including failure to thrive, metabolic systems, learning problems, and behavioral problems[23]. These cognitive and neurobehavioral dysfunctions, if left untreated, eventually affect the overall quality of life[24].

In metabolic process, pediatric OSA increase pro-inflammatory markers, especially IL-17, IL-23,

high-sensitivity C-reactive protein (HS-CRP), TNF-α, IL-1β, IL-6, and IL-10[25]. A previously published study in non-obese children with moderate to severe OSA reported that elevated plasma CRP levels declined after adenotonsillectomy[26].

Diagnosis

In-lab overnight sleep study or polysomnography (PSG) is currently considered the gold standard for diagnosing both pediatric and adult OSA. The recording channels usually comprise 1) electro-encephalography (EEG) for scoring sleep staging and arousal, 2) electro-oculography to assess eyes movement, 3) submental electromyography to assess rapid eye movement (REM) stage, 4) tibial electromyography to detect legs movement, 5) oronasal thermal airflow sensors to detect apneas and nasal pressure transducer to detect hypopneas, 6) pulse oximetry to detect arterial oxygen saturation, 7) respiratory inductance plethysmography belts placed in chest and abdomen to detect respiratory effort, 8) electrocardiography, and 9) audiovisual recording. PSG usually reports an apnea hypopnea index (AHI) that measures a frequency of apnea and hypopnea per hour of sleep. Pediatric obstructive Apnea is the peak signal excursions drop to 90% or more of pre-event baseline for the duration of two breaths or more, and the event with continued inspiratory effort throughout the entire period of decreased airflow. While pediatric obstructive hypopnea is the peak signal excursions drop of 30% or more of pre-event baseline for the duration of two breaths or more and must also cause a sleep arousal or more than a 3% decrease in oxyhemoglobin saturation[27,28].

The AHI is widely used as the basis for OSA diagnosis. According to the International Classification of Sleep Disorder-Third Edition, the severity of OSA based on the AHI scores is categorized differently for adults and children[29]. For children, AHI of less than 1 to 5 is mild, 6 to 10 is moderate, and more than 10 is severe OSA[2]. In addition to an AHI, the lowest oxygen saturation during sleep and sleep fragmentations should be taken into consideration for the clinical assessment of OSA.

The OSA cost is high as it requires an overnight hospital monitoring. Furthermore, there is limited availability of pediatric sleep specialist for scoring and analysis. When PSGs are not available, pediatric sleep questionnaires, nocturnal pulse oximetry studies, ambulatory PSG, and nocturnal video recording are used as alternative diagnostic tools for OSA in limiting resourced countries[30-32].

Treatments

OSA should be managed as a chronic disorder, with multidisciplinary collaboration[33]. For pediatric OSA, adenoidectomy and tonsillectomy (A&T) are the first line treatment since adenotonsillar hypertrophy is the most common cause[34]. The success rate for A&T varies between studies. In 50% of patients, post-operative AHI may be reduced to less than 1 but may recur in 68% of patients after 36 months[35]. In 2013, Guilleminault and Huang studied the recurrence of OSA in teenagers following childhood A&T. The result showed that A&T improved symptoms and PSG results but did not cure the upper airway obstruction and may still lead to abnormal orofacial development[36].

Positive airway pressure (PAP) therapy is indicated in selected groups of pediatric patients including those who are not good surgical candidates, those with major craniofacial deformities, and those poor surgical outcomes. Studies demonstrated significant improvement in sleepiness, snoring, PSG parameters, and oxyhemoglobin saturation while with continuous PAP (CPAP) in pediatric patients[2,37]. Like adult patients, adherence and compliance are main obstacles to effective CPAP therapy. Therefore, CPAP is not recommended as a first-line therapy for OSA when A&T is an option[37-40].

Pharmacological treatments including local nasal corticosteroids, leukotriene antagonists, and a combination of both are recommended for OSA treatment in children with mild or residual OSA, and allergies. They were found to have small clinical effects in term of reducing mucosal edema and adenotonsillar hypertrophy. Therefore, they are not suggested as a primary treatment of moderate or severe OSA[41].

Tracheostomy is usually reserved as a last resort for patients who have not responded favorably to other less invasive procedures. It is also used as more of a temporary measure to protect the airway in patients with morbid obesity and craniofacial syndromes. However, it comes with significant social and morbidity risks such as infection, hemorrhage, and psychosocial difficulties[42].

Up until a few decades ago, the treatment of patients of pediatric OSA was largely offered by physicians as outlined in this section. It was not until the mid-1980s when the subject of OSA began entering the dental and orthodontic community[43,44]. The purpose of the present article was to provide the

Thai medical community with an understanding of the current evidence surrounding various orthodontic treatments of pediatric OSA, and for orthodontists to be knowledgeable and play a role in the identification and management of patients with this condition.

Orthodontic management of pediatric OSA

Role of orthodontists

The role of orthodontists in managing pediatric OSA has become increasingly important since they are often the first to notice the signs and symptoms[1]. Orthodontists have an opportunity to screen for OSA symptoms and to detect adenotonsillar hypertrophy during an oral exam. Patients with pediatric OSA were also found to have narrow and constricted maxilla often with high-arched palate[45] (Figure 1). Katyal et al reported that patients with high risks for pediatric OSA are almost three time more likely to exhibit posterior dental crossbite when compared with low-risk patients[46]. Additionally, children with OSA have a significantly increased overjet, a reduced overbite, shorter mandible, and more obtuse mandibular plane angle[45]. These dentoalveolar and craniofacial characteristics can be recognized early during an orthodontic exam. Guilleminault and Quo suggested that these skeletal malocclusions significantly contribute to the development of pediatric OSA and could be corrected by orthodontic treatments[1]. Orthodontists are in a unique position to identify patients with high-risks, make a diagnostic referral, collaborate with sleep physicians and other medical specialists, and deliver appropriate treatment to patients with pediatric OSA[47].

Rapid maxillary expansion

Rapid maxillary expansion (RME), also known as rapid palatal expansion (RPE), is a dentofacial orthopedic treatment indicated for constricted maxillary arches in growing (Figure 2). A maxillary expander is placed on the upper dentition and palate. It is usually activated daily for four to six weeks to expand a mid-palatal suture until a desirable expansion is achieved. The opening of a mid-palatal suture can be confirmed radiographically as a radiotransparent area in the middle of hard palate[48]. A new bone is formed in the expansion space after a retention period of four to six months. Then the expander appliance can be removed. RME creates a significant increase in dentoalveolar dimensions such as palatal width, intermolar and intercanine distances[49] (Figure 3). The midpalatal suture shows the greatest width increase at the central incisor level, followed by the canine level, and the first molar level. RME was also found to produce a significant width increase in the intermaxillary, internasal, maxillonasal, frontomaxillary, and fronto-nasal sutures[50]. The palatal suture becomes fully ossified after puberty. As a result, the suture will no longer respond a conventional RME, and maxillary osteotomy is required to achieve a desired maxillary

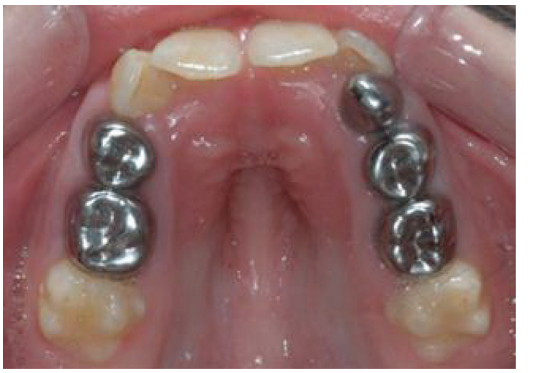

Figure 1. Narrow and constricted maxilla often with high-arched palate[45,46].

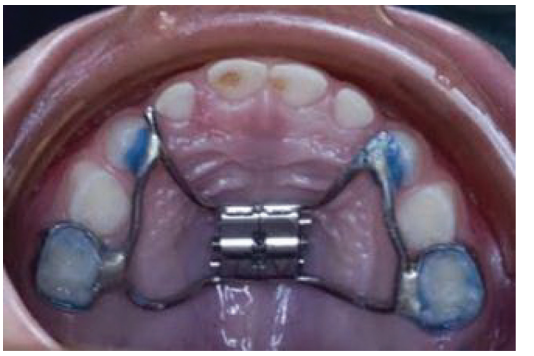

Figure 2. Rapid maxillary expansion device before activation[48].

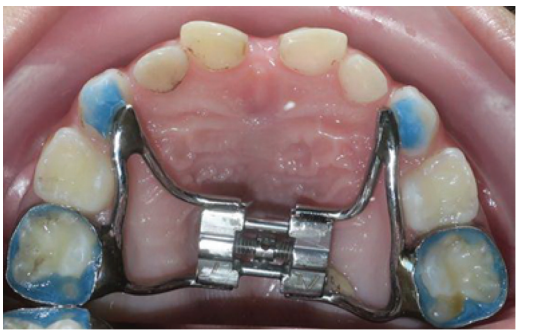

Figure 3. Rapid maxillary expansion device after activation. RME creates a significant increase in dentoalveolar dimensions such as palatal width, intermolar and intercanine distances[49].

expansion. Interestingly, Pheonixa et al demonstrated that the distance from hyoid bone to mandibular plane decreased significantly in patients treated with RME. The authors reckoned that hyoid bone position appears to normalize after a maxillary orthopedic expansion[51].

RME effects on nasomaxillary complex

Pirelli, Saponara, and Attanasio used two-dimensional radiographs to study the effects of RME. They found that the treatment widens nasal fossa and releases a deviated septum from a lateral wall of a nasal cavity, thus restoring a normal nasal airflow by creating anatomical changes[48]. Several other studies employed three-dimensional imaging analyses to investigate the effects of RME. All of them found that RME creates a significant increase in nasopharyngeal airway volume[49,52-55]. Babacan et al reported a 13.8% nasal volume increase in patients without decongestant and a 15.16% increase in patients with decongestant after RME[55]. Another study reported an 18% nasal volume increase with a 22.8% increase in minimal cross-section area of the nasal cavity following RME[49]. Two investigations employed acoustic rhinometry to study nasal airway resistance after RME therapy. They found that the mean resistance was reduced by 25.5% and remained stable in a long-term follow-up[49,54]. As these studies confirmed that RME leads to an expansion of nasal cavity, an increase of airflow, and a reduction of nasal resistance, this could explain how RME alleviate pediatric OSA.

Effects of RME on respiratory parameters

Pirelli, Saponara, and Guilleminault studied the effects of RME on children, with a mean age of 8.7 years, with confirmed OSA and maxillary constriction, without adenotonsillar hypertrophy. They reported a mean pre-treatment AHI of 12.2 events per hour was reduced to less than one event per hour at the 4-month follow-up after RME[56]. Another study investigated children with moderate to severe OSA, with a mean AHI of 12.18 events per hour, and narrow maxilla, without adenotonsillar hypertrophy or with a history of adenotonsillectomy. The authors demonstrated that the AHI was reduced to 0.5±1.2 event per hour four months after RME[48]. Villa et al conducted a prospective study to assess the RME effect in children, with a mean age of 6.6 years, with confirmed OSA of mean AHI 5.8 events per hour, and narrow, high-vaulted maxilla. The study showed a significant AHI decrease to one event per hour in patients with mild adenotonsillar hypertrophy, and to 2.3 events per hour in patients with severe adenotonsillar hypertrophy 12 months after RME[57]. Another prospective study reported an AHI decrease from of 5.8 to 1.6 events per hour in children, with a mean age of 7.5 years, and with moderate OSA, narrow maxilla, and high-arched palate 12 months after the treatment. In the same study, the authors also demonstrated a 5.3% increase in blood oxygen saturation[53]. Miano et al evaluated children with a mean age 6.4 years with mostly severe OSA and a mean pre-treatment AHI of 17.4 events per hour, narrow maxilla, and varying degree of adenotonsillar hypertrophy. The results showed that AHI decreased to 5.4 events per hour after one year of RME. Furthermore, these patients also demonstrated a nearly normal sleep architecture[58]. The results from these studies lead us to conclude that RME could be an efficacious treatment for pediatric OSA, especially those without adenotonsillar enlargement, and those with mild adenotonsillar hypertrophy or with a history of adenotonsillectomy. However, more well-designed studies are required to substantiate the efficacy of RME as an alternative treatment for pediatric OSA.

Long-term follow-up of RME

Villa et al performed RME in pediatric OSA patients of a mean age of 6.6 years, with narrow maxilla and varying degree of adenotonsillar hypertrophy. As a group, the mean AHI decreased from 6.3 to 2.4 events per hour 12 months after RME, and to 2.3 events per hour at the 36-month follow-up[59]. Pirelli, Saponara, and Guilleminault followed up patients annually for a mean period of over 12 years after the completion of RME. Most of these patients were re-assessed with PSG in their late teens or early 20s. The results demonstrated that PSG findings of these patients remain normal in a long-term follow-up after the completion of RME treatment. These studies showed that the treatment result following RME therapy could remain stable in a long-term follow-up[60]. More investigations will be needed to confirm this.

Combined RME and adenotonsillectomy

Guilleminault et al[61] performed a pilot randomized controlled trial to investigate the effects of RME versus adenotonsillectomy (A&T). They recruited thirty-one children of a mean age of 6.5 years, with OSA confirmed by PSG. The samples also presented with narrow maxilla and moderate adenotonsillar hypertophy. They were randomly assigned to two treatment approaches. The first

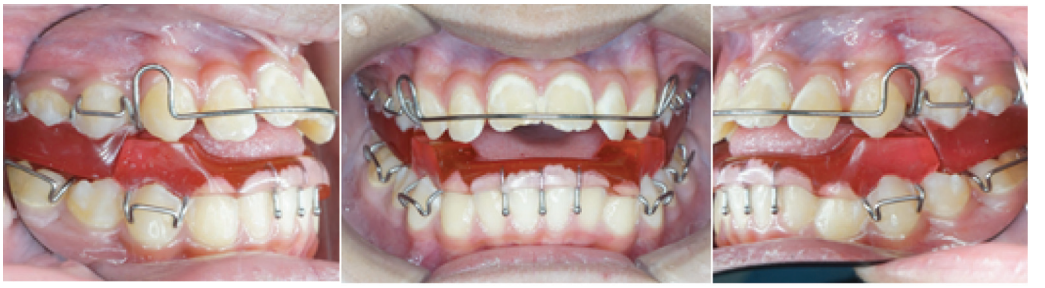

Figure 4. Twin block appliance[63,64].

group received A&T followed by RME, whereas the second group underwent RME first, then A&T. The improvement was not significantly different between the two approaches following the first treatment. All subjects went on to complete both A&T and RME except one patient who had successful treatment with the orthodontic therapy alone. Those patients who underwent both treatments displayed an overall significant improvement with their PSG findings compared to the baseline and compared to the first treatment[61]. However, there is no significant difference between the two approaches. Pirelli et al[62] conducted a similarly designed study. They reported that significantly more children with OSA were successfully treated with RME alone compared to A&T alone, but the final outcomes after completion of both treatments show no significant difference between the treatment groups. In both studies, most patients were not treated adequately either by adenotonsillectomy alone or RME alone. These results may indicate that a combination treatment may be needed for some patients, especially those with narrow maxilla and moderate or severe adenotonsillar hypertrophy[62].

These studies demonstrated that dentofacial skeletal hypoplasia could play a significant role in the development of pediatric OSA in children in addition to adenotonsillar hypertrophy[40]. RME is a promising alternative for pediatric OSAS that create a significant anatomical and functional improvement. A recent systematic review and meta-analysis concluded that RME could provide a consistent reduction of AHI and increase blood oxygen saturation in children with OSA both in the short term of less than 3-year and in the long-term of more than 3year[20]. In addition, high-risk patients for OSA displayed a significantly improved quality of life following RME therapy[46]. Given the less invasive nature of RME and the reported success rates, RME could be considered as the first-line treatment especially for those with concurrent pediatric OSA and transverse maxillary constriction. Obviously, more high-quality randomized clinical controlled trials are needed to validate the efficacy of RME as a treatment alternative for pediatric RME as well as to substantiate the present proposal.

Functional appliances

Mandibular advancement devices (MAD) such as Herbst and Twinblock (Figure 4), also known as functional appliances (FA) in orthodontics, are traditionally employed to correct Angle's Class II malocclusion with mandibular hypoplasia[63]. They were thought to be capable of producing additional growth for a hypoplastic mandible in growing children. However, outcomes from most recent randomized controlled trials conclude that these appliances do not make the mandible grow more than what they are genetically capable[63,64]. Many researchers and clinicians began to use FA to treat pediatric OSA since mandibular hypoplasia is one of the risk factors for pediatric OSA, coupled with staggering evidence of MAD as an effective therapy for adult OSA[12,65]. Cozza et al[66] studied the effects of an FA called modified Monobloc in children with a mean age of 5.91 years and with moderate OSA treatment at a median pre-treatment AHI 7.8 events per hour, and mandibular retrognathia. They reported an AHI decrease to 3.6 events per hour after treatment and concluded that FA may be an effective therapy for mild to moderate pediatric OSA[66]. Villa et al[67] conducted an RCT to investigate the efficacy an FA therapy for pediatric OSA treatment in a group of patients with a mean age of 7.1 years. They reported that almost three quarters of these patients tolerated the appliance well. The treatment was able to reduce the AHI from 7.1 to 2.6 events per hour and improve clinical symptoms. They concluded that FA therapy is an efficacious and well-tolerated treatment for pediatric OSA[67]. Twin blocks, another type of FA, was utilized in children with a mean age of 9.7 years

and with moderate to severe OSA and mandibular retrognathia without obesity and adenotonsillar enlargement. The authors reported a significant reduction of mean AHI from 14.08 to 3.39 events per hour. The oxygen saturation nadir also significantly improved from 77.78% to 93.63%[68]. Schütz et al employed Herbst appliance, a fixed FA, in conjunction with RME in a group of patients with a mean age of 12.6 years. They reported an improvement in sleep architecture and a significant reduction of respiratory effort-related arousals[69]. Finally, a systematic review on the efficacy of FA as an alternative treatment of pediatric OSA concluded that FA could result in short-term improvements of AHI. According to those studies, FA appears to improve AHI in patients with pediatric OSA and mandibular retrognathia. However, the current evidence is limited, and more longer-term investigations with a more rigorous design are required to validate the effects of FA on improving pediatric OSA[70].

Maxillary protraction therapy

Midfacial retrusion is one of the risks factors for OSA[12]. Orthodontists have been using maxillary protraction therapy alone or in combination with RME to orthopedically advance the maxilla in growing patients for decades (Figure 5). Recently, researchers began to investigate the effects of maxillary protraction on the nasopharyngeal airway and respiratory parameters during sleep. Sayinsu, Isik, and Arun investigated the effects of maxillary protraction facemask used in conjunction with RME on the sagittal dimension of the airway. The results of the study revealed that the maxilla moved anteriorly while the nasopharyngeal airway dimension showed a significant increase[71]. A recent systematic review and meta-analysis on the efficacy of maxillary protraction therapy to improve pharyngeal airway dimensions in patients with maxillary hypoplasia was performed. The study concluded that the treatment increased post-palatal and nasopharyngeal airway dimensions[72]. It also suggested that maxillary protraction therapy could reduce pediatric OSA risks in children with maxillary hypoplasia. Peanchitlertkajorn was the first to report a combined orthopedic effect of maxillary protraction and expansion on AHI reduction. The author demonstrated that a patient with severe pediatric OSA exhibited a significantly increased nasopharyngeal space, a dramatic reduction of AHI from 51 to 4 events per hour, and improvement of OSA symptoms following the treatment[73]. A recent pilot study investigated the effects of bone anchored maxillary protraction. They reported that majority of the OSA children showed an improvement in respiratory and airway parameters, as well as symptoms after the therapy. The authors also suggested that bone anchored maxillary protraction may be considered as a treatment option to treat midface retrusion and sleep apnea[74]. However, there is lack of high-quality evidence to conclude that maxillary protraction can be used as an effective treatment for pediatric OSA. More rigorously designed studies are needed to substantiate the efficacy of the therapy.

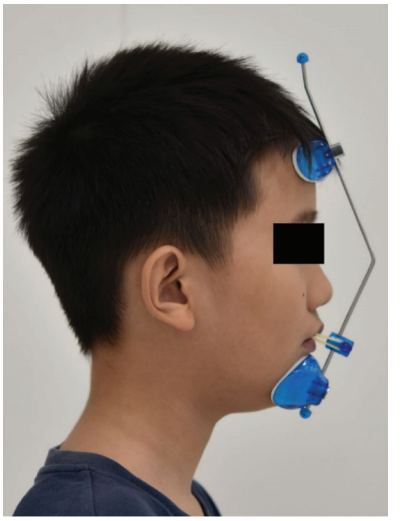

Figure 5. Protraction facemask used in conjunction with RME to orthopedically advance the maxilla[71].

Myofunctional therapy

Myofunctional therapy (MFT) is one of the recommended treatments for pediatric OSA. The therapy aims to improve the tongue muscle activities and other oropharyngeal musculature. MFT consists of a set of repetitive isotonic and isometric exercises for the oropharyngeal musculature, designed to increase upper respiratory tract patency. Villa et al reported that MFT normalized oropharyngeal muscle tone, reduced oral breathing, restored normal tongue resting position, and increase blood oxygen saturation level[75]. MFT also further reduced AHI in patients with residual OSA following adenotonsillectomy and could be considered as a supplemental treatment for pediatric OSA[76]. A systematic review and meta-analysis by Camacho et al concluded that MFT could lessen the AHI by 62%[77]. However, Huang et al reported that the compliance level for traditional MFT is very low in children[78]. In addition, economic and social conditions could potentially impact

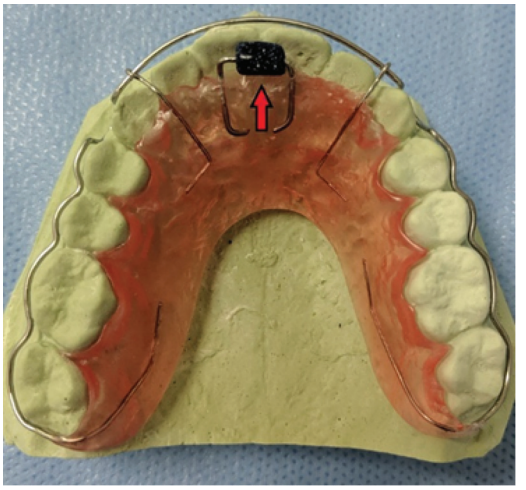

Figure 6. A compliance-free oral MFT appliance designed to stimulate tongue muscle activities to effectively treat pediatric OSA[78].

the performance of daily orofacial exercises. To circumvent patient compliance, sleep clinicians started using a compliance-free oral MFT appliance designed to stimulate tongue muscle activities to effectively treat pediatric OSA (Figure 6). The present article will focus on the efficacy of compliance-free MFT with an oral appliance as it focuses on orthodontic roles. To date, only a few studies investigated the efficacy of such therapy in pediatric OSA. Chang et al[79] designed an oral MFT appliance with a bead close to the tip of tongue to stimulates tongue activity. Patients with pediatric OSA used the appliance during sleep for six months. They concluded that the passive MFT with this specially designed device worn during sleep may improve OSA[79].

A recent study compared patients with pediatric OSA wearing a similarly designed oral MFT device with a tongue bead nightly in the treatment group with those without the device in the control group. The authors reported a significant improvement of the AHI, oxygen desaturation, airway morphology, and quality of life compared with the control group at a 12-month follow-up. Additional high-quality evidence is required to substantiate the efficacy of an oral MFT device as an alternative therapy for pediatric OSA[80].

Conclusion

Pediatric OSA is a common sleep disorder affecting 1% to 5% of children. Orthodontist as a healthcare provider could have a significant role in screening, diagnostic referral, and treatment. The management approach for pediatric OSA requires multidisciplinary collaboration to obtain a maximal effectiveness. It is the presenters hope that medical professionals will be aware of these orthodontic treatment approaches for pediatric OSA. Similarly, orthodontists must seek additional education in pediatric OSA so that they can communicate and work together with physicians in selecting an appropriate treatment based on the current evidence.

What is already known on this topic?

For pediatric OSA, adenoidectomy and tonsillectomy (A&T) are the first line treatment. However, the success rate for A&T varies considerably between studies. PAP therapy is indicated in selected groups of pediatric patients. However, the compliance and adherence are often poor among children.

What this study adds?

The management approach for pediatric OSA requires multidisciplinary collaboration. Orthodontist as a healthcare provider could have a significant role in screening, diagnostic referral, and especially treatment.

Conflicts of interest

The authors declare no conflict of interest.

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