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Pediatric Obstructive Sleep Apnea: Oral Manifestations, MultiSystemic Consequences, and Clinical Implications

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Abstract
Pediatric obstructive sleep apnea (OSA) is a sleep-related breathing disorder characterized by recurrent upper-airway obstruction and disturbed ventilation during sleep. If untreated, it may affect behavior, learning, cardiovascular health, metabolism, growth and quality of life. Adenotonsillar hypertrophy and obesity are major risk factors, while children with Down syndrome, craniofacial anomalies and neuromuscular disorders represent particularly vulnerable groups. Dental professionals may encounter several associated features, including habitual mouth breathing, a high-arched or narrow palate, maxillary constriction, increased overjet, mandibular retrognathia and vertical facial growth. These findings may support risk recognition but cannot establish a diagnosis. Current craniofacial evidence remains inconsistent, and statistically significant differences may not always be clinically meaningful. Systemic associations include inattention, hyperactivity, poor academic performance, blood pressure dysregulation, metabolic abnormalities, asthma and nocturnal enuresis. A focused dental assessment should combine sleep-related questions with examination of the tonsils, palate, dental arches, occlusion, mandibular position and facial growth pattern. Screening questionnaires may help organize risk assessment, but polysomnography remains the diagnostic standard. Dentists should not independently diagnose or treat pediatric OSA; their primary responsibilities are early recognition, family education, appropriate referral and participation in multidisciplinary follow-up. Timely collaboration among dental professionals, pediatricians, otolaryngologists, sleep specialists and other healthcare providers may reduce the long-term oral and systemic burden of pediatric OSA.

Keywords: Pediatric obstructive sleep apnea; sleep-disordered breathing; pediatric dentistry; craniofacial morphology; malocclusion; systemic comorbidities; dental screening.

I. Introduction

Pediatric obstructive sleep apnea is characterized by prolonged partial upper-airway obstruction, intermittent complete obstruction, or both, leading to disturbed ventilation and disruption of normal sleep.[1]Its reported prevalence ranges from approximately 1.2% to 5.7%, although differences in diagnostic criteria and study populations influence these estimates. Habitual snoring is more common than OSA and was reported in approximately 10% of preschool- and school-aged children, while clinically relevant disease was estimated to affect

2–3%. Snoring alone should therefore not be equated with OSA, but frequent snoring accompanied by breathing pauses, gasping, restless sleep or daytime problems requires further assessment.[2,3]

Pediatric OSA may affect neurobehavioral development, academic performance, cardiovascular regulation, metabolism, growth and quality of life.[4] The disorder is often under recognized because children may present with hyperactivity, inattention or learning difficulties rather than the marked daytime sleepiness commonly observed in adults. Oral and craniofacial features may also be present, including tonsillar enlargement, a high-arched palate, maxillary constriction, mandibular retrognathia and increased vertical facial growth.[5,6]

Dentists and orthodontists routinely examine the oral cavity, occlusion and developing craniofacial complex. They may therefore identify warning signs before a child receives formal sleep evaluation. These findings cannot diagnose OSA but may justify focused questioning and medical referral. This focused narrative review summarizes the pathophysiology, predisposing conditions, oral and craniofacial manifestations, systemic consequences, screening approaches and multidisciplinary management of pediatric obstructive sleep apnea, with particular emphasis on the role of dental professionals in early risk recognition and referral.

Pathophysiology of Pediatric OSA

Pediatric OSA develops through an interaction between anatomical airway narrowing and the physiological mechanisms that maintain

airway patency during sleep. Adenotonsillar hypertrophy, maxillary constriction, mandibular retrognathia, nasal obstruction and enlarged soft tissues may reduce the dimensions of the nasopharyngeal or oropharyngeal airway. Anatomical narrowing alone does not explain why children with similar upper-airway features can have different disease severity.[7,8]

During wakefulness, pharyngeal dilator muscles help stabilize the airway and oppose collapse. Their activity decreases at sleep onset, increasing airway resistance in susceptible children. Obstructive events are particularly common during rapid eye movement sleep, when upper airway muscle tone is reduced and respiratory control becomes more variable. Repeated airway narrowing may produce increased respiratory effort, flow limitation, hypoxemia, hypercapnia and arousal from sleep.[7,9]

Intermittent hypoxia and sleep fragmentation activate sympathetic, oxidative and inflammatory pathways that may contribute to end-organ morbidity. Repeated changes in intrathoracic pressure may place additional stress on the cardiovascular and respiratory systems.[3,10] Disease expression is therefore shaped by anatomy, neuromuscular compensation, sleep stage, ventilatory control, inflammation and individual susceptibility rather than by a single obstructive feature.[7]

Predisposing Conditions and Risk Factors

Adenotonsillar hypertrophy and obesity are the two major risk factors in otherwise healthy children.[2] Enlarged adenoids and tonsils reduce the available airway space and are a common cause of obstruction in younger children. Obesity may further increase airway collapsibility through soft-tissue loading, reduced lung volume and altered respiratory mechanics.[7] Approximately 30–60% of children with obesity may have OSA, although obesity also confounds the relationship between OSA and cardiometabolic disease.[10]

Children with Down syndrome have a markedly increased risk because of midfacial hypoplasia, a small oropharynx, narrow upper airway, relative macroglossia, generalized hypotonia, adenotonsillar hypertrophy, obesity and hypothyroidism. Sleep-disordered breathing was reported in 31–63% of children with Down syndrome, compared with 0.7–13% of the general pediatric population. Clinical variables, including tonsillar size, body mass index and reported symptoms, did not reliably identify affected children in this group.[11]

Craniofacial anomalies and neuromuscular disorders may increase risk by reducing skeletal airway dimensions or weakening the muscles that maintain airway patency.[2,10] Nasal obstruction related to allergic rhinitis, turbinate hypertrophy or septal deviation may also increase upper-airway resistance.[7]

Asthma and sleep-disordered breathing appear to have a bidirectional association. Children with asthma had 1.9-times higher odds of sleep-disordered breathing than children without asthma (OR 1.9; 95% CI: 1.2–2.2). Shared pathways may include upper- and lower-airway inflammation, leukotriene activity, adenotonsillar enlargement, bronchoconstriction and intermittent hypoxia. Poorly controlled asthma may worsen nocturnal hypoxemia, while OSA related inflammation and altered airway reflexes may aggravate asthma symptoms.[12]

Oral and Craniofacial Manifestations

Chronic upper-airway obstruction may encourage habitual mouth breathing and altered mandibular and tongue posture. A mouth-breathing child may hold the mandible and tongue in a lower position to facilitate airflow, potentially modifying the functional environment in which

the facial skeleton and dental arches develop. Reported features include a high-arched or narrow palate, maxillary constriction, mandibular retrognathia and increased lower facial height. This pattern has often been described as a long-face or adenoidal facial appearance.[2,7]

Flores-Mir et al. found significant differences in mandibular plane, SNB and ANB measurements between children with OSA and controls. Their findings suggested a tendency toward a retrusive chin, steep mandibular plane, vertical growth and skeletal Class II relationship. Considerable heterogeneity was present, including I^2 values of 79.53% for the basion–sella-nasion angle, 89.54% for the sella-nasion–palatal plane angle and 96.82% for the mandibular plane–sella-nasion angle.[5]

A meta-analysis of 16 cross-sectional studies provided further evidence of dental and craniofacial differences in children with OSA. Compared with healthy controls, affected children had a mean overjet that was 0.86 mm greater and an ANB angle that was 1.78° greater. The mandibular plane angle was 3.65° greater, whereas the SNB angle was 2.10° lower, suggesting a tendency toward mandibular retroposition and vertical facial growth. Maxillary length was 1.62 mm shorter, while the upper first and second deciduous molar widths were reduced by 1.86 mm and 1.06 mm, respectively.[6]

These results are not fully consistent across reviews. Fagundes et al. identified mandibular

retrognathia, greater lower facial height, narrower intercanine width and reduced nasopharyngeal dimensions in some individual studies. However, their pooled analyses found no significant differences in SNA, SNB, ANB, NSBa, U1–L1 or U1–SN measurements, and the certainty of evidence ranged from very low to moderate. Their stricter eligibility criteria required polysomnography-confirmed OSA and polysomnography-negative controls, which may partly explain the difference from earlier findings.[13]

Statistical differences may also be too small to be recognized reliably during an individual dental examination. Cross-sectional studies cannot determine whether craniofacial characteristics preceded OSA, developed partly through chronic mouth breathing or arose from shared developmental influences.[6,13] Tonsillar hypertrophy, adenoidal facies, high-arched palate, increased overjet, maxillary narrowing, mandibular retrognathia and vertical growth should therefore be treated as risk indicators rather than diagnostic criteria.[2,5] No single malocclusion, facial pattern or cephalometric value can confirm or exclude pediatric OSA.[6,13]

Multisystem Consequences and Comorbidities

Neurobehavioral and psychological effects

Pediatric OSA has been associated with inattention, hyperactivity, aggressive or oppositional behavior, impaired cognition and reduced academic performance. Sleep fragmentation may impair attention, memory, executive function and emotional regulation even when total sleep duration appears adequate. Habitual snoring without confirmed OSA has also been associated with milder behavioral abnormalities, although the clinical significance varies among children.[3,4]

Daytime sleepiness may be difficult to recognize because younger children can present with hyperactivity rather than obvious tiredness. General parental reports identified sleepiness in only 7% of affected children, whereas targeted questionnaires reported symptoms in approximately 40–50%. Objective testing identified excessive daytime sleepiness in approximately 13–20% of children with OSA, with obesity increasing its likelihood.[2,3]

Irritability, depressed mood, reduced interest in daily activities and poorer quality of life have also been described.[3] These manifestations overlap with primary psychiatric and developmental conditions. OSA may produce ADHD-like behavior, but the evidence does not justify describing it as a direct cause of clinical ADHD or depression in every affected child.[4]

Cardiovascular effects

Pediatric OSA has been associated with altered blood-pressure regulation, systemic hypertension, autonomic dysfunction, endothelial dysfunction and changes in ventricular structure and function. Intermittent hypoxia, sympathetic activation, inflammation and repeated intrathoracic pressure changes are proposed mechanisms. Cardiovascular abnormalities are often subclinical during childhood but may indicate an early pathway toward later disease.[10]

A meta-analysis of 12 cross-sectional and two cohort studies found higher systolic blood pressure in children with mild and moderate-to-severe OSA than in healthy controls, with more pronounced differences at night. Moderate-to-severe childhood OSA was associated with a 4.02-mmHg higher systolic blood pressure in adulthood (95% CI: 1.32–6.72 mmHg). This longitudinal association suggests that childhood disease may have cardiovascular implications beyond the pediatric period, although observational evidence cannot establish causality.[14]

Pulmonary hypertension appears uncommon in otherwise uncomplicated pediatric OSA. In a cohort of 2,020 children with OSA, pulmonary hypertension was present in 1.8%, and every affected child had an associated cardiac condition. Evidence relating OSA to pediatric arrhythmias is also limited, although altered P-wave and QT dispersion has been reported in children with severe disease.[10]

Metabolic and growth-related effects

Metabolic associations include insulin resistance, dyslipidemia, hypertension and metabolic syndrome, particularly when OSA coexists with obesity. A systematic review of adolescents with obesity found that OSA was associated with greater risks of dyslipidemia, insulin resistance and hypertension. Although obesity remains a major determinant of metabolic dysfunction, OSA appeared to add to this burden independently. Earlier evidence suggested approximately six-fold higher odds of metabolic syndrome among adolescents with OSA, but definitions of pediatric metabolic syndrome were not standardized.[3,15]

Failure to thrive is now reported in 5% or fewer of pediatric OSA cases. Proposed mechanisms include disruption of growth-hormone secretion and reduced insulin-like growth factor-1, although growth responses vary among children. Improvement in growth may occur after treatment, but children with obesity require monitoring

because postoperative weight gain can also occur.[3]

Respiratory effects

Asthma and OSA may aggravate one another through shared inflammatory and mechanical pathways. In a cohort of 40,518 children with asthma, adenotonsillectomy was associated with reductions of 30.2% in acute asthma exacerbations, 25.6% in asthma-related emergency visits and 35.8% in asthma-related hospitalizations. Bronchodilator, inhaled corticosteroid and systemic corticosteroid use decreased by 16.7%, 21.5% and 23.7%, respectively. Among children with poorly controlled asthma and OSA, annual exacerbations decreased from 4.1 ± 1.3 to 1.8 ± 1.4 following adenotonsillectomy. These findings were largely observational and do not prove that surgery will improve asthma in every child.[12]

Nocturnal enuresis

Nocturnal enuresis was present in 1,113 of 3,550 children with sleep-disordered breathing, representing approximately one-third of the pooled population. Its prevalence decreased from 31% before adenotonsillectomy to 16% after surgery among children with available follow-up data. Impaired arousal, altered nocturnal urine regulation and changes in bladder pressure have been proposed as possible explanations. Most included studies did not distinguish primary from secondary enuresis, and the observational findings do not establish that upper-airway treatment directly resolves bed-wetting.[16]

Role of Dental Professionals in Early Recognition

Dentists can contribute by combining a brief sleep history with oral, dental and craniofacial examination. Caregivers should be asked about snoring on three or more nights per week, witnessed pauses, gasping, choking, labored breathing, restless sleep and habitual mouth breathing. Morning headache, unusual sleeping posture, secondary enuresis, daytime sleepiness, hyperactivity and learning or behavioral problems provide additional reasons for referral.[2]

The examination should record tonsillar enlargement, high-arched or narrow palate, maxillary constriction, increased overjet, mandibular retrognathia and a long-face growth pattern.[5,6] Body habitus and known diagnoses such as Down syndrome, asthma, craniofacial anomalies or neuromuscular disease should also be considered.[11,12] The absence of these findings does not exclude OSA, and tonsillar size alone correlates poorly with disease presence or severity.[2,10]

Questionnaires may provide a consistent framework for chairside screening. The Sleep-Related Breathing Disorder scale of the Pediatric Sleep Questionnaire had 76% sensitivity and 43% specificity when OSA was defined as AHI ≥1 event/hour. OSA-18 had lower sensitivity at 56% but higher specificity at 73%. The SRBD-PSQ may identify more children at risk, but its low specificity results in many false-positive screens. Neither questionnaire can confirm OSA or safely exclude it when clinical suspicion remains high.[17]

Dental professionals should explain to caregivers that referral does not mean the child has OSA. It indicates that the combination of sleep symptoms and clinical features justifies medical assessment. This distinction prevents unnecessary alarm while supporting timely evaluation.

Diagnosis, Referral and Multidisciplinary Management

Overnight laboratory polysomnography remains the diagnostic standard because history, physical examination, tonsillar grading and screening questionnaires have limited diagnostic accuracy. An AHI greater than 1 event/hour is considered abnormal, while an AHI of 1–4, 5–9 and ≥10 events/hour may be categorized as mild, moderate and severe OSA, respectively.[2,10]

Dental professionals who suspect OSA should refer the child to a pediatrician, otolaryngologist or sleep specialist rather than initiating treatment solely from dental findings.

Adenotonsillectomy is recommended for appropriate children with polysomnography confirmed OSA and adenotonsillar hypertrophy. Children younger than 3 years or those with severe OSA require overnight postoperative monitoring, particularly when the obstructive AHI is ≥10 events/hour or the oxygen-saturation nadir is <80%.[2,18] Families should be informed that OSA may persist or recur, especially in children with obesity, asthma, craniofacial abnormalities or severe baseline disease.[12,18]

CPAP is appropriate when surgery is unsuitable or clinically important OSA persists after treatment, although adherence can be difficult in children. Weight management should accompany other therapies for children with overweight or obesity rather than delaying active OSA treatment. Intranasal corticosteroids or leukotriene modifiers may benefit selected children with mild or residual disease, but treatment response and long-term effectiveness vary.[2,3]

Rapid maxillary expansion, oral appliances and other orthodontic approaches may have a role in

carefully selected patients, but current evidence does not support their routine use for every child with OSA. Such treatment should follow medical diagnosis and multidisciplinary assessment. Clinical reassessment is generally advised approximately 6–8 weeks after therapy, with objective testing for high-risk children or those with persistent symptoms.[2,3] Children with Down syndrome should undergo polysomnography by 4 years of age, and continued surveillance may be necessary because risk can increase or recur with age.[11]

Future Directions

Longitudinal studies are needed to determine whether craniofacial characteristics precede OSA or change in response to chronic airway obstruction. Standardized diagnostic thresholds would improve comparison across pediatric studies. Screening instruments require validation in dental and orthodontic settings before widespread implementation. Future research should identify which children are most likely to benefit from orthodontic intervention and whether early treatment alters craniofacial development. Longer follow-up is also required to clarify whether treatment reduces cardiovascular, metabolic and neurocognitive risk into adulthood.

II. Conclusion

Pediatric OSA is an oral–systemic disorder with potential effects on craniofacial growth, behavior, learning, cardiovascular regulation, metabolism, respiration and urinary control. Dental and craniofacial findings may support early recognition but cannot establish a diagnosis. Dentists can contribute by asking focused sleep questions, recognizing relevant clinical features, educating families and initiating appropriate referral. Polysomnography and specialist assessment remain necessary for definitive diagnosis and treatment planning. Management may include adenotonsillectomy, CPAP, weight reduction, anti-inflammatory treatment and selected orthodontic interventions. Timely multidisciplinary care offers the best opportunity to reduce preventable morbidity while avoiding unsupported diagnosis or treatment based solely on dental morphology.

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