Pediatric Obstructive Sleep Apnea Syndrome: A Hidden Disorder with Lifelong Consequences—Narrative Review

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ABSTRACT

An estimated 1% to 5% of youngsters are thought to suffer from obstructive sleep apnoea (OSA). Untreated paediatric OSA can lead to cardiovascular problems, such as systemic and pulmonary hypertension, behavioural abnormalities, poor academic performance, and severe neurocognitive impairment. There are many different treatment options available, including both non-surgical and surgical methods. As our knowledge of the pathophysiology of paediatric OSA continues to grow, so do the available management choices. In addition to considering alternate surgical and non-surgical approaches, this study attempts to provide an overview of the regularly used treatment modalities for paediatric OSA, with a focus on adenotonsillectomy and positive airway pressure therapy. Recent advancements in therapeutic approaches and diagnostic methods are also addressed.

Keywords: sleep-disorder, polysomnography, adenotonsillectomy, Pediatric SDB

How to cite this article: Ranganathan S, Ramachandran S, Rajvikram N, Sekaran DC, Keerthana S. Pediatric Obstructive Sleep Apnea Syndrome: A Hidden Disorder with Lifelong Consequences—Narrative Review. Int J Drug Deliv Technol. 2026;16(71s): 934-944. DOI: 10.25258/ijddt.16.71s.108

INTRODUCTION

A fundamental physiological function that is dynamic and cyclical, the sleep cycle is essential to general health. Significant developmental changes that occur throughout the early childhood years have an impact on later typical sleep-wake cycles.[1] Paediatric obstructive sleep apnoea (POSA) and sleep disordered breathing have received more attention recently and are now acknowledged as significant clinical and scientific issues.[2] These disorders include obstructive hypopnea, snoring, upper airway resistance syndrome, and obstructive sleep apnoea (OSA), among other sleep-related breathing disorders. Because paediatric SDB is linked to a number of comorbidities, including obesity, cardiovascular problems, and neurocognitive impairments, it poses a serious public health concern. Recurrent episodes of partial or total upper airway blockage during sleep are the hallmark of Obstructive Sleep Apnoea Syndrome (OSAS), which causes disturbed breathing and changes the architecture of sleep.[2]

Children with underlying genetic conditions such Down syndrome, craniofacial abnormalities, and neuromuscular diseases are at a higher risk of developing Obstructive Sleep Apnoea (OSA). Clinically, paediatric OSA typically presents as caregiver-reported snoring or difficulty breathing while they sleep, along with hyperactive and excessive sleepiness during the day. Untreated OSA in children has serious aftereffects, including behavioural issues, neurocognitive impairment, poor academic performance, and elevated cardiovascular risk. As a result, paediatric OSA contributes significantly to the burden of long-term healthcare. Overnight polysomnography (PSG) performed in a sleep center is the gold standard for diagnosis. The obstructive apnea–hypopnea index (AHI), which is derived from PSG recordings, reflects the severity of the disease. In general, paediatric OSA is categorised as mild if the AHI is between 1 and 5 occurrences per hour, moderate if it is between 5 and ≤10 events per hour, and severe if it is more than 10 events per hour [3,7].

EPIDEMIOLOGY

Considering an estimated global rate ranging from 1-4% in the paediatric population, paediatric obstructive sleep apnoea (POSA), the most severe end of the spectrum of sleep-disordered breathing, is a very common illness.[8] The age distribution is bimodal, according to epidemiological data. Adenotonsillar enlargement is related with the first prevalence peak, which is seen between the ages of 2 and 8; excessive weight gain is mostly responsible for the second increase, which takes place during adolescence. Although the prevalence of POSA in prepubertal children seems to be similar for both

sexes, certain research indicate that teenage males are more likely than females to have the condition.[9] Males regularly have a two- to three-fold higher risk than females in maturity. children of African-American and Asian descent, those with concurrent respiratory disorders like asthma or allergic rhinitis, youngsters who are obese, and people with a positive family history of OSA have all been found to have higher prevalence rates.[10,11] Although the main cause of POSA is still adenotonsillar hypertrophy, the rising incidence of childhood obesity has significantly raised illness rates in all age groups, including teenagers.

ETILOGY

Etiological Factor	Description
Adenotonsillar Hypertrophy	Enlargement of the tonsils and adenoids is a primary contributing factor, leading to mechanical obstruction of the upper airway during sleep.
Macroglossia and Upper Airway Soft Tissue Enlargement	Increased size of oral or pharyngeal soft tissues, including the tongue, may narrow the airway lumen and impair airflow, particularly in the supine position.
Allergic Conditions	Chronic allergic rhinitis and persistent upper airway inflammation can cause nasal obstruction and increased airway resistance, predisposing children to POSA.
Obesity	Excess adipose tissue in the neck and pharyngeal region contributes to airway narrowing and reduced airway stability during sleep.
Reduced Neuromuscular Tone	Decreased muscle tone or weakness of the upper airway dilator muscles compromises airway patency, especially during sleep.
Craniofacial Abnormalities	Structural anomalies such as maxillary constriction, high-arched or narrow palate, retrognathia, or reduced airway dimensions can restrict airflow and increase the risk of POSA.
Neuromuscular Disorders	Disorders affecting neuromuscular control can cause respiratory and upper airway muscles to become less coordinated and toned, which can result in airway collapse as you sleep.
Prematurity	Children born prematurely may exhibit underdeveloped airway structures and immature neuromuscular control, increasing susceptibility to sleep-related breathing disorders.[12-14]

PATHOPHYSIOLOGY

The pathophysiology of pediatric Obstructive Sleep Apnea Syndrome (OSAS) is predominantly associated with adenotonsillar hypertrophy, while obesity, craniofacial abnormalities, and neuromuscular disorders represent less frequent contributing factors.[9] In affected children, airway obstruction most commonly occurs at the level of the tonsils and adenoids, whereas normal children demonstrate airway narrowing primarily at the soft palate.[15] Airway patency is typically preserved during wakefulness due to increased upper airway muscle tone, although adenotonsillar size alone does not consistently correlate with disease severity.[16,17] Despite generally normal ventilatory drive, altered central regulation of upper airway neuromotor activity during sleep predisposes to airway collapse.[18] Although children possess a relatively less collapsible airway and higher baseline pharyngeal muscle tone compared with adults, sleep-related reduction in neuromuscular compensation plays a key role in the development of airway obstruction

INVESTIGATION

Clinical Suspicion

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Concentrated Sleep Record

• Regular snoring

• Daytime drowsiness , Behavioural issues, learning challenges, and attention deficiencies

• Having trouble breathing or seeing periods of apnoea as you sleep

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Physical and Growth Assessment

• Detailed oropharyngeal examination

• Assessment of tonsillar size

• Evaluation of craniofacial anomalies and maxillomandibular development

• Growth assessment (obesity or failure to thrive)

• Screening for systemic or pulmonary hypertension

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Airway Assessment

• Direct laryngoscopy for tonsillar and adenoidal evaluation

• Flexible nasopharyngoscopy for dynamic airway assessment

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Diagnostic Investigations

• Overnight polysomnography (PSG) test– gold standard
• Upper airway imaging (MRI / CT)
• Soft tissue radiographs (lateral / anteroposterior views)

EVALUATION OF THE SEVERITY OF PEDIATRIC SLEEP APNEA

Overnight polysomnography (PSG) results are used to assess the severity of paediatric obstructive sleep apnoea (OSA), which is crucial for directing treatment choices and forecasting clinical results. By counting the number of apneic and hypopneic episodes per hour of sleep, the Apnea–Hypopnea Index (AHI) is used to measure the severity of the disease. Paediatric OSA is categorised as mild (>1 to ≤5 events/hour), moderate (>5 to ≤10 events/hour), severe (>10 events/hour), and normal (≤1 event/hour) based on AHI readings [19].

DIAGNOSTIC CRITERIA OF OSAS IN CHILDREN

Commonly Observed Signs	Less Frequently Observed Signs
Habitual nocturnal snoring	Excessive daytime sleepiness
Predominant mouth breathing	Reduced appetite
Restless or fragmented sleep, with or without arousals	Poor weight gain or growth faltering
Witnessed pauses in breathing during sleep	Recurrent vomiting
Recurrent respiratory tract infections	Swallowing or feeding difficulties
Persistent nasal discharge	Behavioral or emotional disturbances
Excessive sweating during sleep	Recurrent otitis media
Nocturnal enuresis	—

Treatment of Pediatric Obstructive Sleep Apnea(POSA)

ADENOTONSILLECTOMY

For children with obstructive sleep apnoea (OSA), it is generally considered the first-line therapy approach. When enlarged tonsils and adenoids are surgically removed, the upper airway lumen enlarges, decreasing the risk of airway collapse during sleep. Adenotonsillar hypertrophy is a known risk factor for airway obstruction in children. Adenotonsillectomy directly addresses the underlying pathophysiology of paediatric OSA by removing the main anatomical blockage.

There is excellent clinical data to support the efficacy of adenotonsillectomy. A major randomised controlled trial called the Childhood Adenotonsillectomy Trial (CHAT) assessed the results of surgical surgery against careful waiting in children with OSA. About 79% of children who had surgery in the experiment showed remission of obstructive episodes, compared to 46% in the non-surgical observation group. While there were no significant differences between the groups in the core neurocognitive outcomes, which include measures of executive function and attention, a number of secondary outcomes demonstrated notable improvement after surgery. These included positive changes in polysomnographic indices, improved behaviour as observed by carers, decreased daytime sleepiness, and increased quality of life.

A later Cochrane systematic review that compared conservative therapy with adenotonsillectomy found that there is moderate-quality evidence to support surgical intervention for improving behavioural outcomes, overall quality of life, and OSA symptoms. However, the review also noted that there was no discernible difference in neurocognitive function or attentional measures between children who underwent surgery and those who were placed under watchful waiting.

Adenotonsillectomy is linked to possible postoperative problems despite its effectiveness. Respiratory compromise is the most often reported consequence. It usually manifests as ongoing oxygen desaturation in the early postoperative phase and may call for further oxygen therapy. About 9% of instances have been recorded to have this problem, which usually goes away during recovery. There have been reports of hemorrhagic complications at rates of roughly 2–3%, including primary haemorrhage during the first 24 hours and secondary haemorrhage afterward. Dehydration, discomfort, and decreased oral intake are further postoperative issues.Despite being uncommon, adenotonsillectomy-related mortality has been documented; estimated rates range from 1 in 16,000 to 1 in 35,000 surgeries. Age under three years, severe OSA, underlying heart disease, failure to thrive, obesity disorder, neuromuscular abnormalities, and the presence of active respiratory infections at the time of surgery are some of the patient-related factors that raise the risk of postoperative complications. Perioperative surveillance and cautious patient selection are therefore crucial. In order to enable educated decision-making in the management of paediatric OSA, it is crucial that carers get comprehensive counselling regarding the expected advantages, success rates, potential hazards, and limitations of adenotonsillectomy when surgical management is being considered [20,21].

UVULOPALATOPHARYNGOPLASTY (UPPP)

Only 40–50% of mild to moderate cases of paediatric obstructive sleep apnoea have been shown to improve following uvulopalatopharyngoplasty, and the benefits decline over time.. The procedure is associated with notable postoperative risks, including velopharyngeal insufficiency, hemorrhage, dysphagia, voice changes, and airway narrowing, with rare mortality reported.[3] The uvulopalatal flap technique reduces postoperative pain and the risk of velopharyngeal insufficiency but retains similar complication risks and is therefore mainly performed in adults. Tissue ablation techniques offer greater precision and lower thermal injury but often require multiple sessions and carry a small risk of ulceration or infection.[22]

Mandibular Distraction Osteogenesis

Mandibular distraction osteogenesis is a surgical treatment option for selected pediatric patients, particularly older children with fused cartilage or those who do not require maxillomandibular advancement. This technique, often combined with rapid maxillary expansion and adenotonsillectomy, has shown favorable outcomes in children aged 5–16 years presenting with craniofacial features such as a high-arched palate and unilateral or bilateral posterior crossbite.[3] In syndromic patients, mandibular distraction increases oropharyngeal airway space by repositioning the tongue anteriorly, thereby reducing airway obstruction. Rapid maxillary expansion may be performed concurrently with mandibular distraction during adolescence to further enhance airway patency.[23]

NEUROSTIMULATION

Hypoglossal nerve stimulation (HGNS) is a novel surgical approach for obstructive sleep apnea, approved for adult use since 2014. An implantable device detects respiratory effort during sleep and delivers electrical stimulation to the hypoglossal nerve, promoting tongue protrusion and maintaining airway patency. Limited pediatric data, primarily in children with Down syndrome, have demonstrated marked reductions in apnea–hypopnea index and good treatment adherence. However, HGNS is not currently approved for pediatric patients. Neurostimulation may represent a promising option for selected complex pediatric cases, particularly in children who are intolerant of continuous positive airway pressure therapy.[24,25]

NON-SURGICAL TREATMENT

Other non-surgical treatment options are available when pediatric OSA does not improve after surgery or when surgery is contraindicated due to associated medical conditions. These therapies, including medical and supportive interventions, are increasingly recognized as safe and effective, especially for children with mild to moderate obstructive sleep apnea.

Anti-inflammatory medications

* Steroids and leukotriene receptor antagonists reduce upper-airway inflammation and decrease tonsil and adenoid size in pediatric OSA.
* Leukotriene activity is increased in children with OSA; drugs like montelukast counteract this inflammatory pathway.
* Clinical trials show leukotriene antagonists significantly reduce AHI in children with mild OSA.
* Intranasal steroids (e.g., budesonide) also improve AHI by shrinking lymphoid tissue.
* Combined use of leukotriene antagonists and nasal steroids may be beneficial, though superiority over single therapy is unproven.
* These medications are useful in mild OSA, in children unfit for surgery, or after failed adenotonsillectomy, with ongoing symptom monitoring required[29,30]

HIGH FLOW NASAL CANNULA

High-flow nasal cannula therapy has emerged as a potential non-invasive alternative to continuous positive airway pressure in the management of pediatric obstructive sleep apnea. This modality delivers a steady flow of heated and humidified air through a soft nasal interface, helping to maintain airway patency during sleep.Although the precise mechanism of action is not fully understood, proposed explanations include mechanical support of the upper airway, reduction of nasal congestion due to humidification, and enhancement of upper-airway muscle tone. Early studies have demonstrated meaningful improvements in the apnea–hypopnea index with this therapy, suggesting a beneficial role in select patients.While larger and more definitive studies are still required to establish its long-term efficacy and indications, high-flow nasal cannula therapy may be considered particularly in younger children and in those who are unable to tolerate CPAP therapy.[31,32]

ORAL APPLIANCES

Mandibular advancement devices are well established in adults in range from mild to moderate obstructive sleep apnea, functioning by repositioning the lower jaw forward to widen the upper airway and reduce collapse. In children, evidence for their use is more limited, but emerging data suggest potential benefit. Reviews of pediatric studies, including randomized controlled trials, indicate that these appliances can lead to modest reductions in apnea severity. Despite these encouraging findings, concerns remain regarding long-term use in growing children, particularly the possibility of effects on facial growth and dental development. As a result, while oral appliances may offer a non-invasive treatment option for selected pediatric patients, further research is required to clarify their long-term safety and effectiveness[33,34]

OXYGEN THERAPY

Upper airway obstruction in obstructive sleep apnea results in reduced airflow and recurrent drops in blood oxygen levels. Supplemental oxygen has therefore been used to address nocturnal desaturation and may also influence ventilatory control by modifying the body's response to hypoxemia. This approach has gained particular attention in infants, as CPAP is not approved for children weighing under 13 kg. Studies in infants receiving low-flow oxygen during sleep have demonstrated improvements in overall oxygen saturation, lowest oxygen levels, and, in some cases, reductions in the apnea–hypopnea index. Research in older children has similarly shown better oxygenation, although effects on apnea frequency have been inconsistent.[35,36] While average carbon dioxide levels generally remain stable, occasional cases of elevated carbon dioxide have been reported, highlighting the need for careful monitoring. Oxygen therapy is best viewed as a temporary supportive option, especially in young infants with mild disease who are expected to improve with growth, with close surveillance for hypercapnia.

POSITIONAL THERAPY

OSA may worsen in the supine position due to backward collapse of the tongue and airway structures. Positional therapy aims to promote side-sleeping using behavioral methods or assistive devices. While adult studies show benefit, pediatric studies report mixed results. There is limited evidence supporting its effectiveness in children. Long-term safety and practicality in the pediatric population remain unclear. As a result, positional therapy is not routinely recommended. It may be considered only as an adjunct in selected cases[37,38].

WATCHFUL WAITING

Children undergo continuous growth, which may naturally improve factors contributing to OSA. Some cases of mild pediatric OSA resolve without medical or surgical intervention. Clinical trials have shown that nearly half of children improve with observation alone. Persistence of OSA is more likely in obese children or those with higher baseline AHI. For mild instances with no serious comorbidities, watchful waiting is appropriate. In order to identify deteriorating symptoms, regular follow-up is necessary. This strategy ought to be tailored according to risk factors.

CONCLUSION

Even caretakers and medical experts regularly fail to recognise and misdiagnose paediatric obstructive sleep apnoea. For early detection and prompt intervention, paediatricians, pulmonologists, otolaryngologists, and paediatric dentists must raise awareness. Negative impacts on behaviour, growth, cardiovascular health, and neurocognitive development can be avoided with early diagnosis and proper treatment. Understanding of illness causes and severity evaluation has increased because to developments in paediatric sleep medicine and polysomnographic techniques. Given that treatment must be tailored to each patient's age, severity, and related comorbidities, a multidisciplinary approach is essential. To enhance long-term outcomes for kids with obstructive sleep apnoea, broaden therapy options, and improve diagnostic criteria, more research and education are required.

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