

Treatment of pediatric obstructive sleep apnea

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In the majority of cases, pediatric obstructive sleep apnea (OSA) is associated with adenotonsillar hypertrophy. Therefore, adenotonsillectomy is typically considered as the first line of treatment. However, the severity of pediatric OSA is not always directly correlated with the size of the adenoids and tonsils. Other factors, such as upper airway anatomy or obesity, may interact in a multifactorial manner to contribute to its occurrence. For these reasons, sleep apnea in obese children may resemble the condition in adults. Furthermore, in these cases, if adenotonsillar hypertrophy is present, adenotonsillectomy is likely to be prioritized. Reevaluation should be conducted 6 to 8 weeks post-surgery, and additional treatment for residual sleep apnea should be performed thereafter when necessary.

Keywords: Adenoids; Palatine tonsil; Sleep apnea

Introduction

Sleep-related respiratory disorders in pediatric populations encompass a spectrum of conditions, ranging from neonatal apnea to pediatric sleep-related breathing disorders, encompassing all of the manifestations that occur during sleep. Therefore, understanding pediatric sleep-related breathing disorders requires knowledge not only of the anatomical characteristics of the pediatric respiratory system, but also of the physiology of sleep. Specifically, obstructive sleep apnea (OSA) occurs when the physiological balance is disrupted, resulting from a complex interplay of anatomical and physiological factors. The primary anatomical factor of OSA is hypertrophy of the tonsils and adenoids. Particularly in pre-school-aged children, anatomically narrow airways and relatively large tonsils and adenoids are the predominant contributors to this disorder. Moreover, the

upper airway contains numerous proprioceptors that regulate muscle tone, the loss of which leads to reduced muscle tone during the onset of sleep, resulting in increased upper airway resistance. Consequently, individuals with neuromuscular disorders are at an increased risk of developing OSA due to decreased muscle tone (Table 1) [1,2].

In 2012, the American Academy of Pediatrics published guidelines for the treatment of pediatric OSA [3]. These guidelines primarily focus on the regulation of hypertrophic lymphoid tissues and positive airway pressure (PAP) therapy. Key treatments outlined include adenotonsillectomy, PAP therapy, weight loss, and pharmacotherapy [1]. However, various other treatments, including orthodontic treatment, high-flow nasal cannula treatment, myofunctional therapy, and upper airway surgeries, are also currently being employed for the clinical management of pediatric OSA [4].

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Table 1. Representative disorders or conditions predisposing to obstructive sleep apnea in childhood

A. Adenotonsillar hypertrophy or allergic rhinitis
B. Obesity
C. Special craniofacial characteristics or profound craniofacial anomalies
Small mandible with/without mandibular malpositioning
Narrow nasomaxillary complex with/without high and narrow hard palate
Marked nasomaxillary (midface) deficiency (e.g., Apert syndrome, Crouzon syndrome, Pfeiffer syndrome, repaired cleft palate)
Marked mandibular hypoplasia (e.g., Pierre Robin sequence, severe juvenile rheumatoid arthritis, Treacher Collins syndrome, Nager syndrome, Stickler syndrome)
D. Abnormal neuromotor tone or control of breathing
Cerebral palsy
Duchenne muscular dystrophy
E. Combinations of the above disorders or conditions
Down syndrome
Achondroplasia
Prader-Willi syndrome
Mucopolysaccharidoses

Tonsillectomy and adenoidectomy

Tonsillectomy and adenoidectomy are widely recognized as the most effective primary treatment for OSA [5]. The success rate of this surgery in children with OSA is approximately 79%, significantly higher than the 46% success rate observed in individuals managed by observation alone (i.e. without surgery). However, in high-risk groups with severe obesity, craniofacial anomalies, or neuromuscular disorders, there is a higher likelihood of persistent symptoms or recurrence of OSA even after successful surgery. Additionally, surgical interventions carry inherent risks. Therefore, in high-risk cases, the benefits of surgery must be carefully weighed, and continuous postoperative monitoring for recurrence is crucial.

With the exception of postoperative pain, complications following surgery for OSA are rare and typically minor. However, serious complications, such as excessive bleeding from the surgical site, pulmonary edema, and airway obstruction, can occur, with an incidence of 1 in 16,000 to 35,000 cases resulting in postoperative complications. The criteria for high-risk groups of surgical complications include an age of less than 3 years, craniofacial anomalies affecting the upper airway, growth disorders, muscle tone

abnormalities, obesity, neuromusculoskeletal disorders, cardiovascular complications of OSA (right ventricular hypertrophy and pulmonary hypertension), a history of upper airway trauma, concomitant tonsillectomy and adenoidectomy with craniofacial surgery, and respiratory infections. According to polysomnography criteria, individuals with severe OSA (apnea hypopnea index ≥ 10 or minimum arterial oxygen saturation $< 80\%$, or both) are considered to be at high-risk for complications after tonsillectomy and adenoidectomy [6].

In efforts to reduce postoperative complications, various surgical techniques employing different instruments, including radiofrequency, microdebrider, coblator, and harmonic scalpel, have been introduced. Partial intracapsular tonsillectomy and adenoidectomy (PITA), which entails less bleeding and pain, is also a treatment option. However, it is noted that PITA may lead to tonsillar tissue regrowth and the potential for the recurrence of OSA [7].

PAP therapy

Following tonsillectomy and adenoidectomy, residual OSA persists in 13% to 29% of low-risk pediatric patients. In high-risk pediatric populations, such as those with obesity, neuromuscular disorders, or conditions like Down syndrome (trisomy 21), over 70% may continue to exhibit OSA even after surgery. Additionally, children who refuse surgery or have contraindications to general anesthesia cannot undergo tonsillectomy and adenoidectomy. In these cases, the most effective treatment option is PAP therapy [8].

Although PAP therapy has in use for over 30 years, in recent years its usage has increased over 3-fold compared to previous rates of intervention. At present, various types of PAP devices are available in children, including auto-titrating PAP, continuous PAP (CPAP), and bilevel PAP (BiPAP). However, CPAP or BiPAP are predominantly used. The selection of the PAP device based on an individual patient's needs can enhance compliance. The types and indications of PAP therapy for pediatric OSA treatment are as follows (Table 2). To determine the appropriate pressure for CPAP therapy, a sleep study needs to be conducted. Unlike in adults, split-night polysomnography is not typically performed in children to determine optimal pressure, as the sudden application of a mask during the study can negatively affect compliance. Thus, gradual adaptation to

Table 2. PAP devices used in pediatric patients

Mode	Primary indication and clinical utility
CPAP: Fixed-pressure CPAP	OSA
Auto-CPAP: Auto-titrating CPAP mode	1. OSA 2. Positional or REM-related OSA 3. PAP therapy acclimatization prior to PSG 4. CPAP patients with sudden changes in OSA severity due to surgery or rapid weight change
BiPAP-S: Spontaneous BiPAP mode	Patients with OSA who are intolerant to CPAP at high pressures due to discomfort exhaling, not mitigated by comfort features
Auto-BiPAP: Auto-titrating BiPAP mode	1. Positional or REM-related OSA where patient is intolerant to high Auto-PAP pressures 2. BiPAP therapy acclimatization prior to PSG
BiPAP-ST: Spontaneous-timed BiPAP mode	Children with OSA who present with mixed apnea, CPAP emergent central apnea, or persistent hypoventilation following resolution of OSA with CPAP
VAPS: VAPS BiPAP mode	Obesity hypoventilation syndrome Congenital central hypoventilation syndrome

PAP, positive airway pressure; CPAP, continuous PAP; OSA, obstructive sleep apnea; REM, rapid eye movement; PSG, polysomnography; BiPAP, bilevel PAP; VAPS, volume-assured pressure support.

wearing the mask followed by titration polysomnography for pressure adjustment is recommended [9].

A significant challenge of PAP therapy in children is lower compliance compared to adults. Children in their growth phase require periodic mask replacement due to facial skeletal growth. Furthermore, pressure caused by the mask may adversely affect facial bone development. Moreover, parental involvement is essential, necessitating education for parents and caregivers. Recent advances in interfaces have somewhat improved comfort with PAP therapy, making it effectively implementable even in infants and adolescents [10].

Weight loss

While there is a substantial amount of evidence indicating that surgical or nonsurgical weight loss improves OSA in adults, literature regarding the effects of weight loss on pediatric OSA improvement is somewhat limited. That being said, overall, studies recommend weight loss for obese patients with pediatric OSA [11,12]. In cases of severely obese patients with pediatric OSA where weight loss is unsuccessful, bariatric surgery may be considered, although this option should be evaluated carefully before proceeding [13,14].

Medical treatment

In the treatment of OSA, pharmacotherapy aims to reduce

the size of the tonsils and adenoids. An increase in the expression of leukotriene receptor 1, 2, and leukotriene C4 synthase has been reported in the tonsillar tissue of patients with pediatric OSA compared to that in healthy children. Several clinical studies have reported on the efficacy of the leukotriene receptor antagonist montelukast in pediatric OSA [15]. Nasal corticosteroids have also been reported to be effective in the treatment of pediatric OSA, as their anti-inflammatory action is known to reduce the size of the tonsils and adenoids [16]. Although some studies have reported on the effects of combined therapy with these two drugs, further research is needed to fully ascertain the benefits of combination therapy. Therefore, montelukast or nasal corticosteroid therapy may be considered as an alternative treatment for pediatric OSA in cases where surgery is not feasible, as well as in mild cases.

Other treatment methods

Orthodontic interventions targeting malocclusion and maxillofacial anomalies are also associated with the treatment of OSA. Dental orthodontic treatments applied to pediatric OSA include rapid maxillary expansion, intraoral appliances, midface distraction osteogenesis, and mandibular osteotomies [17,18]. Various studies have reported on the effectiveness of orthodontic treatment in increasing airway volume and improving OSA. Among these, intraoral appliances and rapid maxillary expansion are commonly

employed methods. In particular, rapid maxillary expansion may be considered as a primary treatment method for patients with pediatric OSA with midface hypoplasia in the central one-third of the face, narrow and high palates, and submerged maxillary arches with associated malocclusion, provided there is no obesity or adenotonsillar hypertrophy (grade 1) [19]. In children, rapid maxillary expansion is typically performed without surgery using dental orthodontic appliances, applying force directly to the teeth and maxilla using expanders with multiple arms. Surgical intervention may be necessary after midpalatal suture fusion [19]. In cases of persistent OSA, an evaluation for multilevel obstruction should be conducted following adenotonsillectomy and orthodontic treatment. Depending on the case, additional interventions, such as uvulopalatal flap, lingual tonsillectomy, or nasal surgery, may need to be performed.

Intraoral appliances are suitable for mild to moderate OSA and are usually applied after any permanent dentition has erupted. Upper airway myofunctional therapy strengthens upper airway muscles through exercises involving the tongue, soft palate, facial muscles, and mandible to alleviate OSA. Recently, passive upper airway myofunctional therapy using intraoral appliances for mandibular advancement has been introduced as a treatment option for OSA [20]. While upper airway myofunctional therapy is effective for patients with mild OSA, compliance remains a limitation of this approach [21,22]. High-flow nasal cannula therapy delivers humidified air to the upper airway through a nasal cannula and can be considered as an alternative therapy for infants or children with maxillofacial developmental anomalies who cannot tolerate mask-based therapies, such as CPAP [23].

In cases of severe respiratory distress due to upper airway obstruction that poses a life-threatening situation, in addition to being unresponsive to other methods, such as CPAP, a tracheostomy may be warranted [24].

Conclusions

Adenotonsillar hypertrophy and obesity represent the predominant predisposing factors for OSA syndrome in pediatric populations, often accompanied by significant morbidity. Common treatment modalities include adenoidectomy alone or in combination with tonsillectomy, intranasal corticosteroids, oral appliances, and nasal CPAP. In cases of

severe upper airway obstruction where other interventions prove ineffective in establishing airway patency, tracheostomy remains a crucial and urgent intervention.

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Conflicts of interest

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