The Asian Paediatric Pulmonology Society (APPS) Position Statement on Childhood Obstructive Sleep Apnea Syndrome

Daniel Kwok-Keung Ng1, Yu-Shu Huang1, Oon-Hoe Teoh1, Arrowan Preutthipan4, Zhi-Fei Xu3, Takeshi Sugiyama4, Kin-Sun Wong2, Ka-Li Kwok4, Brigitte Kim-Yook Fung1, Rachel Shui-Ping Lee1, Jonathan Pak-Heng Ng1, Shuk-Yu Leung1, Da-Tian Che1, Albert Martin Li10, Tat-Kong Wong11, Indu Khosla12, Anna M Nathan13, Mary Therese M Leopando13, Hussein Al Kindy16

1Department of Paediatrics, Kwong Wah Hospital, Hong Kong, 2Department of Child Psychiatry and Sleep Center, Chang Gung Memorial Hospital and University, Taoyuan, Taiwan, 3Respiratory Medicine Service, Department of Paediatrics, KK Women’s & Children’s Hospital, Singapore, 4Pediatric Pulmonary Division, Department of Pediatrics, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand, 5Department of Respiratory Medicine, Beijing Children’s Hospital, Capital Medical University, Beijing, China, 6Department of Pediatrics, Faculty of Medicine, University of Yamanashi, Yamanashi, Japan, 7Department of Pediatrics, Chang Gung Memorial Hospital, Chang Gung University, Taoyuan, Taiwan, 8Physiotherapy Department, Kwong Wah Hospital, Hong Kong, 9Department of Pulmonary Medicine, Children’s Hospital of Shanghai, Jiaotong University School of Medicine, Shanghai, China, 10Department of Paediatrics, The Chinese University of Hong Kong, Hong Kong, 11Department of Paediatrics and Adolescent Medicine, University of Hong Kong, 12Department of Pediatrics, Cloudnine Hospital, Bangalore, India, 13Department of Paediatrics, University Malaya, Kuala Lumpur, Malaysia, 14Department of Pediatrics, Philippine Children’s Medical Center, Manila, Philippines, 15Child Health Department, Sultan Qaboos University Hospital, Muscat, Oman

Abstract

With recognition of the importance of obstructive sleep apnea syndrome (OSAS) in children, practice guidelines have been developed for the management of OSAS in the USA and Europe. A panel of experts in pediatric OSAS in Asia were appointed by the Asian Paediatric Pulmonology Society (APPS) to prepare a position statement for management of childhood OSAS in Asia. The purpose of this statement is to provide a reference standard in the diagnosis and management of childhood OSAS for doctors working in Asia. The expert panel determined the scope of this statement. Focused literature search related to the key topics was conducted by panel members. The final content of this statement was agreed on by all panel members and approved by the council of APPS. The current statement covered diagnostic approach, diagnostic criteria, management algorithm, drug-induced sleep endoscopy, medical treatment including medications and positive pressure ventilation, surgical treatment including adenotonsillectomy, orthodontic treatment, and orofacial myofunctional therapy (OMT). Diagnostic criteria of childhood OSAS from 1 year to 18 years were presented that include both clinical (criteria A) and polysomnography findings (criteria B) in the diagnosis of childhood OSAS. The use of nocturnal pulse oximetry as a screening tool was suggested using the McGill oximetry score. Management of OSAS with medical treatment, tonsillectomy and adenoidectomy (TandA), positive airway pressure, orthodontic devices, nasal valves, and OMT were reviewed. Management of persistent OSAS after TandA was addressed, and the importance of weight control was emphasized. The position statement provides a guideline to the management of childhood OSAS in Asia.

Keywords: Child, polysomnography, sleep apnea syndrome, snoring

Introduction

Obstructive sleep apnea syndrome (OSAS) was reported to affect 1%–6% of prepubertal children.1,2 While standard management guideline has been developed for use in the developed countries in the USA and Europe, management of childhood OSAS in Asia has not been standardized.1,3,4 The aim of this position statement is to provide guidance to the management of childhood OSAS in Asian children for general pediatricians and general practitioners. To this aim, a group of experts in pediatric OSAS gathered in 2015 during the 1st Annual Scientific Meeting of the Asian Paediatric Pulmonology Society (APPS) held in Hong Kong in October 2015. A panel was formed and was given the task to prepare the position statement based on the current literature, especially that from Asia and the consensus among the group. The group presented the drafted statement in the International Paediatric Sleep Association in Taiwan in March. The position statement provides a guideline to the management of childhood OSAS in Asia.

Address for correspondence: Dr. Daniel Kwok-Keung Ng, Department of Paediatrics, Kwong Wah Hospital, Hong Kong, China. E-mail: dkkng@ha.org.hk

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2016 and comments were received and the group developed the second draft which was presented in the 2nd Annual Scientific Meeting of APPS in Singapore in November 2016. Further comments were received and revision was done. The final draft was presented to the guideline committee of APPS which recommended the statement to be presented to the executive committee of APPS and approval was granted for the statement to be released as the official position statement of APPS in March 2017.

**Definition of Obstructive Sleep Apnea Syndrome**

The diagnostic criteria of childhood OSAS are defined in Table 1. The current definition does not cover children younger than 1 year old as infants, especially those younger than 3 months, have different types of breathing disorders during sleep.[15]

**Risk Factors for Childhood Obstructive Sleep Apnea Syndrome**

Adenotonsillar hypertrophy is the most recognized risk factor of OSAS in children.[6,7] Allergic rhinitis and obesity are other common risk factors. [8-12] Other risk factors include well-known structural abnormalities of the airway, such as micrognathia and midfacial hypoplasia, Down syndrome, Prader–Willi syndrome, achondroplasia, and less well-known and subtle defects such as congenital teeth agenesis and septum deviation, short lingual frenulum, and chronic mouth breathing. [13-18] Neuromuscular disorders such as muscular dystrophies, cerebral palsy, and Chiari malformation are at high risk for OSAS. Other factors include gastroesophageal reflux and premature birth.[19-21] Children with a family history of OSAS are at an increased risk for OSAS. Environmental tobacco smoke exposure was also associated with OSAS.[22,23]

**Complications of Childhood Obstructive Sleep Apnea Syndrome**

Childhood OSAS is associated with neurological and cardiovascular morbidities.[24-29] These neurological morbidities include attention deficit/hyperactivity disorder, hypersomnolence, parasomnia (confusional arousals, sleep terrors, sleep walking, nightmares, and bruxism), depression, aggression, somatization, abnormal social behaviors, and nocturnal enuresis.[30-39] Cardiovascular morbidities include elevated systolic and diastolic blood pressure, dysfunction of autonomic regulation, reduced cerebral blood flow, left ventricular remodeling, and endothelial dysfunction.[25,29,40-46] Childhood OSAS is also associated with growth impairment.[47,48]

**Diagnostic Approach**

Children of all ages should be screened by their family physicians or pediatricians for the presence of snoring, especially habitual snoring, i.e. 3 or more nights per week and symptoms suggestive of OSAS during routine health checkup [Tables 2 and 3]. If positive, further focused evaluation should be performed.[11]

If there is reported habitual snoring with signs and/or symptoms suggestive of OSAS, further evaluation and management is advised. The approach may vary, depending on the resources available. An algorithm for the evaluation of children with suspected OSAS is suggested in Figure 1.

Sleep polysomnography (PSG), wherever available, is considered the gold standard for diagnosis of OSAS. Attended PSG in the sleep laboratory is preferred, especially for children younger than 4 years old. Several studies demonstrated the validity of unattended study in children but these unattended studies should involve monitoring of electroencephalogram or a way to monitor autonomic nervous system disruption, for example, electrocardiogram + SpO\textsubscript{2} plethysmography.[42,49,50] Nap studies should not be used to substitute these overnight studies.

When PSG, attended or otherwise, is not available, analysis of nocturnal pulse oximetry would provide the second best objective assessment of the child’s condition. This monitoring underscores abnormal breathing during sleep as it misses the hypopnea with only arousal. Nocturnal pulse oximetry is a useful diagnostic test only when the OSAS is associated with significant oxygen desaturation. A positive diagnostic test is made when there are 3 or more desaturation clusters (defined as 5 or more desaturations to <90% occurring in a 10–30 min period) [Table 4].[47-49] The positive predictive value and negative predictive value (NPV) of the test were 96.8% and
58.11%, respectively. The major limitation of nocturnal pulse oximetry monitoring is the low NPV when OSAS could not be ruled out.

**Drug-induced Sleep Endoscopy**

Endoscopy has been used to evaluate the upper airway for a long time. Good sedation is essential and medications such as midazolam, fentanyl, or propofol are commonly used. As OSAS children are prone to have obstructive apnea/hypopnea with sedation, it is important to have a competent medical practitioner to provide sedation and intervene whenever necessary. Structured reporting format for the findings of endoscopy is important. There are often multilevel obstructions found in patients with sleep-disordered breathing (SDB).

Evaluation of four-site “VOTE” was suggested. However, this missed out the adenoids in children. Hence, evaluation of six sites was suggested.[63,64] At the retrolingual level, the degree of hypertrophy of lingual tonsils and features of reflux laryngitis which were commonly associated with obstructive sleep apnea (OSA) should also be noted.[65] Having knowledge of number of sites of obstruction will help to plan management.

**Medical Treatment of Childhood Obstructive Sleep Apnea Syndrome**

**Intranasal corticosteroids**

The use of intranasal corticosteroids was shown in a case series by Alexopoulos et al. that their use could improve
For such high-risk patients, TandA (Tonsillectomy and Adenoidectomy) is the first-line treatment for children with OSAS with adenotonsillar hypertrophy. The Childhood Adenotonsillectomy Trial (CHAT), a randomized trial of early adenotonsillectomy (eAT) compared to watchful waiting with supportive care (WWSC) for mild-to-moderate childhood OSAS, i.e., AHI ≤5, showed normalization of PSG findings in 79% versus 46% of the respective groups on assessment after 7 months. There were also significantly greater reported reduction in symptoms and improvement in behavior and quality of life in the eAT group than the WWSC group. The significance of the normalization rate of 46% in WWSC group, who nevertheless had worse behavioral performance, warrants further study. Postoperative complications were reported to be higher in those aged below 3 years, presence of cardiac complications, congenital craniofacial anomalies, neuromuscular disorders, and severe obesity. For such high-risk patients, TandA should be performed in facilities with pediatric intensive care.

A meta-analysis of the above studies was conducted by Liu et al. in a retrospective study showed, involving 836 mild OSA children aged between 2- and 14-years, that the combined use of intranasal corticosteroids and montelukast brought about a significant improvement in AHI.

Combined intranasal corticosteroids and montelukast

Two nonrandomized studies were identified. Kheirandish et al. in an open-label control trial (involving 36 children of more than 6 years old) demonstrated that combined use of oral montelukast (4 mg for children <6 years old or 5 mg for children ≥6 years old) and intranasal budesonide (32 mcg/nostril per day) for 12 weeks in postadenotonsillectomy children with residual mild OSA could reduce AHI significantly in treatment group (mean AHI dropped from 3.9 to 0.3) when compared to control group (mean AHI increased from 3.6 to 4.7).

Kheirandish-Gozal et al. in a retrospective study showed, involving 836 mild OSA children aged between 2- and 14-years, that the combined use of intranasal corticosteroids and montelukast brought about a significant improvement in AHI.

A meta-analysis of the above studies was conducted by the authors (DKN, JPN, and SYL) and it showed a reduction of AHI by 2.7 with the use of montelukast [Figure 3].

PSG findings and OSA symptoms in children with mild SDB.

Later, randomized placebo-controlled trials involving the use of different intranasal corticosteroids, mometasone furoate, budesonide, and fluticasone propionate aqueous spray were shown to decrease apnea-hypopnea index (AHI) [Table 5].

A meta-analysis of the above studies conducted by Liu et al. in 2016 showed a reduction of AHI by 1.1 with the use of intranasal corticosteroids in children with OSA.

**Leukotriene receptor antagonist**

Montelukast given for 16 weeks at a dosage of 4 mg/day for <6 years old or 5 mg/day for ≥6 years old was shown by Goldbart et al. in an open-label case–control study involving 46 children aged between 2- and 10-years to be effective in reducing AHI significantly in treatment group, pretreatment 3.0/h to posttreatment 2.0/h, when compared to control group, pretreatment 3.2/h to posttreatment 4.1/h.

Subsequently, Goldbart et al. conducted a double-blind, randomized, placebo-controlled trial in children aged between 2- and 10-years that administration of montelukast improved obstructive apnea index (OAI) and AHI significantly.

Kheirandish-Gozal et al. published a double-blind, randomized, placebo-controlled trial on the effect of montelukast on OSA children. The study involved 64 OSA children aged between 2- and 10-years and it showed that AHI of treated children decreased from 9.2 to 4.2 (P < 0.0001) while AHI did not change in those receiving placebo.

A meta-analysis of the above studies was conducted by the authors (DKN, JPN, and SYL) and it showed a reduction of AHI by 2.7 with the concurrent use of intranasal corticosteroids and montelukast [Figure 3].

### Table 3: Symptoms of obstructive sleep apnea syndrome

<table>
<thead>
<tr>
<th>Symptom</th>
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<tr>
<td>Underweight or overweight</td>
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<tr>
<td>Tonsillar hypertrophy</td>
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<tr>
<td>Adenoidal facies</td>
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<tr>
<td>Micrognathia/retrusgnathia</td>
</tr>
<tr>
<td>High-arched palate</td>
</tr>
<tr>
<td>High Mallampati score</td>
</tr>
<tr>
<td>Cross or open bite</td>
</tr>
<tr>
<td>Increased overjet</td>
</tr>
<tr>
<td>Short lingual frenulum</td>
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<tr>
<td>Hypertension</td>
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</table>

### Table 3: Sign of obstructive sleep apnea syndrome

<table>
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<tr>
<th>Sign</th>
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<tr>
<td>Nasal cavity</td>
</tr>
<tr>
<td>2. Adenoid</td>
</tr>
<tr>
<td>3. Velum</td>
</tr>
<tr>
<td>4. Tonsils and lateral pharyngeal wall</td>
</tr>
<tr>
<td>5. Tongue</td>
</tr>
<tr>
<td>6. Supraglottis</td>
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</tbody>
</table>

**Figure 2:** Six important sites recommended for evaluation of obstructive sleep apnea with drug-induced sleep endoscopy.

**Figure 3:** Six important sites recommended for evaluation of obstructive sleep apnea with drug-induced sleep endoscopy.
service. Furthermore, a delay in performing TandA should be considered for patients with recent respiratory infections. Reevaluation with PSG several months after TandA is recommended to evaluate for residual OSAS. There were no studies evaluating the timing of postoperative PSG evaluation. The recommendation of a few months is to allow healing and resolution of inflammation and swelling of the operative site before reassessment. If PSG is not available, other options outlined in the “management algorithm of OSAS” may be considered.

The prevalence of residual OSAS after TandA ranged from 34% to 87% in the literature, depending on the characteristics of the study population and AHI definition used for residual OSAS. A meta-analysis of the effect of TandA on AHI was undertaken by the authors (DKN, JPN, and SYL). Databases including PubMed, MEDLINE, EMBASE, and Cochrane Review from 1998 to 2015 were searched. The keywords used included tonsillectomy, adenoidecctomy, OSA, sleep apnea, sleep apnea syndrome, and children. Success as defined by postoperative AHI <5 for all children and obese children was 80% and 55%, respectively [Figures 5 and 6], and it decreased to 55% and 30%, respectively, if success was defined as AHI <1–2 [Figures 7 and 8].

The risk factors for residual OSAS after TandA are severe OSA at baseline, asthma, obesity, or weight gain after TandA, trisomy 21, cerebral palsy, craniofacial abnormalities, upper/lower airway abnormalities, for example, laryngomalacia.

Growth data from the CHAT showed that TandA for OSAS in children resulted in significantly greater than expected weight increase. Significant improvements in AHI were observed in children with OSAS who underwent TandA, with significant reductions in AHI in both the treatment and placebo groups. The use of intranasal corticosteroids in the treatment of obstructive sleep apnea syndrome was also evaluated. The table below shows the study results for three different intranasal corticosteroid studies:

Table 5: Three different intranasal corticosteroids studies in the treatment of obstructive sleep apnea syndrome

<table>
<thead>
<tr>
<th>Drug</th>
<th>Sample size</th>
<th>Age (year)</th>
<th>Regimen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mometasone furoate</td>
<td>62</td>
<td>6-18</td>
<td>100 µg/nostril daily for 4 months</td>
</tr>
<tr>
<td>Budesonide</td>
<td>62</td>
<td>2-12</td>
<td>32 µg/nostril daily for 6 weeks</td>
</tr>
<tr>
<td>Fluticasone propionate</td>
<td>25</td>
<td>1-10</td>
<td>50 µg/nostril twice per day for 1 week, followed by 50 µg/nostril daily for 5 week</td>
</tr>
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</table>

Figure 3: Forest plot for the effects of montelukast on apnea–hypopnea index.

Figure 4: Forest plot for the effects of montelukast combined with intranasal steroids on apnea–hypopnea index.
gain from baseline, even in initially overweight children.\textsuperscript{[121]} This puts overweight children at greater risk of residual or recurrent OSAS after TandA.\textsuperscript{[120]}

The management of residual OSAS after TandA is dependent on the severity of the residual OSAS. Further diagnostic tests (e.g., drug-induced sleep endoscopy [DISE], cine magnetic resonance imaging) to evaluate the level of obstruction may be useful.\textsuperscript{[63,117,119,121]}

Huang \textit{et al.} demonstrated that 53\% of children had an AHI >1 at 6-month follow-up after TandA, it increased to 68\% at the end of the 36-month follow-up. Risk factors for recurrence of OSAS such as severe OSAS, obesity, and a large increase in body mass index after TandA, allergic rhinitis, enuresis, and older age were identified.\textsuperscript{[115]} Biggs \textit{et al.} performed a 4-year follow-up study for school-aged children (12–16 years old). Improvement in SDB was associated with improvements in some aspects of neurocognition but not behavior among the children. Therefore, it was suggested that a longer period of follow-up was required to observe the neurocognitive changes.\textsuperscript{[122]} The treatment options for persistent or recurrent OSAS after TandA are listed in Table 6.

**Orthodontic Treatment**

Orthodontic treatment (e.g., rapid maxillary expansion [RME], mandibular advancement devices [MAD]) may be an effective treatment option for childhood OSAS in a selected group of patients. There are, however, limited studies on orthodontic treatment for pediatric OSA, with the majority of studies being nonrandomized clinical trials.

RME is an orthodontic treatment which increases the transverse diameter of the hard palate by reopening the mid-palatal suture with an expandable dental appliance inserted into the
mouth close to the hard palate. It also has a secondary impact on placement of the mandible. It may be an option in the management of OSA in children with maxillary contraction, with long-term treatment effect shown in follow-up studies.\textsuperscript{113,124-128} A meta-analysis of RME was undertaken by Huynh \textit{et al.} who reported that the AHI decreased by 6.2 after using RME from four studies.\textsuperscript{129} MADs increase the upper airway size by positioning the mandible and tongue forward.\textsuperscript{130} In the same review by Huynh \textit{et al.}, a meta-analysis of MADs on two studies was undertaken.\textsuperscript{129,131,132} With MAD, the AHI decreased by 5.1.

The authors (DKN, JPN, and SYL) updated the meta-analysis by searching databases including PubMed, MEDLINE, EMBASE, and Cochrane Review from 2001 to 2015. The keywords used included sleep apnea, OSA, sleep apnea syndrome, MAD, and children. RevMan (version 5.2, The Cochrane Collaborations, London, UK) was used for the meta-analysis. AHI was found to be decreased by 6.5 with MAD treatment [Figure 9] from three studies.\textsuperscript{131-133}

**Figure 7:** Forest plot for success in achieving an apnea–hypopnea index <1–2 postoperatively in normal children (those have not been classified by body mass index). There was significant heterogeneity among these studies ($I^2 = 85.77$). Data were analyzed with random-effects model estimate.

**Figure 8:** Forest plot for success in achieving an apnea–hypopnea index <1–2 postoperatively in obese children. There was significant heterogeneity among these studies ($I^2 = 8.11$). Data were analyzed with random-effects model estimate. Obese was defined as Z-score from 1.2 to $\geq 2.33$ or body mass index $\geq 95^{th}$ percentile.

**Nasal Expiratory Positive Airway Pressure Valve**

This device comprises two small adhesive disposable valves applied to both nares. The valves have negligible resistance during inspiration, but generate resistance during expiration, creating a positive end-expiratory pressure from 4 to 17 cmH$_2$O.\textsuperscript{134} Initial studies showed reduction in AHI and symptoms in adults with OSA, but subsequent studies did
not show benefit in adults with moderate-to-severe OSA.\textsuperscript{135,136} A recent randomized, double-blind, placebo-controlled, crossover pilot study of nasal expiratory positive airway pressure (NEPAP) device on 14 CPAP candidates aged 8–16 years showed significant improvement in OAI with NEPAP in some patients but deterioration in a few patients, suggesting that it must only be prescribed under PSG monitoring.\textsuperscript{137}

**Positive Airway Pressure**

The basic mechanism of positive airway pressure (PAP) is to overcome dynamic upper airway obstruction by stenting the airway open by pneumatric pressure. PAP therapy has been found to be effective in improving polysomnographic parameters in pediatric patients with OSAS.\textsuperscript{138-143} In addition, there were also improvements in subjective parental assessment of sleepiness, snoring, and difficulty in breathing during sleep.\textsuperscript{138} Significant improvement in neurobehavioral function in children after 3 months of PAP therapy was demonstrated, even in developmentally delayed children.\textsuperscript{142}

PAP therapy should be considered in children who are not surgical candidates or have contraindications for TandA and those who continue to have moderate/severe OSAS after TandA.\textsuperscript{143-145} PAP may also be considered for children with severe preoperative OSAS, co-existing morbidities such as cor pulmonale, morbid obesity, neuromuscular disorders, and craniofacial abnormalities.\textsuperscript{87,96}

There are two modes of PAP – continuous positive airway pressure (CPAP) and bi-level positive airway pressure (BPAP). There is no difference in adherence between CPAP and BPAP.\textsuperscript{146} The optimal setting should ideally be adjusted under PSG.\textsuperscript{147} The maximum CPAP is 15 cmH\textsubscript{2}O for <12-year-old children and 20 cmH\textsubscript{2}O for ≥12-year-old children. CPAP should be switched to BPAP if the patient demonstrates persistence of OSA despite maximum CPAP. For BPAP, the inspiratory positive airway pressure should be started at 4 cm above the expiratory positive airway pressure (EPAP), and the EPAP pressure set at the level eliminates OSA. Long-term follow-up is needed since the required PAP setting may change over time for growing children with change in airway size and structure, as well as body weight.

If PSG titration is not available, the use of auto-titrating PAP devices for titrating pressures can be considered in patients down to 8 months of age without significant comorbidities although the body weight for auto-titrating PAP is usually above 30 kg.\textsuperscript{148} PAP also can be titrated under DISE in selected centers with expertise.

In areas where none of the above are available, one may offer CPAP with pressure around 6–8 cmH\textsubscript{2}O for nonobese nonsyndromic OSAS and 8–10 cm for obese nonsyndromic children and to monitor for clinical response.\textsuperscript{139} Data downloaded from PAP machines are useful in monitoring treatment adherence as parental reports are often not reliable.\textsuperscript{146} Adherence is the major barrier to PAP as an effective therapy for childhood OSAS.\textsuperscript{146,149,150} Behavioral intervention, education, training, and close follow-up were shown to improve PAP adherence.\textsuperscript{155}
A proper interface is crucial for the successful administration of PAP. The ideal interface should ensure comfort and fit, while minimizing leak.\textsuperscript{[152]} Excessive leak can impact on sleep quality, patient–ventilator synchrony, and the amount of effective ventilation delivered to the patient.\textsuperscript{[153]} If a child mouth breathes significantly, a chin strap should be used.

PAP should be provided with a heated humidifier because of the high flow of dry room air that would overwhelm the capacity of the nose to humidify and warm the incoming air. Notwithstanding the above measure, some patients would still have prominent nasal symptoms that would benefit from intranasal steroids. Skin irritation and ulceration can occur from a tight-fitting mask or from accumulation of skin oils and debris from poor mask maintenance.\textsuperscript{[154-157]} Mid-facial hypoplasia was reported with long-term use of nasal CPAP.\textsuperscript{[158]} A study showed that nasal PAP compliant individuals experienced a retrusion of the mid-face after a few years.\textsuperscript{[159]} Use of nasal mask and nasal pillow on alternate nights might be tried to avoid the pressure effect on mid-face. Facial profile should be assessed every year for adverse impact on growth. For children requiring chin strap, the effect on the mandibular condyle should also be assessed yearly.

**Orofacial Myofunctional Therapy**

Orofacial myofunctional therapy (OMT) is potentially an option for the treatment of OSAS. It is defined as the treatment for the muscles of the face and mouth, which is crucial for the maintenance of the craniofacial integrity to achieve normal nasal breathing.\textsuperscript{[160]} OMT reeducation trains a normal and strong sucking, a good mastication employing both sides of jaw, normal swallowing, normal tongue position, and nasal breathing with lips in good contact at rest.\textsuperscript{[13]} Nasal breathing during wake and sleep is the demonstration of normal respiratory functioning, and persistence of mouth breathing is an indicator of an abnormal respiratory function.\textsuperscript{[161]}

Guilleminault et al. reported a retrospective study of 11 children who received OMT.\textsuperscript{[162]} The exercise group was followed up for the first 6 months. Exercise was repeated several times daily in the first 6 months. At 4-year follow-up, the exercise group remained cured of OSA (AHI 0.5 ± 0.4/h) compared to the control group which had a recurrence of OSA (AHI 5.3 ± 1.5/h).

In a prospective, randomized controlled study done by Villa et al., 27 post-TandA children were randomized to either OMT or control group.\textsuperscript{[163]} Children were required to perform exercises every day at home, at least three times a day, 10–20 repetitions each time. Both groups performed nasal washing twice a day. The treatment group consisted of 14 patients and their pre- and post-exercise AHI was evaluated after 2 months of OMT. The AHI decreased from 4.9 to 1.8 ($P = 0.004$) while the control group had minimal change in AHI (4.6–4.1).

In a retrospective case series study done by Lee et al., 26 children out of 64 children had persistent SDB after TandA and 35 of the 64 children showed a pattern of mouth breathing.\textsuperscript{[164]} Eighteen children of the mouth breathing group were followed up for a year with OMT offered. However, only nine of them underwent 6 months of OMT three times a week. The non-OMT group showed a significant worse AHI, 2.9, when compared to the exercise group, 1.1.

A forest plot was constructed with RevMan (version 5.2, The Cochrane Collaborations, London, UK) for the studies of Villa et al., Guilleminault et al., and Lee et al. by the authors (DKN, JPN, and SYL, respectively). The overall AHI was reduced by 0.81 with 95% confidence interval crossing zero [Figure 10]. Hence, further studies are warranted for OMT in childhood OSAS.

**Conclusion**

This is the first position statement on the management of childhood OSA in Asia, which would serve as a guideline for doctors in this area so that a more uniform approach can be adopted for this disease. While there are still considerable knowledge gap in this area, this statement provides the foundation for future studies.

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**Conflicts of interest**

There are no conflicts of interest.

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