REVIEW ARTICLE

# Pediatric sleep apnea-need for multidisciplinary approach

**J. C. Suri**

Consultant, Professor and Head - Department of Pulmonary, Critical Care & Sleep Medicine, Vardhman Mahavir Medical college and Safdarjang Hospital, New Delhi

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## Abstract

Sleep disordered breathing (SDB) in children is a frequent disease with a prevalence varying from 1–5%. It is distinct from adults with respect to ideology, gender distribution, clinical manifestation, and treatment. Adenotonsillar enlargement is the most common cause of SDB in children. The diagnosis of SDB requires the use of special sensors such as nasal pressure transducer and esophageal pressure monitoring. The treatment of SDB in children includes amelioration of symptoms, normal cranio-facial growth and prevention of adult SDB. Adenotonsillectomy (AT) is the first line treatment of otherwise healthy children and also the initial treatment for children with multifactorial SDB. The success of AT as defined by reduction of AHI below 1 varies between 30 and 50% in various studies. A number of clinical factors such as nasal allergy, narrow and high hard palate, retro-position of mandible, enlargement of nasal inferior turbinates, high Mallampatti scale score, long face syndrome, age more than 8 years at the time of AT, and pretreatment apnea- hypopnea index (AHI) were associated with poor outcome. An impairment of nasal breathing due to adenotonsillar enlargement results in abnormal development of maxilla-mandibular skeleton resulting in narrowed upper airway. Surgery should be performed in young children as early as possible. Majority of patients have residual disease which requires additional treatment with orthodontic procedures such as rapid maxillary expansion (RME) and nasal CPAP. A multidisciplinary approach to evaluation and management of these children may lead to better treatment outcome.

**Keywords:** Pediatric sleep apnea, adenotonsillectomy, polysomnography, Rapid maxillary expansion

## Introduction

Similar to adults, sleep disordered breathing (SDB) in children also presents as a spectrum of disorders with increasing upper airway resistance. These include habitual snoring, upper airway resistance syndrome, obstructive hypoventilation and obstructive sleep apnea and hypopnea syndrome. However, SDB in children is distinct from SDB that occurs in adults, particularly with respect to gender distribution, etiology, clinical manifestations, and treatment. Childhood SDB is common with a prevalence of 1-5% in children aged 2–18 years[1]. In a study conducted by the author in school children in Delhi, SDB was found to be present in 4.8% of children with equal prevalence in both boys and girls[2]. The most common factors involved in the pathogenesis of SDB in childhood are anatomically small upper airway

*Address for correspondence*
**J. C. Suri**
Consultant, Professor and Head
Department of Pulmonary, Critical Care & Sleep Medicine, Safdarjang Hospital, New Delhi, India
Email: docjcsuri@gmail.com

due to adenotonsillar enlargement or craniofacial abnormalities and inability to mount adequate neuromuscular compensation during sleep. The other factors such as low arousal threshold and high loop gain (genetically determined high ventilatory response to hypoxia or hypercapnea) may also contribute. The apneic or hypopneic events are usually associated with oxygen desaturations and are terminated by arousals leading to sleep disruption. The implications of SDB and the associated hypoxemia and sleep fragmentation in children are potentially complex and if left untreated, or treated late, pediatric SDB may lead to significant morbidity affecting multiple organs and systems.Such deleterious consequences may not be completely reversible despite appropriate treatment[3].

The nocturnal symptoms in children suffering from SDB are continuous heavy snoring,disrupted nocturnal sleep (with or without nightmares and/or night terrors),nocturnal enuresis, and paradoxical or labored breathing frequently observed by parents[4–12]. The daytime symptoms include excessive daytime somnolence (EDS) commonly noted by school teacher, abnormal daytime behaviors: such as aggressive behavior toward peers;hyperactivity; significant disciplinary issues, andlearning problems: such asdelayed language development and inadequate performance in school[13,4]. In severe cases child may develop hypertension, cor-pulmonale, and acute cardiac failure[15–20].

The diagnosis of SDB depends on history, sleep questionnaires, physical examination, audiotaping or videotaping and nocturnal polysomnography (PSG)[21–24]. The gold standard test for the diagnosis of SDB is PSG which is currently the only technique shown to detect most accurately various breathing and sleep abnormalities seen in patients of SDB. Polysomnography can be done on patients of all age groups and can distinguish primary snoring from OSA. There has always been controversy regarding the exact polysomnographic parameters which predict morbidity and mortality[25–27]. The diagnosis of SDB in children is made when the apnea- hypopnea index (AHI) is more than 1/hour of sleep. This is based on a number of studies on normative respiratory events values in children between ages of 1–18 years[28,29]. The respiratory events are rarely associated with clear EEG arousals and significant oxygen desaturations, i.e. $SaO_2$ drops by 3 or 4%. However, many pediatric patients may not have typical apnea or hypopnea on PSG and instead show prolonged periods of flow limitations or hypoventilation as primary PSG abnormality. These patients will require additional sensors such as nasal pressure transducer, esophageal pressure monitoring, peripheral arterial tonometry (PAT), and end tidal $CO_2$ (capnography) for the detection of abnormal breathing patterns[30,31].

The decision to treat a patient should be individualized based on careful assessment of the age of the patient, severity of PSG abnormality and comorbidity or complications related to OSA. Any child with symptoms of regular snoring, restless sleep, and mouth breathing who has significant behavioural and learning issues and an AHI of more than 1 should be seriously considered for treatment.

Adenotonsillectomy (AT) is the first line treatment of otherwise healthy children who have SDB and adenotonsillar enlargement and also the initial treatment for children with multifactorial SDB.

To do or not to do PSG before AT is controversial and has been a matter of debate. Practice guidelines published by the American academy of pediatrics[32] and the American thoracic society[33] recommend routine use of PSG prior to AT, whereas guidelines issued by the American academy of otolaryngology–head and neck surgery[34] do not support this view and suggest that PSG should be performed only under select circumstances.In India less than 1% of children receive preoperative PSG.One of the reasons for recommending PSG before AT is the accuracy of diagnosis as it has been observed thathistory and physical examination are not accurate in predicting the presence or absence of SDB. Adeno-tonsillar size has also not been found to consistently correlate with the presence or severity of SDB. In a systematic review, majority of studies concluded that the clinical evaluation was not reliable for diagnosis of SDB compared to standard PSG[35]. Many screening tools such as questionnaires, oximetry, and home audio/video recording have been studied but not found to be as reliable as standard PSG[36]. The other reason for doing PSG before AT is to select patients who are at a high risk of developing serious complications immediately after AT. A number of clinical and polysomnographical parameters have been identified which are associated with increased morbidity and mortality following AT37[38]., The PSG findings of an AHI of more than 40/hour and a minimum oxygen saturation of less than70% may indicate increased risk for postoperative complications[37]. On the contrary, it has also been suggested that simple snoring may not be benign and is associated with significant neurocognitive and behavioral consequences and such patients may benefit from the surgical treatment which would otherwise be not indicated on PSG criteria[39]. This would mean that all children who have snoring and significant daytime consequences of SDB, can proceed with surgery (AT) without PSG.

The outcome of AT has been studied extensively and a number of meta-analyses and systematic reviews have been published recently[40–46]. The evidence from these studies clearly shows that after AT there is a significant improvement in the AHI in most cases making it an important first line treatment, but the cure as defined by post-operative AHI of less than 1 could be achieved in 30–50% of the patients. A number of factors have been proposed for the poor outcome of AT in these patients. In the study conducted by Guilleminault et al[41], inadequate response was associated with presence of nasal allergy, narrow and high hard palate, retro-position of mandible, enlargement of nasal inferior turbinates, high Mallampatti scale score, and long face syndrome. In a large multicentre retrospective study involving 578 patients, factors associated with the poor outcome of AT were age of the patient, i.e. more than 7 years and pre adenotonsillectomy AHI index[47]. In a study conducted in our department involving 50 patients the overall success of AT was 37.8% when the criteria for cure was an AHI of less than 1/hour. The predictors of poor outcome were pretreatment AHI index, high arch palate, Mallampati score of III and IV and age of the patient (>8 years). Only one-third of patients below 8 years of age and none of the patients above 8 years were cured of their SDB after AT[48]. It is clear from these studies that timing of AT has an important bearing on the success of treatment, the earlier it is done the better is the result.

It is well known that the cranio-facial skeleton grows very quickly in children and by the age of 4 years 60% and by 10/11 years 90% of the developmentof adult face is complete. By applying constant force, tongue plays an important role in the growth and development of hard palate in children breathing through their nose. Experiments on baby rhesus monkeys have shown thatwhen nasal breathing was impaired artificially by putting hollow silicon nasal plugs, it led to chronic mouth breathing which ultimately resulted in cranio-facial changes such as narrowing of dental arches, decrease in maxillary arch length, anterior crossbite, maxillary overjet, and increase in anterior face height[49].Nasal obstruction due to adenoids and tonsillar enlargement early in life may adversely affect the growth of the face by causing chronic mouth breathing and loss of contribution of tongue. This would lead to the development of similar changes as described in monkey studies resulting in an anatomically narrow upper airway before puberty. These features in the milder forms have also been noted in children with adenotonsillar enlargement without SDB and would predispose them to develop SDB in later life. These facts highlight the importance of nasal breathing early in life for the normal development of cranio-facial structures and necessitate its early restoration before the child becomes habitual mouth breather and permanent changes in the facial morphology occur.

With very few patients getting cured of their SDB after AT, it would be necessary to do some form of post-operative assessment, preferably a PSG to look for the presence and severity of residual disease. Majority of these patients would therefore, need additional treatment to cause complete resolution of disease. Orthodontic procedures such as rapid and slow maxillary expansion may be useful in patients with narrow maxilla[50,51]. Rapid maxillary expansion(RME) uses a devise with central screw and 4 arms which anchor on permanent pre-molars and first molar on both sides and by applying orthopaedic forces on mid-palatine sutures causes widening of the maxillary base[51]. In a comparative study of AT and RME as the primary mode of treatment for the pediatric SDB, it was found that neither was effective however, a combination of AT and orthodontic procedures resulted in cure of SDB in majority of patients[52].RME is quite effective in causing lateral widening of the maxillary base but has limited capability in anteroposterior lengthening capabilities. In such a situation, distraction osteogenesis may be performed provided oral-facial growth is well advanced.

Nasal continuous positive airway pressure (CPAP) is the treatment of choice in patients who have contraindication to surgery or who refuse surgery[53–56]. Children with neuromuscular diseases, obesity, severe bleeding tendency, craniofacial abnormality, and small tonsils are usually not suitable for surgery.Home nasal CPAP has been used in infants, pre-pubertal children, and pubertal children. The difficulty in application of nasal CPAP relates to training of the family and child as well as finding the appropriate nasal interface. Children often need to be trained to tolerate the facial interface. Behavioral modification techniques and daytime training may help with this training[55]. The prolonged use of nasal CPAP in children may rarely result in the development ofmidface hypoplasia[56].

## Conclusion

SDB in children is associated with complex and multiple abnormalities of soft tissue and cranio-facial skeleton of upper airway. SDB in prepubertal children presents with wide range of physical, psychological, social, and behavioral symptoms. By the age of 4 years 60% and by 10 years 90%of the development of face is complete. AT is associated with significant improvements in sleep disordered breathing in the vast majority of children. However, in only 35–50% of children was OSA effectively cured by AT. Older children (>7–8years of age) with severe OSA and those with high Mallampatti score are particularly at increased risk for residual OSA. Early surgery may allow normal face growth and prevent the development of permanent morphometric changes of upper airway due to impaired nasal breathing. Majority of these patients may require additional treatment with orthodontic procedures or nasal PAP. Partial treatment may lead to reappearance of problem post puberty and lead to development of adult OSA. So a multidisciplinary approach to evaluation and management of these children may lead to overall better treatment outcome.

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