Review

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Multidisciplinary Management of Pediatric Obstructive Sleep Apnea: From Diagnosis to Long-Term Follow-Up

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Pediatric obstructive sleep apnea (OSA) is a multifactorial condition with serious neurocognitive, cardiovascular, and metabolic consequences. While adenotonsillectomy (AT) remains the first-line therapy for many cases, complex presentations involving obesity, craniofacial anomalies, or neuromuscular disorders require coordinated multidisciplinary management. This review provides a comprehensive synthesis of current strategies across diagnosis, treatment, and long-term follow-up. Diagnostic approaches include clinical screening with a combination of physical examination, polysomnography, imaging, and drug-induced sleep endoscopy. Treatment options range from surgical interventions to positive airway pressure therapy, pharmacologic treatments, dental and orthodontic approaches, behavioral modification, and psychological therapy. Special considerations are given to high-risk populations such as children with Down syndrome, Prader-Willi syndrome, neuromuscular disease, and extreme obesity. The emphasis is on residual or refractory OSA and the importance of individualized treatment pathways, shared decision-making, and multidisciplinary clinics. Longitudinal care—neurocognitive monitoring, cardiometabolic surveillance, and digital health tool use—is critical for optimizing long-term outcomes. By synthesizing evidence across specialties, this review provides practical guidance for comprehensive, patient-centered care in pediatric OSA.

Keywords: pediatric obstructive sleep apnea; multidisciplinary management; polysomnography; adenotonsillectomy; positive airway pressure therapy

Introduction

Pediatric obstructive sleep apnea (OSA) is a sleep disorder characterized by repeated episodes of upper airway obstruction during sleep, leading to ventilatory disruption, intermittent hypoxemia, and sleep fragmentation [1,2]. Clinical presentation differs from adults in that children tend more frequently to present with behavioral disturbances such as hyperactivity, attention deficits, or poor academic performance, rather than obvious excessive daytime sleepiness. Characteristic symptoms include frequent snoring, witnessed apneas, and nocturnal enuresis (bed-wetting) during sleep; daytime somnolence may also occur but is often less pronounced or may be masked by hyperactive behavior [3]. Untreated pediatric OSA can result in significant morbidity in the form of neurocognitive and cardiovascular dysfunction (e.g., hypertension, endothelial dysfunction) and metabolic derangements like insulin resistance and dyslipidemia [4,5]. Given these risks, pediatric OSA is a significant public health concern that needs early diagnosis and comprehensive management. Though adenotonsillectomy (AT) remains the cornerstone for the management of OSA in children, most cases involve multiple contributing factors, including obesity, craniofacial abnormalities, neuromuscular disorders, and behavioral conditions. In such cases, single-specialty management—often led by otolaryngologists—is insufficient [2,3]. Multidisciplinary teams improve compliance and long-term outcomes with pediatric OSA, particularly complicated cases [6].

This review seeks to offer a pragmatic, multidisciplinary summary of pediatric OSA care from diagnosis to long-term follow-up. By integrating perspectives from multiple specialties, it offers proposes coordinated strategies. Unlike traditional reviews that emphasize surgical or pharmacologic approaches in isolation, this review incorporates clinical workflows, algorithm-based diagnostic pathways, and multidisciplinary treatment strategies.

Topics include epidemiology and pathophysiology, screening and diagnostic tests, operative and non-operative treatment, longitudinal follow-up, management in high-risk populations, resource-limited setting challenges, and future directions. The goal is to support clinicians in translating current evidence into effective, comprehensive care for children with OSA.

Epidemiology and Pathophysiological Basis

Prevalence and Risk Factors

The prevalence of pediatric OSA has been estimated to range from 1.2% to 5.7%, with the highest rates reported in children aged 4 to 6 years [7]. Variability in prevalence estimates is largely attributable to differences in study population, diagnostic criteria, and assessment methods [4]. The most common risk factor is adenotonsillar hypertrophy, particularly in younger children [8]. Obesity is also a significant risk factor, especially among adolescents, due to fat deposition in the upper airway with consequent obstruction during sleep. Craniofacial anomalies, such as micrognathia and midface hypoplasia, predispose children to OSA by altering airway anatomy [9].

Neuromuscular illnesses, including cerebral palsy and muscular dystrophy, are associated with higher risk of OSA due to hypotonia that compromises airway patency. Additionally, those with genetic syndromes such as Prader–Willi syndrome and Down syndrome are at an increased risk of developing OSA, owing to features such as macroglossia, midface hypoplasia, and generalized hypotonia [4].

Environmental exposures such as tobacco smoke and allergens, may also contribute to the development or exacerbation of OSA. Furthermore, male sex has been identified as a potential risk factor, with one study reporting a higher prevalence in boys than in girls [8].

Mechanistic Insights (Airway Anatomy, Neuromuscular Tone, Inflammation)

Pediatric OSA is characterized by recurrent upper airway obstruction during sleep, resulting from the interaction of complex anatomical, neuromuscular, and inflammatory mechanisms [10]. From an anatomical perspective, adenotonsillar hypertrophy, craniofacial abnormalities (e.g., retrognathia and midface hypoplasia), and obesity-related fat deposition in the upper airway contribute to airway narrowing and increased susceptibility to inspiratory collapse [10]. Neuromuscular mechanisms involve reduced tone and impaired responsiveness of the pharyngeal dilator muscles during sleep, which further predispose the airway to obstruction [10,11]. Inflammatory processes also play a significant role. Recurrent episodes of intermittent hypoxia and oxidative stress associated with apneic events may induce both local and systemic inflammation, leading to tissue remodeling and further compromise of airway integrity [11]. Elevated levels of proinflammatory cytokines, including interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), have been reported in children with OSA and correlate with disease severity [11]. These pathophysiological mechanisms underscore the need for multimodal evaluation and individualized treatment strategies in pediatric OSA.

Diagnostic Approaches: A Multidisciplinary Workflow

Initial Clinical Screening: Pediatric Sleep Questionnaire (PSQ) and Physical Examination Findings

The Pediatric Sleep Questionnaire (PSQ) is a validated screening tool used to identify sleep-disordered breathing in children aged 2 to 18 years [12]. In settings where polysomnography (PSG) is unavailable, the PSQ may serve as a practical alternative for initial risk assessment. The questionnaire consists of 22 items evaluating symptoms such as snoring frequency, witnessed apneas, daytime sleepiness, and behavioral concerns, including inattention and hyperactivity.

Studies have demonstrated that the PSQ can predict moderate-to-severe OSA with acceptable accuracy in obese children, with an area under the receiver operating characteristic curve (AUC) of 0.88, sensitivity of 80%, and specificity of 100% at a cutoff value of 0.65 [13]. A PSQ score $>$0.33 indicates a high risk of OSA and has shown good sensitivity and specificity across various populations [12–14].

Physical examination findings also contribute to the initial evaluation of pediatric OSA. Common features include adenotonsillar hypertrophy, craniofacial abnormalities (e.g., retrognathia and midface hypoplasia), and signs of obesity, such as increased neck circumference. These factors may contribute to upper airway obstruction during sleep [15]. However, neither the PSQ nor physical examination alone is sufficient for a definitive diagnosis. Objective sleep testing, particularly PSG for measurement of the apnea–hypopnea index (AHI) and related parameters, is required [15]. Once a high risk of OSA is identified through screening, a structured multidisciplinary diagnostic pathway should be implemented to guide further evaluation and management (see Fig. 1).

Role of ENT Specialists, Pediatricians, Dentists, and Sleep Specialists in Early Evaluation

Multidisciplinary collaboration among pediatricians, otolaryngologists (ENT specialists), dentists, and sleep specialists enhances diagnostic accuracy and facilitates individualized treatment planning [16].

ENT specialists play a central role in evaluating anatomical contributors to airway obstruction, including adenotonsillar hypertrophy, nasal septal deviation, and turbinate hypertrophy. They perform detailed assessments, such as flexible nasopharyngoscopy and endoscopic examination, to identify sites of airway obstruction. ENT evaluation is particularly important when surgical intervention, such as adenotonsillectomy (AT), is being considered [17].

Pediatricians are typically the first point of contact and are responsible for initial screening during routine healthcare visits. They assess risk factors, including obesity,

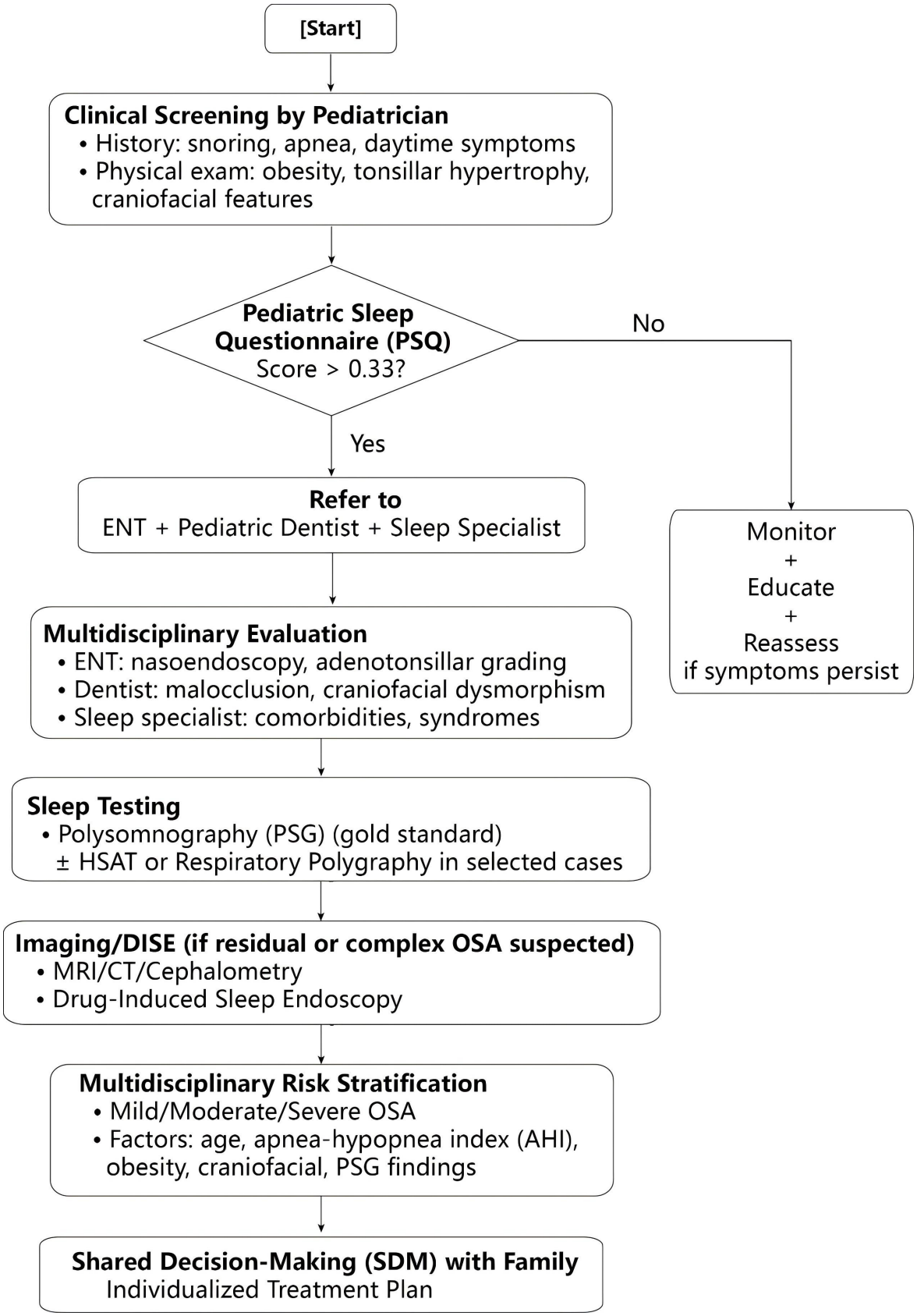

Fig. 1. Diagnostic algorithm for pediatric obstructive sleep apnea (OSA): from clinical screening to multidisciplinary diagnosis. This streamlined algorithm outlines the stepwise diagnostic pathway for pediatric OSA, starting with clinical screening and the Pediatric Sleep Questionnaire (PSQ), followed by multidisciplinary evaluation, objective sleep testing, and risk stratification leading to shared decision-making (SDM) for individualized treatment.

craniofacial abnormalities, and neuromuscular disorders, as well as symptoms such as snoring, daytime sleepiness, and behavioral disturbances. Screening tools such as the PSQ are commonly used to identify at-risk children and facilitate timely referral for specialist evaluation [13].

Orthodontists and pediatric dentists are well positioned to identify craniofacial abnormalities and oral features associated with OSA [18]. During routine dental examinations, they may detect findings such as tonsillar hypertrophy, retrognathia, and a high-arched palate. Their role in therapeutic interventions is discussed in Section 4.2.3.

Sleep specialists play a pivotal role by interpreting sleep study results (e.g., PSG and HSAT), providing expertise in sleep physiology, and guiding diagnostic and therapeutic decision-making. Their involvement ensures accurate integration of objective sleep data into clinical assessment and supports appropriate treatment planning, as illustrated in the diagnostic algorithm (Fig. 1) [4,16].

Sleep Studies: Polysomnography, Home Sleep Apnea Testing, and Respiratory Polygraphy

Polysomnography (PSG) is the gold standard for diagnosing pediatric OSA. It involves overnight monitoring of multiple physiological parameters, including electroencephalography, electrooculography, electromyography, electrocardiography, airflow, respiratory effort, and oxygen saturation. PSG provides quantitative data on sleep architecture and respiratory events, enabling accurate diagnosis and severity classification [15].

Home Sleep Apnea Testing (HSAT) has been explored as an alternative to in-laboratory PSG, particularly in settings with limited access to sleep laboratories. HSAT utilizes portable devices that measure a limited set of parameters, such as airflow, respiratory effort, and oxygen saturation. Although HSAT is convenient and cost-effective, its use in children remains limited due to concerns regarding accuracy, particularly in younger patients and those with comorbidities [19,20]. Most validation studies in pediatric populations involve small sample sizes and often exclude younger children or those with significant comorbid conditions. Technical limitations, including signal loss, poor sensor tolerance, and reduced sensitivity for mild disease, further compromise reliability. Therefore, while HSAT may be considered in selected older children and adolescents for screening or follow-up, it is not recommended as a standalone diagnostic tool. PSG remains the reference standard.

Respiratory polygraphy (RP) is another diagnostic modality that records respiratory parameters without assessing sleep stages [21,22]. It typically measures airflow, respiratory effort, and oxygen saturation. Although less comprehensive than PSG, RP may be useful in specific clinical contexts, such as resource-limited settings or when PSG is unavailable [23]. However, the absence of sleep stage data may lead to misestimation of OSA severity. In summary, although PSG remains the gold standard for pediatric OSA diagnosis, HSAT and RP may serve as complementary tools in selected scenarios. The choice of diagnostic modality should be individualized based on patient characteristics, resource availability, and clinical expertise.

Imaging Modalities and Drug-Induced Sleep Endoscopy (DISE)

Imaging modalities and drug-induced sleep endoscopy (DISE) play important roles in evaluating pediatric OSA, particularly in complex or treatment-resistant cases [24,25]. When used appropriately, these approaches provide complementary information and support individualized treatment planning.

Various imaging techniques are used to assess anatomical contributors to OSA. Lateral neck radiographs can identify adenoidal hypertrophy, while cephalometric imaging evaluates craniofacial structures. Magnetic resonance imaging (MRI) and computed tomography (CT) provide high-resolution visualization of soft tissue and skeletal anatomy, facilitating identification of airway obstruction. These modalities are particularly useful in children with craniofacial abnormalities or persistent OSA following AT [26]. However, concerns regarding radiation exposure and the need for sedation in younger children limit their routine use.

DISE involves pharmacologically induced sleep to allow dynamic visualization of the upper airway using flexible endoscopy [27]. This technique enables identification of specific sites and patterns of airway collapse, thereby guiding targeted surgical interventions [26]. DISE is particularly useful in children with residual OSA after AT, minimal adenotonsillar hypertrophy, or craniofacial syndromes [28]. Evidence suggests that DISE can influence surgical decision-making and improve outcomes in selected cases [29]. It is generally considered safe when performed by experienced teams [29,30]. Standardized classification systems, such as the VOTE classification, have been developed to characterize sites of obstruction and improve interobserver reliability and treatment planning [28,30,31].

However, most available evidence is derived from single-center observational studies with relatively small pediatric cohorts. The lack of randomized controlled trials limits causal inference regarding the impact of DISE-guided interventions on long-term outcomes. Therefore, although DISE is increasingly used as a diagnostic adjunct, its clinical utility should be interpreted cautiously pending further high-quality evidence.

Risk Stratification and Shared Decision-Making in Complex Pediatric OSA Cases

Effective management of pediatric OSA requires a multidisciplinary approach that incorporates risk stratification and shared decision-making (SDM) to develop individualized care plans. Risk stratification considers fac-

tors such as age, obesity, adenotonsillar hypertrophy, and PSG findings to assess disease severity and guide treatment selection. For example, children aged 1 to 5 years, those with obesity, and those presenting with frequent snoring or excessive daytime sleepiness are at increased risk of moderate-to-severe OSA [32]. Shared decision-making is a collaborative process in which clinicians, patients, and families jointly determine treatment strategies based on clinical evidence, patient values, and preferences. SDM is particularly important in pediatric OSA due to the involvement of multiple stakeholders and the availability of diverse treatment options. Implementation of SDM has been associated with improved treatment adherence and clinical outcomes [33].

Multidisciplinary clinics that integrate otolaryngologists, sleep physicians, pediatricians, dentists, nutritionists, and psychologists facilitate comprehensive evaluation and coordinated care for complex OSA cases [34]. These models support holistic management by addressing anatomical, physiological, behavioral, and psychosocial factors. Effective SDM within multidisciplinary settings requires clear communication, mutual respect, and the use of decision aids to inform families about the risks and benefits of available treatment options [35]. This approach enhances family engagement and aligns treatment decisions with patient-centered goals.

A stepwise multidisciplinary diagnostic algorithm integrating screening tools, clinical assessment, and objective testing is presented in Fig. 1. This evidence-based framework incorporates validated measures such as the PSQ, specialist evaluation, and advanced diagnostic modalities, including PSG, imaging, and DISE. It enables personalized risk stratification and facilitates shared decision-making in treatment planning for pediatric OSA.

Management Strategies for Pediatric OSA

Following risk stratification and confirmatory diagnosis, treatment of pediatric OSA should be individualized based on disease severity, anatomical factors, and comorbidities. As outlined in the diagnostic algorithm (Fig. 1), therapeutic decisions should align with a stepwise evaluation process and patient-specific risk profiles. The following sections summarize surgical and non-surgical management approaches based on current evidence and multidisciplinary consensus.

Surgical Interventions

Indications and Limitations for Adenotonsillectomy (AT)

Adenotonsillectomy (AT) is widely accepted as the first-line surgical treatment for pediatric OSA, particularly in children older than 2 years with adenotonsillar hypertrophy [36]. Indications include moderate-to-severe OSA confirmed by PSG (apnea–hypopnea index [AHI] >5 events/hour) or the presence of significant clinical symptoms, such as habitual snoring, witnessed apneas, daytime sleepiness, behavioral disturbances, or growth impairment [36,37]. However, residual OSA may persist, particularly in children with obesity, craniofacial abnormalities, or neuromuscular disorders, necessitating further evaluation and ongoing management.

Alternative Surgical Approaches and DISE-Guided Strategies

In cases where AT is contraindicated or insufficient, additional surgical interventions may be considered. Intracapsular tonsillotomy involves partial removal of tonsillar tissue while preserving the tonsillar capsule and has been associated with reduced postoperative pain and bleeding compared with total tonsillectomy. However, it carries a risk of tonsillar regrowth and recurrence of OSA [38–40]. Drug-induced sleep endoscopy (DISE) enables dynamic visualization of upper airway collapse during sedation and can guide targeted surgical procedures, such as lingual tonsillectomy or epiglottopexy, in children with residual or complex OSA [29,31]. Preoperative planning using DISE has been associated with improved outcomes in selected pediatric populations.

Nevertheless, the current evidence base for DISE-guided surgery remains limited. Most studies are retrospective or involve small prospective cohorts with heterogeneous patient populations and outcome measures. The lack of large randomized controlled trials limits conclusions regarding its incremental benefit. Therefore, clinicians should weigh the potential advantages of DISE against its cost and logistical requirements.

Non-Surgical and Adjunctive Therapies

Positive Airway Pressure (PAP) Therapy: Initiation and Adherence

PAP therapy, including continuous PAP (CPAP) and bilevel PAP (BiPAP), is an effective non-surgical treatment for pediatric OSA, particularly in children with residual disease, contraindications to surgery, or persistent symptoms following AT. PAP therapy maintains airway patency during sleep by delivering continuous or variable pressure, thereby reducing apneic events and improving sleep quality. Initiation of PAP therapy requires consideration of the child's age, developmental status, and comorbidities. Pressure titration, typically performed during overnight PSG, is necessary to determine optimal therapeutic settings that effectively eliminate obstructive events while minimizing discomfort [41]. Proper mask fitting is essential, as poorly fitted interfaces may result in air leakage, skin irritation, and reduced adherence.

Adherence to PAP therapy in pediatric populations is often suboptimal, with reported average nightly use of 4–5 hours and adherence rates ranging from 25% to 83% [42,43]. Common barriers include discomfort, device noise, social stigma, and lack of immediate symptom relief.

Strategies to improve adherence include desensitization protocols, behavioral interventions (e.g., positive reinforcement and cognitive-behavioral therapy), and active parental involvement. Early education and demonstration of device use can enhance acceptance and cooperation. Regular follow-up and multidisciplinary support are essential for identifying and addressing adherence barriers, optimizing device settings, and reinforcing long-term compliance [44].

Pharmacologic Agents: Nasal Corticosteroids and Leukotriene Receptor Antagonists

Pharmacologic therapy using intranasal corticosteroids (INCS) and leukotriene receptor antagonists (LTRAs), such as montelukast, has been proposed for children with mild-to-moderate OSA, particularly when surgery is not indicated or declined. These treatments target inflammatory processes contributing to upper airway obstruction, including adenotonsillar hypertrophy. INCS (e.g., fluticasone, budesonide, and mometasone) reduce nasal inflammation and adenoidal size, thereby improving airway patency during sleep [45]. Systematic reviews have demonstrated improvements in respiratory symptoms and reductions in adenoidal hypertrophy in children with sleep-disordered breathing [46]. Montelukast exerts anti-inflammatory effects by inhibiting leukotriene-mediated pathways and has been shown to reduce AHI and improve oxygen saturation in pediatric patients [47,48]. Combination therapy with INCS and montelukast may provide additive or synergistic benefits, resulting in greater reductions in AHI compared with monotherapy [45,46,48].

However, current evidence is primarily derived from small, single-center studies with short follow-up durations and heterogeneous populations. These limitations restrict generalizability and highlight the need for larger multicenter randomized controlled trials. Although generally well tolerated, long-term safety data in children remain limited. Therefore, these therapies are typically recommended for short-term use in mild OSA or as adjunctive treatments when surgical options are unsuitable. Regular follow-up is necessary to evaluate efficacy and monitor for adverse effects.

Dental and Orthodontic Interventions

Dental and orthodontic interventions are important components of multidisciplinary management, particularly in children with craniofacial abnormalities contributing to airway obstruction [18]. These approaches aim to modify craniofacial structures to improve airway patency and reduce OSA severity.

Rapid maxillary expansion (RME) increases the transverse dimension of the maxilla by separating the midpalatal suture, thereby enlarging the nasal cavity and reducing airway resistance. Studies have demonstrated that RME can reduce AHI and improve OSA symptoms in children with maxillary constriction [49,50]. Mandibular advancement devices (MADs) reposition the mandible anteriorly during sleep, increasing upper airway space and reducing airway collapse. These devices have shown efficacy in selected pediatric patients, particularly those with retrognathia or mandibular deficiency. Functional appliances, such as the Twin Block, have also been associated with improvements in airway dimensions and sleep-disordered breathing symptoms [51]. Myofunctional therapy involves targeted exercises to strengthen orofacial muscles, improve tongue posture, and promote nasal breathing. As an adjunct to orthodontic treatment, it may enhance treatment stability and reduce OSA symptoms, particularly in children with oral functional abnormalities [52,53].

Despite promising findings, most evidence is derived from observational studies with small sample sizes and limited follow-up. Variability in study design, patient characteristics, and outcome measures further limits interpretation. High-quality multicenter trials are needed to establish standardized protocols and identify optimal candidates for these interventions.

Nutritional and Behavioral Modification Programs

Nutritional and behavioral interventions are essential in the management of pediatric OSA, particularly in overweight and obese children. Excess adipose tissue in the upper airway contributes to airway narrowing and collapsibility during sleep. Weight reduction through dietary modification and behavioral interventions can improve OSA severity and overall health outcomes. A balanced, calorie-appropriate diet rich in fruits, vegetables, whole grains, and lean protein, combined with reduced intake of saturated fats and added sugars, supports weight management. Studies have shown that children receiving dietary counseling following AT demonstrate improved sleep outcomes compared with those without such support [54]. Behavioral interventions, including cognitive-behavioral therapy (CBT), can promote healthy eating habits, increase physical activity, and improve sleep hygiene [55,56]. Family-based behavioral programs, in which caregivers actively participate, have demonstrated greater effectiveness in achieving and maintaining weight loss in children [57]. Comprehensive programs that integrate nutritional education with behavioral strategies are recommended by the American Academy of Pediatrics for managing pediatric obesity and related conditions, including OSA.

Psychological Support and Neurocognitive Monitoring

Pediatric OSA can significantly affect neurocognitive development and psychological well-being. Sleep disruption and intermittent hypoxia may lead to cognitive deficits, behavioral disturbances, and emotional dysregulation. Children with OSA commonly exhibit impairments in attention, executive function, and memory, which may contribute to academic difficulties and social challenges

[58]. They are also at increased risk of mood disorders, including anxiety and depression. Some studies have reported higher scores on anxiety and depression scales in children with OSA, with improvement observed following treatment [59]. Early childhood represents a critical period for brain development, and prolonged sleep disruption may result in persistent neurocognitive consequences [58,60]. Therefore, early diagnosis and intervention are essential. Management strategies include cognitive-behavioral therapy, educational support (e.g., individualized education programs), and active family involvement. Collaboration among healthcare providers, educators, and caregivers is essential to optimize developmental and psychological outcomes.

Combination and Stepwise Management

Residual OSA After Surgery

Residual OSA, defined as persistent disease following AT, occurs in approximately 20%–40% of children and is more common in those with comorbid conditions such as obesity, craniofacial abnormalities, neuromuscular disorders, or Down syndrome [61]. Management requires comprehensive reassessment to identify underlying causes. DISE can provide dynamic evaluation of the upper airway and identify residual sites of obstruction not addressed by AT [61,62].

Depending on findings, additional surgical interventions (e.g., lingual tonsillectomy or epiglottopexy) may be considered. When surgery is not feasible or contraindicated, non-surgical approaches, particularly PAP therapy, play a central role.

Integrated Pathways for Refractory or Complex Cases

Refractory OSA, defined as a clinically significant disease persisting despite multiple interventions (e.g., AT, PAP, orthodontic therapy), represents some of the most challenging cases to manage and often requires highly personalized, multimodal treatment. These more complex cases are best managed through synergistic, multidisciplinary care by pediatric subspecialists, reflecting the multifactorial etiology of OSA. Orthodontic intervention, such as rapid maxillary expansion (RME) and mandibular advancement devices, have shown promising results in managing OSA in patients with craniofacial abnormalities [63]. These interventions aim to address the narrowed upper airway space and reduce obstruction during sleep. One study demonstrated that RME led to marked improvements in polysomnographic parameters in children with residual OSA after AT [63,64]. Weight control is also a critical factor, especially in the obese child, in whom increased adipose tissue volume contributes to airway narrowing. Dietary counseling and behavior modification are fundamental interventions to achieve and maintain a normal weight, which alone can alleviate the severity of OSA. In children with syndromic conditions or neuromuscular disorders, specific interventions are required. For example, children with Down syndrome may be treated with a combination of surgical interventions, PAP therapy, and myofunctional therapy to address hypotonia and airway obstructions [64].

Importantly, these individualized treatment plans mirror the stepwise approach described in Fig. 1, in which diagnostic stratification guides the selection of surgical, non-surgical, and adjuvant interventions. Intentional alignment of management strategies with the diagnostic algorithm establishes continuity from evaluation to therapy in pediatric OSA care.

These individualized treatment pathways underscore the need for a structured, tiered management approach to pediatric OSA. Table 1 summarizes a stepwise framework that integrates severity stratification, therapeutic options, and multidisciplinary roles across increasingly complex clinical scenarios. This framework is intended to support, but not replace, clinical decision-making. Management strategies should always be tailored to the individual child's disease severity, comorbidities, and findings from multidisciplinary assessment. It also highlights the critical role of long-term follow-up in sustaining treatment success and monitoring developmental outcomes.

Post-Treatment Monitoring and Long-Term Follow-Up

Effective long-term management of pediatric OSA requires a structured follow-up program to monitor postoperative recovery, detect recurrence, and assess neurocognitive and cardiometabolic outcomes. Multidisciplinary follow-up facilitates early identification of persistent disease and enables timely adjustment of treatment strategies based on individual risk factors and developmental trajectory. Table 2 outlines a suggested timeline for coordinated evaluations across different stages of care.

Early Postoperative Care

Adenotonsillectomy (AT) is the first-line treatment for pediatric OSA; however, postoperative respiratory complications (PORCs) may occur, particularly in high-risk populations [65,66]. High-risk groups include children younger than 3 years, those with obesity, craniofacial abnormalities, or severe OSA. These patients require close monitoring due to an increased risk of respiratory compromise. Postoperative care should include continuous pulse oximetry and clinical observation for signs of respiratory distress [67,68]. Adequate pain control and hydration are essential components of early postoperative management. Caregiver education regarding expected recovery and warning signs is critical to ensure timely recognition of complications. Decisions regarding hospitalization should be based on individual risk factors and intraoperative findings. Children with severe OSA (e.g., AHI $\geq$10 events/hour), significant

Table 1. Stepwise and multimodal management strategies for pediatric obstructive sleep apnea.

Step	Clinical scenario	Intervention options	Multidisciplinary involvement
1	Mild OSA without anatomical risk factors	Intranasal corticosteroids ± montelukast	Pediatrician, Allergist
2	Moderate-to-severe OSA with adenotonsillar hypertrophy	AT	ENT Surgeon
3	Post-AT residual OSA	PAP therapy, DISE-guided surgery	Sleep Specialist, ENT
4	Craniofacial abnormalities	Orthodontic interventions (RME, MAD), myofunctional therapy	Pediatric Dentist, Orthodontist
5	Obesity-associated OSA	Nutritional therapy, behavioral modification, emerging pharmacotherapy	Dietitian, Psychologist, Endocrinologist
6	Syndromic or neuromuscular OSA	Tailored multi-modal management including NIV, surgery, behavioral/educational support	Neurologist, Pulmonologist, Behavioral Therapist
7	Long-term management	Cognitive, metabolic, and growth surveillance; Telehealth/remote monitoring	Full Multidisciplinary Team (MDT)

This table outlines a stepwise, multidisciplinary algorithm for the management of pediatric OSA across a range of clinical contexts. Interventions are tiered according to disease complexity and severity, ranging from pharmacotherapy for mild cases to syndromic and neuromuscular manifestations requiring multimodal management. The final step emphasizes the importance of longitudinal surveillance (e.g., neurocognitive, cardiometabolic, and growth outcomes). AT, Adenotonsillectomy; ENT, Otolaryngologist; DISE, Drug-Induced Sleep Endoscopy; MAD, Mandibular Advancement Device; MDT, Multidisciplinary Team; NIV, noninvasive ventilation; OSA, obstructive sleep apnea; RME, rapid maxillary expansion.

Table 2. Suggested follow-up timetable in multidisciplinary clinics.

Timepoint	Evaluations/Interventions	MDT involved
Diagnosis (Baseline)	PSG, PSQ, ENT & Dental exam, Growth and behavior assessment	Pediatrician, ENT, Dentist, Sleep Medicine Physician
Post-Treatment (1 month)	Surgical recovery, PAP adherence check, symptom reevaluation	ENT, Sleep Medicine Physician, Nurse
3 6 months	PSG if residual symptoms, orthodontic initiation, behavioral feedback	Sleep Medicine Physician, Dentist, Behavioral Therapist
12 months	Cognitive/neuropsych screening, growth tracking, repeat PSG if needed	Neurologist, Pediatrician, Psychologist
Annually	Metabolic labs, growth markers, recurrence risk assessment	Endocrinologist, Pediatrician
As Needed	DISE, imaging, therapy adjustments, telemedicine review	Full MDT

This table summarizes a proposed multidisciplinary follow-up schedule for infants with OSA. It shows key evaluation milestones, clinical goals, and recommended specialist input in supporting long-term monitoring and individualized care planning. DISE, Drug-Induced Sleep Endoscopy; ENT, Otolaryngologist; MDT, Multidisciplinary Team; PAP, Positive Airway Pressure; PSQ, Pediatric Sleep Questionnaire; PSG, Polysomnography.

comorbidities, or perioperative complications may benefit from overnight monitoring in a controlled setting [69].

Monitoring Residual/Recurrent Symptoms

Despite AT, residual or recurrent OSA is common, particularly in children with obesity, craniofacial abnormalities, or severe preoperative disease. Residual OSA has been reported in up to 38% of children one year after AT, with higher prevalence in those with elevated body mass index (BMI) and malocclusion [70]. Follow-up PSG is the preferred method for evaluating residual disease. Clinical guidelines recommend repeat PSG in children with persistent symptoms, especially those with moderate-to-severe preoperative OSA or significant risk factors [71]. Early detection of residual OSA allows for timely intervention and optimization of treatment strategies. Management should be individualized based on patient characteristics and comorbidities. Ongoing clinical surveillance is essential to assess treatment effectiveness and guide adjustments in care.

Long-Term Monitoring: Growth, Cognition, and Cardiometabolic Health

Children with OSA require long-term follow-up to monitor growth, neurocognitive development, and cardiometabolic health. Early identification of abnormalities allows for prompt intervention and improved long-term outcomes.

Growth monitoring: OSA has been associated with impaired growth, potentially due to disrupted sleep and altered growth hormone secretion [5]. Regular assessment of height, weight, and BMI is necessary to identify deviations from expected growth trajectories [72].

Neurocognitive development: Children with OSA may exhibit deficits in attention, memory, and executive function, which can persist even after treatment, including AT [73]. Periodic neurocognitive and behavioral assessments are recommended to detect ongoing or emerging impairments.

Cardiometabolic health: OSA is associated with an increased risk of hypertension and metabolic dysfunction. Regular evaluation of blood pressure, lipid profiles, and glucose levels is recommended to identify early signs of cardiometabolic complications.

Children with persistent or untreated symptoms require continued monitoring to prevent long-term adverse outcomes [5,72].

Role of Telemedicine and Digital Tools

Telemedicine and digital health technologies have significantly expanded the capacity to manage pediatric OSA by improving access to care, enabling continuous monitoring, and supporting individualized treatment strategies.

Telemedicine facilitates remote consultations, symptom monitoring, and treatment adjustments without requiring in-person visits. This approach is particularly beneficial for families in rural or underserved areas, reducing travel burden and associated costs. It also enables delivery of educational interventions and behavioral support to improve treatment adherence [74]. Digital tools, including wearable devices and mobile health applications, enable real-time monitoring of physiological parameters and treatment adherence. For example, remote monitoring systems can track CPAP usage and provide feedback to clinicians, allowing timely intervention when adherence declines. These technologies have been associated with improved compliance in pediatric populations [75]. Artificial intelligence and machine learning algorithms further enhance telemedicine capabilities by analyzing large datasets to identify patterns, predict treatment responses, and support personalized care planning [76]. These approaches may improve risk stratification and resource allocation.

Despite these advantages, limitations include the inability to perform comprehensive physical examinations, technological barriers for some families, and concerns regarding data security and privacy. A hybrid model integrating telemedicine with in-person care may help address these challenges and optimize clinical outcomes.

Considerations in Special Populations

Children With Syndromes (e.g., Down Syndrome, Prader-Willi Syndrome)

Children with genetic syndromes, including Down syndrome and Prader–Willi syndrome, have a substantially higher prevalence of OSA compared with the general pediatric population due to a combination of anatomical, neuromuscular, and metabolic factors.

In children with Down syndrome, the prevalence of OSA ranges from 50% to 76%, significantly higher than in the general pediatric population. Contributing factors include craniofacial abnormalities (e.g., midface hypoplasia and macroglossia), generalized hypotonia, obesity, and adenotonsillar hypertrophy [77]. Early screening is essential, as OSA in this population is associated with adverse neurocognitive and cardiovascular outcomes. PSG is recommended for all children with Down syndrome by 4 years of age, regardless of symptoms [77]. AT is typically the first-line treatment; however, residual OSA is common due to persistent anatomical and neuromuscular factors. PAP therapy is frequently required, although adherence may be challenging. Emerging therapies, such as hypoglossal nerve stimulation, are under investigation.

Prader–Willi syndrome is characterized by hypotonia, obesity, and craniofacial abnormalities, all of which contribute to a high prevalence of OSA, reported in up to 80% of patients. The phenotype of sleep-disordered breathing evolves over time, with central sleep apnea predominating in infancy and OSA becoming more prevalent in later childhood [78]. Growth hormone (GH) therapy, commonly used to improve growth and body composition, has com-

plex effects on OSA. Some studies suggest that GH therapy improves ventilatory drive and reduces adiposity, whereas others report worsening of OSA or increased risk of adverse events, particularly in untreated or severe cases [79,80]. Therefore, baseline PSG is recommended prior to initiation of GH therapy, with follow-up assessments to monitor for changes in respiratory status [81]. Management should be individualized, balancing potential benefits with risks. AT may provide partial improvement, but residual OSA is common, and PAP therapy is often required [82]. Multidisciplinary care involving endocrinologists, sleep specialists, and behavioral therapists is essential.

Neuromuscular Disorders and Severe Obesity

Children with neuromuscular disorders (NMDs) and severe obesity represent high-risk populations with distinct pathophysiological mechanisms and management challenges.

In NMDs such as Duchenne muscular dystrophy and spinal muscular atrophy, reduced muscle tone and impaired ventilatory control predispose to airway collapse and hypoventilation during sleep [83,84]. Clinical symptoms may be subtle or overlap with underlying neuromuscular disease, making PSG essential for diagnosis. Management typically includes non-invasive ventilation (NIV), such as BiPAP, to support nocturnal respiration [3,83]. Close monitoring is required due to the progressive nature of these conditions and the risk of respiratory deterioration.

Severe pediatric obesity is strongly associated with OSA, with prevalence rates reported as high as 60% in some cohorts [85]. Excess adipose tissue in the upper airway and neck contributes to airway narrowing and increased collapsibility [86]. Obesity-related inflammation and reduced lung volumes further exacerbate respiratory dysfunction. Management includes weight reduction through lifestyle modification, pharmacologic therapy, and, in selected cases, bariatric surgery. PAP therapy is effective but often limited by adherence challenges [87]. Emerging therapies, such as glucagon-like peptide-1 (GLP-1) receptor agonists (e.g., tirzepatide), have shown promise in reducing OSA severity in adults through weight loss; however, their use in pediatric populations remains investigational and should be limited to controlled clinical settings [88].

Multidisciplinary care involving pediatricians, pulmonologists, sleep specialists, dietitians, and rehabilitation professionals is essential to address the complex needs of these patients.

Tailoring Diagnostic Thresholds and Treatment Plans

Pediatric OSA requires individualized diagnostic and therapeutic approaches that account for developmental stage, comorbidities, and anatomical variability. PSG remains the primary diagnostic tool, with OSA severity classified based on the apnea–hypopnea index (AHI). In children, an AHI $\geq$1 event/hour is considered abnormal, with thresholds commonly defined as mild (1–4.9 events/hour), moderate (5–9.9 events/hour), and severe ($\geq$10 events/hour) [38]. However, these cutting points might not represent the clinical relevance in various pediatric populations.

For example, those with craniofacial deformity or neuromuscular disease can present clinically with severe presentation but comparatively less severe AHI. In such cases, additional parameters—including oxygen desaturation, arousal index, and carbon dioxide retention—should be considered [2]. In addition, behavioral information and quality-of-life assessment can report on the functional consequence of OSA, having greater effect on decision-making than PSG information [89].

In addition, standard AHI cut points may not be equally applicable across different age groups and comorbidities. Children may develop significant neurocognitive or cardiovascular consequences even at relatively low AHI scores, whereas adolescents with obesity may require higher cut points to identify clinically significant disease. Similarly, children with Down syndrome, Prader-Willi syndrome, or neuromuscular disorders may experience disproportionate morbidity even with only mild-to-moderate elevation in AHI. These nuances highlight the importance of context-dependent interpretation of AHI thresholds rather than their uniform application across the entire pediatric population.

Treatment programs should be tailored to age, severity of OSA, and comorbidities. AT remains the first-line treatment for the majority of children with adenotonsillar hypertrophy. For those in whom surgery is not indicated or who have persistent OSA following surgery, other forms of therapy, such as CPAP, anti-inflammatory medications, or orthodontic treatment, may be indicated [3]. Reassessments and follow-ups are necessary to evaluate treatment effectiveness and to adjust management protocols accordingly.

Challenges in Resource-Limited Settings

Management of pediatric OSA in resource-limited settings is particularly challenging due to constraints in diagnostic capacity, trained personnel, and therapeutic resources. Innovative and context-specific strategies are required to address these limitations.

Simplified Screening Protocols

In the absence of polysomnography (PSG), alternative screening approaches have been explored. Nocturnal pulse oximetry combined with pulse rate variability analysis has shown promise in identifying children at risk of moderate-to-severe OSA, enabling home-based assessment and improved resource utilization [90]. Addition-

ally, machine learning–based algorithms applied to non-invasive clinical data have been proposed to enhance diagnostic accuracy, with some studies demonstrating improved performance compared with traditional questionnaires [91]. The CHASE-OSA score, a simplified predictive model, has demonstrated 86% sensitivity and 70% specificity for detecting moderate-to-severe pediatric OSA [92].

Community-Based Task-Sharing Models

Task-sharing strategies, which involve redistributing healthcare responsibilities among trained non-specialist workers, have been proposed to address workforce shortages [93]. Community-based models, in which trained personnel perform initial screening and referral, can expand access to care. Frameworks such as COATS (Concepts and Opportunities to Advance Task Shifting and Task Sharing) provide structured approaches for implementing these models across diverse healthcare settings [93].

Low-Cost PAP Solutions

PAP therapy remains a cornerstone in the management of pediatric OSA, particularly for children who are not candidates for surgery or who have persistent disease following surgical intervention [44,94]. However, the cost and maintenance requirements of conventional PAP devices limit their use in resource-constrained settings. Emerging solutions, including auto-titrating PAP devices and simplified interfaces, aim to reduce costs and improve usability. Community-based education and support programs have also been shown to improve adherence to PAP therapy.

Beyond device-related challenges, barriers such as limited access to PSG, shortages of trained providers, and uneven geographic distribution of sleep centers further restrict timely diagnosis and treatment. In addition, multidisciplinary care often requires infrastructure that may not be available in low-resource settings. Addressing these challenges will require innovative care models, targeted funding, and health policy initiatives to improve equitable access to diagnosis and treatment.

Future Perspectives

Advances in the understanding of pediatric OSA pathophysiology and management are enabling more integrated, technology-driven, and patient-centered models of care. This section highlights emerging strategies and innovations with the potential to improve diagnosis and treatment.

Multidisciplinary Clinic Models and Care Navigation

The multifactorial nature of pediatric OSA, involving anatomical, neurological, and behavioral components, supports the need for interdisciplinary care models. Multidisciplinary clinics that integrate pediatricians, otolaryngologists, sleep specialists, orthodontists, and other healthcare professionals have been associated with improved diagnostic accuracy, more efficient treatment planning, and better clinical outcomes. These models facilitate comprehensive evaluation and coordinated management, reducing delays in diagnosis and treatment initiation. Multidisciplinary sleep centers have been particularly effective in managing complex pediatric OSA cases and improving treatment adherence and caregiver satisfaction.

AI-Assisted Diagnostics, Biomarkers, and Precision Care

Artificial intelligence (AI) and machine learning technologies are transforming the diagnosis and management of pediatric OSA. AI-enabled wearable and imaging devices allow for non-invasive, rapid, and potentially accurate assessment of sleep patterns and airway obstruction. For example, wearable devices such as the Belun Ring have been developed to assist in OSA detection and sleep stage analysis, offering a more accessible alternative to traditional PSG in selected contexts. In addition, biomarker discovery supported by AI analysis is contributing to the development of precision medicine approaches in pediatric OSA [95]. These biomarkers may enable patient stratification based on disease severity and predicted treatment response, facilitating individualized therapy. Overall, AI applications have the potential to improve diagnostic efficiency, enable earlier intervention, and reduce long-term complications associated with pediatric OSA [95].

Current Evidence Gaps and Multicenter Research Needs

Despite these advances, significant gaps remain in the evidence base for pediatric OSA management. Large-scale, multicenter studies are needed to evaluate the long-term safety and efficacy of emerging diagnostic technologies and therapeutic interventions. Comparative studies assessing different surgical approaches, including lingual tonsillectomy and craniofacial procedures, are particularly needed in diverse pediatric populations. In addition, further research is required to clarify the impact of weight reduction and behavioral interventions on long-term OSA outcomes.

Longitudinal studies are essential to evaluate neurocognitive, developmental, and cardiometabolic outcomes into adolescence and adulthood. Moreover, while AI-assisted diagnostics and biomarker-driven precision medicine are promising, rigorous validation in pediatric populations is necessary before widespread clinical implementation. Addressing these gaps through collaborative, interdisciplinary research will be critical to advancing evidence-based care in pediatric OSA.

Conclusion

Pediatric OSA is a multifactorial condition that requires timely diagnosis, individualized treatment, and long-

term follow-up. Effective management depends on a multidisciplinary approach that integrates surgical, medical, orthodontic, and behavioral strategies to address the diverse contributors to airway obstruction. Advances in diagnostic techniques, including imaging and sleep studies, together with evolving therapeutic options, are enabling more personalized care. Long-term follow-up is essential to ensure sustained treatment efficacy, optimize developmental outcomes, and manage residual or recurrent disease.

Future progress in pediatric OSA management will depend on high-quality multicenter research, validation of emerging technologies, and the development of innovative care models that address challenges related to cost, access, and care coordination. Strengthening collaboration across specialties and healthcare systems will be key to delivering equitable and sustainable care for children with OSA.

Availability of Data and Materials

No datasets were generated or analysed during the current study as it is a review article. All materials cited are available from the referenced publications. Reasonable requests for supporting information can be directed to the corresponding author.

Author Contributions

JH conceived the idea, supervised the project, and critically revised the manuscript for important intellectual content. LN contributed to the analysis and interpretation of the literature, and wrote the original draft. HL, TZ, LZ, YL, and ZC contributed to literature review and important manuscript revision. All authors reviewed and approved the final manuscript. All authors have participated sufficiently in the work to take public responsibility for appropriate portions of the content and agreed to be accountable for all aspects of the work in ensuring that questions related to its accuracy or integrity.

Ethics Approval and Consent to Participate

Not applicable.

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Conflict of Interest

The authors declare no conflict of interest.

References

[1] Yang Q, Huang X, Lin Y, Chen K, Lu Q, Lin W, *et al*. Exploring the Multifaceted Landscape of Pediatric Obstructive Sleep Apnea: Insights into Prevalence, Severity, and Coexisting Conditions. Nature and Science of Sleep. 2024; 16: 359–368. https://doi.org/10.2147/NSS.S452221.

[2] Bitners AC, Arens R. Evaluation and Management of Children with Obstructive Sleep Apnea Syndrome. Lung. 2020; 198: 257–270. https://doi.org/10.1007/s00408-020-00342-5.

[3] Marcus CL, Brooks LJ, Draper KA, Gozal D, Halbower AC, Jones J, *et al*. Diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics. 2012; 130: 576–584. https://doi.org/10.1542/peds.2012-1671.

[4] Bhatt A, St-Laurent A, Graham ME. Pediatric obstructive sleep apnea. CMAJ: Canadian Medical Association Journal. 2024; 196: E241. https://doi.org/10.1503/cmaj.230897.

[5] Kim JS, Choi JH. Pediatric obstructive sleep apnea: clinical manifestations and consequences. Korean Journal of Otorhinolaryngology-Head and Neck Surgery. 2024; 67: 515–524. https://doi.org/10.3342/kjorl-hns.2024.00374.

[6] Gupta S, Sharma R. Pediatric Obstructive Sleep Apnea: Diagnostic Challenges and Management Strategies. Cureus. 2024; 16: e75347. https://doi.org/10.7759/cureus.75347.

[7] Chang SJ, Chae KY. Obstructive sleep apnea syndrome in children: Epidemiology, pathophysiology, diagnosis and sequelae. Korean Journal of Pediatrics. 2010; 53: 863–871. https://doi.org/10.3345/kjp.2010.53.10.863.

[8] Hasuneh MM, Toubasi AA, Khraisat B, Aldabbas H, Al-Iede M. Risk Factors of Obstructive Sleep Apnea (OSA) in Pediatric Patients: A Systematic Review and Meta-analysis. Journal of Pediatric Health Care: Official Publication of National Association of Pediatric Nurse Associates & Practitioners. 2024; 38: 717–726. https://doi.org/10.1016/j.pedhc.2024.05.008.

[9] Gulotta G, Iannella G, Vicini C, Polimeni A, Greco A, de Vincentiis M, *et al*. Risk Factors for Obstructive Sleep Apnea Syndrome in Children: State of the Art. International Journal of Environmental Research and Public Health. 2019; 16: 3235. https://doi.org/10.3390/ijerph16183235.

[10] Arens R, Sin S, Wootton DM. Pathophysiology of obstructive sleep apnea syndrome in childhood. In Gozal D, Kheirandish-Gozal L, (eds.) Pediatric Sleep Medicine (pp. 437–457). Springer: Cham. 2021. https://doi.org/10.1007/978-3-030-65574-7_35.

[11] Saint-Fleur AL, Christophides A, Gummalla P, Kier C. Much Ado about Sleep: Current Concepts on Mechanisms and Predisposition to Pediatric Obstructive Sleep Apnea. Children (Basel, Switzerland). 2021; 8: 1032. https://doi.org/10.3390/children8111032.

[12] American Thoracic Society. Paediatric Sleep Questionnaire (PSQ). Available at: https://site.thoracic.org/assemblies/srn/sleep-related-questionnaires/psq (Accessed: 2 July 2025).

[13] Umano GR, Rondinelli G, Luciano M, Pennarella A, Aiello F, Mangoni di Santo Stefano GSRC, *et al*. Pediatric Sleep Questionnaire Predicts Moderate-to-Severe Obstructive Sleep Apnea in Children and Adolescents with Obesity. Children (Basel, Switzerland). 2022; 9: 1303. https://doi.org/10.3390/children9091303.

[14] Shahid A, Wilkinson K, Marcu S, Shapiro CM. Pediatric Sleep Questionnaire (PSQ). In Shahid A, Wilkinson K, Marcu S, Shapiro CM, (eds.) STOP, THAT and One Hundred Other Sleep

Scales (pp. 259–272). Springer: New York, NY. 2011. https://doi.org/10.1007/978-1-4419-9893-4_63.

[15] Kang M, Mo F, Witmans M, Santiago V, Tablizo MA. Trends in Diagnosing Obstructive Sleep Apnea in Pediatrics. Children (Basel, Switzerland). 2022; 9: 306. https://doi.org/10.3390/children9030306.

[16] Jadhav U, Bhanushali J, Sindhu A, Reddy BSK, Toshniwal A, Rashmika M. A Comprehensive Review of Pediatric Obstructive Sleep Apnea: From Assessment to Intervention. Cureus. 2025; 17: e78051. https://doi.org/10.7759/cureus.78051.

[17] Günel C, Başal Y, Sih T. Obstructive sleep apnea in children: ENT perspective. In Cingi C, Arısoy ES, Bayar Muluk N, (eds.) Pediatric ENT Infections (pp. 907–919). Springer: Cham. 2022. https://doi.org/10.1007/978-3-030-80691-0_75.

[18] Mallappa JS, Arya R, Jojo A, Saisree AV, Vishal P, Pai KP. Obstructive sleep apnea: a multidisciplinary challenge—the roles of pediatric dentists, orthodontists, and prosthodontists in diagnosis and management: a comprehensive review. International Journal of Applied Dental Sciences. 2025; 11: 285–291. https://doi.org/10.22271/oral.2025.v11.i2d.2167.

[19] Oceja E, Rodríguez P, Jurado MJ, Luz Alonso M, Del Río G, Villar MÁ, *et al*. Validity and Cost-Effectiveness of Pediatric Home Respiratory Polygraphy for the Diagnosis of Obstructive Sleep Apnea in Children: Rationale, Study Design, and Methodology. Methods and Protocols. 2021; 4: 9. https://doi.org/10.3390/mps4010009.

[20] Kissow Lildal T, Boudewyns A, Kamperis K, Rittig S, Bertelsen JB, Otto M, *et al*. Validity of in-lab and home respiratory polygraphy for detecting obstructive sleep apnea in children. Sleep Medicine. 2023; 103: 195–203. https://doi.org/10.1016/j.sleep.2023.01.016.

[21] Touil Y, Schweitzer C, Renard E, Maréchal M, Da Mota S, Franco P, *et al*. Evaluating the added-value of video recording in respiratory polygraphy for the diagnosis of obstructive sleep apnea in children. Sleep Medicine. 2025; 125: 177–184. https://doi.org/10.1016/j.sleep.2024.11.040.

[22] Tan HL, Gozal D, Ramirez HM, Bandla HPR, Kheirandish-Gozal L. Overnight polysomnography versus respiratory polygraphy in the diagnosis of pediatric obstructive sleep apnea. Sleep. 2014; 37: 255–260. https://doi.org/10.5665/sleep.3392.

[23] Maggio ABR, Beghetti M, Cao Van H, Grasset Salomon C, Barazzone-Argiroffo C, Corbelli R. Home respiratory polygraphy in obstructive sleep apnea syndrome in children: Comparison with a screening questionnaire. International Journal of Pediatric Otorhinolaryngology. 2021; 143: 110635. https://doi.org/10.1016/j.ijporl.2021.110635.

[24] Slaats MA, Van Hoorenbeeck K, Van Eyck A, Vos WG, De Backer JW, Boudewyns A, *et al*. Upper airway imaging in pediatric obstructive sleep apnea syndrome. Sleep Medicine Reviews. 2015; 21: 59–71. https://doi.org/10.1016/j.smrv.2014.08.001.

[25] Patel NB, Bitners AC, Sin S, Arens R. Imaging upper airway obstruction in obstructive sleep apnea. In Isaiah A, Mitchell RB, (eds.) Snoring and Obstructive Sleep Apnea in Children (pp. 165–203). Academic Press: Cambridge, MA. 2024. https://doi.org/10.1016/B978-0-323-99653-2.00013-4.

[26] Iannella G, Magliulo G, Greco A, De Virgilio A, Maniaci A, Lechien JR, *et al*. Clinical Application of Pediatric Sleep Endoscopy: An International Survey. Children (Basel, Switzerland). 2024; 11: 94. https://doi.org/10.3390/children11010094.

[27] Jindal P, Chakrabarty B. Drug Induced Sleep Endoscopy (DISE): An Upcoming Modality for Obstructive Sleep Disordered Breathing in Infants. Indian Journal of Pediatrics. 2024; 91: 1117–1118. https://doi.org/10.1007/s12098-024-05222-x.

[28] Williamson A, 4th, Ibrahim SR, Coutras SW, Carr MM. Pediatric Drug-Induced Sleep Endoscopy: Technique and Scoring System. Cureus. 2020; 12: e10765. https://doi.org/10.7759/cureus.10765.

[29] Wang X, Chen YC, Li L, Pan HG, Teng YS. Effects of drug-induced sleep endoscopy in children with conventional obstructive sleep apnea-hypopnea syndrome: a systematic review and meta-analysis. Sleep & Breathing. 2024; 28: 935–944. https://doi.org/10.1007/s11325-023-02945-7.

[30] Wilcox LJ, Bergeron M, Reghunathan S, Ishman SL. An updated review of pediatric drug-induced sleep endoscopy. Laryngoscope Investigative Otolaryngology. 2017; 2: 423–431. https://doi.org/10.1002/lio2.118.

[31] Arganbright JM, Lee JC, Weatherly RA. Pediatric drug-induced sleep endoscopy: An updated review of the literature. World Journal of Otorhinolaryngology - Head and Neck Surgery. 2021; 7: 221–227. https://doi.org/10.1016/j.wjorl.2021.05.002.

[32] Unchiti K, Samerchua A, Pipanmekaporn T, Leurcharusmee P, Sonsuwan N, Phinyo P, *et al*. Clinical predictors of moderate-to-severe pediatric obstructive sleep apnea. Frontiers in Pediatrics. 2024; 12: 1421467. https://doi.org/10.3389/fped.2024.1421467.

[33] Eaton SM, Clark JD, Cummings CL, Kon AA, Morrison W, Feudtner C, *et al*. Pediatric Shared Decision-Making for Simple and Complex Decisions: Findings From a Delphi Panel. Pediatrics. 2022; 150: e2022057978. https://doi.org/10.1542/peds.2022-057978.

[34] DeVries JK, Nation JJ, Nardone ZB, Lance SH, Stauffer JA, Abichaker GM, *et al*. Multidisciplinary clinic for care of children with complex obstructive sleep apnea. International Journal of Pediatric Otorhinolaryngology. 2020; 138: 110384. https://doi.org/10.1016/j.ijporl.2020.110384.

[35] Jacobs S, Davies N, Butterick KL, Oswell JL, Siapka K, Smith CH. Shared decision-making for children with medical complexity in community health services: a scoping review. BMJ Paediatrics Open. 2023; 7: e001866. https://doi.org/10.1136/bmjpo-2023-001866.

[36] Sarber KM, Ishman SL. Surgical treatment of pediatric obstructive sleep apnea. In Gozal D, Kheirandish-Gozal L, (eds.) Pediatric Sleep Medicine (pp. 465–475). Springer: Cham. 2021. https://doi.org/10.1007/978-3-030-65574-7_37.

[37] Todd CA, Bareiss AK, McCoul ED, Rodriguez KH. Adenotonsillectomy for Obstructive Sleep Apnea and Quality of Life: Systematic Review and Meta-analysis. Otolaryngology–head and Neck Surgery: Official Journal of American Academy of Otolaryngology-Head and Neck Surgery. 2017; 157: 767–773. https://doi.org/10.1177/0194599817717480.

[38] Mitchell RB, Archer SM, Ishman SL, Rosenfeld RM, Coles S, Finestone SA, *et al*. Clinical Practice Guideline: Tonsillectomy in Children (Update). Otolaryngology–head and Neck Surgery: Official Journal of American Academy of Otolaryngology-Head and Neck Surgery. 2019; 160: S1–S42. https://doi.org/10.1177/0194599818801757.

[39] Ordemann AG, Mitchell RB. Surgical management of OSA: adenotonsillectomy. In Kim KB, Movahed R, Malhotra RK, Stanley JJ, (eds.) Management of Obstructive Sleep Apnea (pp. 225–241). Springer: Cham. 2021. https://doi.org/10.1007/978-3-030-54146-0_15.

[40] Sjölander I, Borgström A, Nerfeldt P, Friberg D. Adenotonsillotomy versus adenotonsillectomy in pediatric obstructive sleep apnea: A 5-year RCT. Sleep Medicine: X. 2022; 4: 100055. https://doi.org/10.1016/j.sleepx.2022.100055.

[41] Tovichien P, Kulbun A, Udomittipong K. Comparing Adherence of Continuous and Automatic Positive Airway Pressure (CPAP and APAP) in Obstructive Sleep Apnea (OSA) Children. Frontiers in Pediatrics. 2022; 10: 841705. https://doi.org/10.3389/fped.2022.841705.

[42] Watach AJ, Xanthopoulos MS, Afolabi-Brown O, Saconi B, Fox

KA, Qiu M, *et al*. Positive airway pressure adherence in pediatric obstructive sleep apnea: A systematic scoping review. Sleep Medicine Reviews. 2020; 51: 101273. https://doi.org/10.1016/j.smrv.2020.101273.

[43] Bhattacharjee R, Benjafield AV, Armitstead J, Cistulli PA, Nunez CM, Pepin JLD, *et al*. Adherence in children using positive airway pressure therapy: a big-data analysis. The Lancet. Digital Health. 2020; 2: e94–e101. https://doi.org/10.1016/S2589-7500(19)30214-6.

[44] Bitners AC, Arens R. PAP management and adherence for children and adolescents with OSAS. In Shapiro CM, Gupta M, Zalai D, (eds.) CPAP Adherence (pp. 245–254). Springer: Cham. 2022. https://doi.org/10.1007/978-3-030-93146-9_25.

[45] AlTheyab F, Alkhodair A, Albdah A, Alanazi RK, Alkhodair A. The efficacy and safety of intra-nasal corticosteroid spray in pediatric: Systematic review. American Journal of Otolaryngology. 2024; 45: 104355. https://doi.org/10.1016/j.amjoto.2024.104355.

[46] Zaffanello M, Pietrobelli A, Nosetti L, Antoniazzi F, Frassoldati R, Piacentini G. Intranasal Corticosteroids and Oral Montelukast for Paediatric Obstructive Sleep Apnoea: A Systematic Review. Pharmaceutics. 2025; 17: 588. https://doi.org/10.3390/pharmaceutics17050588.

[47] Liming BJ, Ryan M, Mack D, Ahmad I, Camacho M. Montelukast and Nasal Corticosteroids to Treat Pediatric Obstructive Sleep Apnea: A Systematic Review and Meta-analysis. Otolaryngology–head and Neck Surgery: Official Journal of American Academy of Otolaryngology-Head and Neck Surgery. 2019; 160: 594–602. https://doi.org/10.1177/0194599818815683.

[48] Li C, Kou YF, Ishman SL. Pediatric OSA: evidence-based review of treatment results. Current Otorhinolaryngology Reports. 2021; 9: 246–253. https://doi.org/10.1007/s40136-021-00348-4.

[49] Ferati K, Bexheti-Ferati A, Palermo A, Pezzolla C, Trilli I, Sardano R, *et al*. Diagnosis and Orthodontic Treatment of Obstructive Sleep Apnea Syndrome Children-A Systematic Review. Diagnostics (Basel, Switzerland). 2024; 14: 289. https://doi.org/10.3390/diagnostics14030289.

[50] Militi A, Maio A, Nucera R, Bellocchio AM, Fiorillo L, Galletti F, *et al*. Effects of rapid maxillary expansion in pediatric patients affected by obstructive sleep apnea syndrome: a literature review. Minerva Dental and Oral Science. 2025; 74: 114–119. https://doi.org/10.23736/S2724-6329.24.04993-3.

[51] Huynh NT, Desplats E, Almeida FR. Orthodontics treatments for managing obstructive sleep apnea syndrome in children: A systematic review and meta-analysis. Sleep Medicine Reviews. 2016; 25: 84–94. https://doi.org/10.1016/j.smrv.2015.02.002.

[52] Meghpara S, Chohan M, Bandyopadhyay A, Kozlowski C, Casinas J, Kushida C, *et al*. Myofunctional therapy for OSA: a meta-analysis. Expert Review of Respiratory Medicine. 2022; 16: 285–291. https://doi.org/10.1080/17476348.2021.2001332.

[53] Bandyopadhyay A, Kaneshiro K, Camacho M. Effect of myofunctional therapy on children with obstructive sleep apnea: a meta-analysis. Sleep Medicine. 2020; 75: 210–217. https://doi.org/10.1016/j.sleep.2020.08.003.

[54] Chuang HH, Lin RH, Hsu JF, Chuang LP, Li HY, Fang TJ, *et al*. Dietary profile of pediatric obstructive sleep apnea patients, effects of routine educational counseling, and predictors for outcomes. Frontiers in Public Health. 2023; 11: 1160647. https://doi.org/10.3389/fpubh.2023.1160647.

[55] Bejarano CM, Marker AM, Cushing CC. Cognitive-behavioral therapy for pediatric obesity. In Friedberg RD, Paternostro JK, (eds.) Handbook of Cognitive Behavioral Therapy for Pediatric Medical Conditions (pp. 369–383). Springer: Cham. 2019. https://doi.org/10.1007/978-3-030-21683-2_23.

[56] Davison GM, Monocello LT, Lipsey K, Wilfley DE. Evidence Base Update on Behavioral Treatments for Overweight and Obesity in Children and Adolescents. Journal of Clinical Child and Adolescent Psychology: the Official Journal for the Society of Clinical Child and Adolescent Psychology, American Psychological Association, Division 53. 2023; 52: 589–603. https://doi.org/10.1080/15374416.2023.2251164.

[57] Rhee KE, Corbett T, Patel S, Eichen DM, Strong DR, Kang-Sim E, *et al*. Parenting Training Plus Behavioral Treatment for Children With Obesity: A Randomized Clinical Trial. JAMA Network Open. 2025; 8: e258398. https://doi.org/10.1001/jamanetworkopen.2025.8398.

[58] Beebe DW, Wells CT, Jeffries J, Chini B, Kalra M, Amin R. Neuropsychological effects of pediatric obstructive sleep apnea. Journal of the International Neuropsychological Society: JINS. 2004; 10: 962–975. https://doi.org/10.1017/s135561770410708x.

[59] Pereira L, Song E, Stefani CM, Flores-Mir C, De Luca Canto G, Pacheco-Pereira C. Prevalence of depression in pediatric patients with diagnosed or at high risk for obstructive sleep apnea: A systematic review with meta-analysis. Sleep Medicine Reviews. 2025; 80: 102040. https://doi.org/10.1016/j.smrv.2024.102040.

[60] Mathew N, Redline S, Isaiah A. Neurobehavioral outcomes of pediatric obstructive sleep apnea. In Isaiah A, Mitchell RB, (eds.) Snoring and Obstructive Sleep Apnea in Children (pp. 65–81). Academic Press: Cambridge, MA. 2024. https://doi.org/10.1016/B978-0-323-99653-2.00006-7.

[61] Sharma AV, Padhya T, Nallu S. Management of Pediatric Obstructive Sleep Apnea After Failed Tonsillectomy and Adenoidectomy. Advances in Pediatrics. 2022; 69: 95–105. https://doi.org/10.1016/j.yapd.2022.03.006.

[62] Hazkani I, Stein E, Ghadersohi S, Ida J, Thompson DM, Valika T. Epiglottopexy for refractory obstructive sleep apnea in children - A single-institution experience. American Journal of Otolaryngology. 2023; 44: 103798. https://doi.org/10.1016/j.amjoto.2023.103798.

[63] Xia T, Luo R, Wan M. Long-term recurrence-free survival of orthodontic treatments for Class II malocclusion as the important supplement protocol to adenotonsillectomy in children with obstructive sleep apnea syndrome: a case-control retrospective study. Journal of Clinical Pediatric Dentistry. 2025; 49: 178–187. https://doi.org/10.22514/jocpd.2025.037.

[64] Ji H, Leoniak S, Ulualp SO. Management of pediatric OSA beyond adeno-tonsillectomy. Current Sleep Medicine Reports. 2017; 3: 164–172. https://doi.org/10.1007/s40675-017-0083-2.

[65] Locci C, Cenere C, Sotgiu G, Puci MV, Saderi L, Rizzo D, *et al*. Adenotonsillectomy in Children with Obstructive Sleep Apnea Syndrome: Clinical and Functional Outcomes. Journal of Clinical Medicine. 2023; 12: 5826. https://doi.org/10.3390/jcm12185826.

[66] Halawani M, Alsharif A, Alanazi OI, Awad B, Alsharif A, Alahmadi H, *et al*. Efficacy and Safety of Adenotonsillectomy for Pediatric Obstructive Sleep Apnea Across Various Age Groups: A Systematic Review. Pediatric Reports. 2025; 17: 71. https://doi.org/10.3390/pediatric17040071.

[67] Efune PN, Pinales P, Park J, Poppino KF, Mitchell RB, Szmuk P. Pediatric obstructive sleep apnea: a prospective observational study of respiratory events in the immediate recovery period after adenotonsillectomy. Anaesthesia, Critical Care & Pain Medicine. 2024; 43: 101385. https://doi.org/10.1016/j.accpm.2024.101385.

[68] Caetta A, Timashpolsky A, Tominaga SM, D'Souza N, Goldstein NA. Postoperative respiratory complications after adenotonsillectomy in children with obstructive sleep apnea. International Journal of Pediatric Otorhinolaryngology. 2021; 148:

110835. https://doi.org/10.1016/j.ijporl.2021.110835.

[69] Ehsan Z, Ishman SL, Soghier I, Almeida FR, Boudewyns A, Camacho M, *et al*. Management of Persistent, Post-adenotonsillectomy Obstructive Sleep Apnea in Children: An Official American Thoracic Society Clinical Practice Guideline. American Journal of Respiratory and Critical Care Medicine. 2024; 209: 248–261. https://doi.org/10.1164/rccm.202310-1857ST.

[70] Ianniello F, D'Onofrio D, Paolino MC, Castaldo R, Crescenzi A, Tabarrini A, *et al*. Follow-up of children with obstructive sleep apnea syndrome treated with adenotonsillectomy. European Respiratory Journal. 2014; 38. https://doi.org/10.1183/13993003.erj.38.Suppl_55.p2195.

[71] Aurora RN, Zak RS, Karippot A, Lamm CI, Morgenthaler TI, Auerbach SH, *et al*. Practice parameters for the respiratory indications for polysomnography in children. Sleep. 2011; 34: 379–388. https://doi.org/10.1093/sleep/34.3.379.

[72] Caraballo M. Growth and development in pediatric obstructive sleep apnea. In Isaiah A, Mitchell RB, (eds.) Snoring and Obstructive Sleep Apnea in Children (pp. 121–133). Academic Press: Cambridge, MA. 2024. https://doi.org/10.1016/B978-0-323-99653-2.00010-9.

[73] Chandrakantan A, Adler A. Pediatric obstructive sleep apnea: neurocognitive consequences. Current Anesthesiology Reports. 2019; 9: 110–115. https://doi.org/10.1007/s40140-019-00331-2.

[74] Rizzo L, Barbetta E, Ruberti F, Petz M, Tornesello M, Deolmi M, *et al*. The Role of Telemedicine in Children with Obstructive Sleep Apnea Syndrome (OSAS): A Review of the Literature. Journal of Clinical Medicine. 2024; 13: 2108. https://doi.org/10.3390/jcm13072108.

[75] Yang L, Liu D, Zhong J, Huang J, Qiu S. Application of bidirectional telemedicine in home CPAP for children with obstructive sleep apnea syndrome. International Journal of Pediatric Otorhinolaryngology. 2024; 183: 112047. https://doi.org/10.1016/j.ijporl.2024.112047.

[76] Pépin JL, Baillieul S, Bailly S, Tamisier R. New management pathways for follow-up of CPAP-treated sleep apnoea patients including digital medicine and multimodal telemonitoring. Thorax. 2024; 80: 52–61. https://doi.org/10.1136/thorax-2024-221422.

[77] Gastelum E, Cummins M, Singh A, Montoya M, Urbano GL, Tablizo MA. Treatment Considerations for Obstructive Sleep Apnea in Pediatric Down Syndrome. Children (Basel, Switzerland). 2021; 8: 1074. https://doi.org/10.3390/children8111074.

[78] Itani R, Gillett ES, Perez IA. Sleep Consequences of Prader-Willi Syndrome. Current Neurology and Neuroscience Reports. 2023; 23: 25–32. https://doi.org/10.1007/s11910-023-01254-6.

[79] Passone CDGB, Franco RR, Ito SS, Trindade E, Polak M, Damiani D, *et al*. Growth hormone treatment in Prader-Willi syndrome patients: systematic review and meta-analysis. BMJ Paediatrics Open. 2020; 4: e000630. https://doi.org/10.1136/bmjpo-2019-000630.

[80] Sacco M, Di Giorgio G. Sudden death in Prader-Willi syndrome during growth hormone therapy. Hormone Research. 2005; 63: 29–32. https://doi.org/10.1159/000082525.

[81] Duis J, Pullen LC, Picone M, Friedman N, Hawkins S, Sannar E, *et al*. Diagnosis and management of sleep disorders in Prader-Willi syndrome. Journal of Clinical Sleep Medicine: JCSM: Official Publication of the American Academy of Sleep Medicine. 2022; 18: 1687–1696. https://doi.org/10.5664/jcsm.9938.

[82] Lee CH, Hsu WC, Ko JY, Yeh TH, Lin MT, Kang KT. Adenotonsillectomy for the Treatment of Obstructive Sleep Apnea in Children with Prader-Willi Syndrome: A Meta-analysis. Otolaryngology–head and Neck Surgery: Official Journal of American Academy of Otolaryngology-Head and Neck Surgery. 2020; 162: 168–176. https://doi.org/10.1177/0194599819893115.

[83] Kalyoncu MY, Baysal EE, Selçuk M, Karabulut Ş, Çakar NM, Yıldız CA, *et al*. Retrospective analysis of sleep-disordered breathing in pediatric neuromuscular disease. Trends in Pediatrics. 2025; 6: 40–46. https://doi.org/10.59213/TP.2025.209.

[84] Boddepalli R, Govindarajan R. Sleep issues in pediatric neuromuscular disorders. In Govindarajan R, Bollu P, (eds.) Sleep Issues in Neuromuscular Disorders (pp. 101–116). Springer: Cham. 2018. https://doi.org/10.1007/978-3-319-73068-4_7.

[85] Narang I, Mathew JL. Childhood obesity and obstructive sleep apnea. Journal of Nutrition and Metabolism. 2012; 2012: 134202. https://doi.org/10.1155/2012/134202.

[86] Schwartz AR, Patil SP, Laffan AM, Polotsky V, Schneider H, Smith PL. Obesity and obstructive sleep apnea: pathogenic mechanisms and therapeutic approaches. Proceedings of the American Thoracic Society. 2008; 5: 185–192. https://doi.org/10.1513/pats.200708-137MG.

[87] Sawunyavisuth B, Ngamjarus C, Sawanyawisuth K. Adherence to Continuous Positive Airway Pressure Therapy in Pediatric Patients with Obstructive Sleep Apnea: A Meta-Analysis. Therapeutics and Clinical Risk Management. 2023; 19: 143–162. https://doi.org/10.2147/TCRM.S358737.

[88] Hassan MG, Hassan DG, Hassan GA. Tirzepatide gains US Food and Drug Administration approval for the management of obstructive sleep apnea: Implications for oral health care providers. Journal of the American Dental Association (1939). 2025; 156: 620–625. https://doi.org/10.1016/j.adaj.2025.05.007.

[89] Rubinstein BJ, Baldassari CM. An update on the management of pediatric obstructive sleep apnea. Current Treatment Options in Pediatrics. 2015; 1: 211–223. https://doi.org/10.1007/s40746-015-0022-8.

[90] Garde A, Hoppenbrouwer X, Dehkordi P, Zhou G, Rollinson AU, Wensley D, *et al*. Pediatric pulse oximetry-based OSA screening at different thresholds of the apnea-hypopnea index with an expression of uncertainty for inconclusive classifications. Sleep Medicine. 2019; 60: 45–52. https://doi.org/10.1016/j.sleep.2018.08.027.

[91] Qin H, Zhang L, Li X, Xu Z, Zhang J, Wang S, *et al*. Pediatric obstructive sleep apnea diagnosis: leveraging machine learning with linear discriminant analysis. Frontiers in Pediatrics. 2024; 12: 1328209. https://doi.org/10.3389/fped.2024.1328209.

[92] Unchiti K, Samerchua A, Pipanmekaporn T, Leurcharusmee P, Sonsuwan N, Phinyo P, *et al*. Development and validation of a predictive model (CHASE-OSA) for preoperative assessment of moderate-to-severe pediatric obstructive sleep apnea. Sleep & Breathing = Schlaf & Atmung. 2024; 29: 48. https://doi.org/10.1007/s11325-024-03226-7.

[93] Orkin AM, Rao S, Venugopal J, Kithulegoda N, Wegier P, Ritchie SD, *et al*. Conceptual framework for task shifting and task sharing: an international Delphi study. Human Resources for Health. 2021; 19: 61. https://doi.org/10.1186/s12960-021-00605-z.

[94] Dominguez Batista DM, Paruthi S. Positive airway pressure therapy for pediatric obstructive sleep apnea. In Isaiah A, Mitchell RB, (eds.) Snoring and Obstructive Sleep Apnea in Children (pp. 343–353). Academic Press: Cambridge, MA. 2024. https://doi.org/10.1016/B978-0-323-99653-2.00023-7.

[95] Molten A, Abo A. Artificial intelligence in pediatric healthcare: clinical applications. In Adirim T, (ed.) Digital Health, AI and Generative AI in Healthcare (pp. 85–98). Springer: Cham. 2025. https://doi.org/10.1007/978-3-031-83526-1_7.