Obstructive Sleep Apnea in Children

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Introduction
Obstructive sleep apnea is defined as a breathing disorder characterized by prolonged upper airway partial and/or intermittent complete obstruction that disrupts normal ventilation during sleep and normal sleep patterns accompanied by associated signs and symptoms characteristic of the disorder.1-2 (According to American Academy of pediatrics clinical practice guidelines)

Obstructive sleep apnea syndrome (OSAS) was first reported in children by Guilleminault et al (1976) following which recognition of abnormal breathing during sleep has progressed.3 The prevalence of OSA in childhood is around 2-3% affecting all ages with peak incidence between 2-8 years. 4 Frequent snoring is reported by parents in 3-15% of children while prevalence of reported apneic events is 0.2-4%. 4 OSA is a part of a complex of sleep disordered breathing (SDB) with a spectrum of clinical manifestations ranging from primary snoring to OSAS with upper-airway resistance syndrome (UARS) falling in between the extremes.4

Primary Snoring
Snoring during sleep without associated apneas, gas exchange abnormalities or excessive arousals is defined as primary snoring. This does not progress to OSAS in young children and is known to resolve over time.5

Upper Airway Resistance Syndrome (UARS)
UARS is characterised by snoring, partial airway obstruction leading to increasingly negative intrathoracic pressures during inspiration. This causes arousals, sleep fragmentation but there is preservation of airflow and oxygenation. Thus, there is no evidence of apnea, hypopnea or gas exchange abnormalities on polysomnography. Polysomnography (PSG) shows snoring with marked paradoxical breathing movements or repetitive arousals. Increased respiratory effort with arousals clinches the diagnosis of UARS on esophageal pressure monitoring.7-2

Obstructive Sleep Apnea Syndrome
1-3% children have OSAS and 40% of children referred to sleep medicine or an otolaryngologist may have OSAS. Obstructive sleep apnea is characterised by prolonged upper airway obstruction during sleep resulting in partial or complete cessation of breathing associated with reduction of oxyhemoglobin saturation or hypercarbia, or both. This may lead to obstructive apnea or obstructive hypoventilation. Obstructive hypoventilation is not seen in adults usually. It leads to paradoxical respiratory efforts, hypercarbia and occasionally hypoxemia.7-8 The obstrucive events resulting in hypoxia and multiple arousals contribute to metabolic, cardiovascular and neurocognitive changes.

Pathophysiology
Upper airway is a collapsible tube, the patency of which is determined by the tone of the pharyngeal muscles causing dilatation as well as phasic contractions of these muscles during inspiration. 7-Pathophysiology can be arbitrarily divided into anatomical factors, control of airway patency and obesity.
Anatomical factors
Changes in the anatomical framework of the head and neck, affects the calibre of pharyngeal airway, which in turn contributes to increased risk of airway obstruction. The most common cause for childhood OSA is adenotonsillar hypertrophy. Arens et al used MRI to study the airway of children with OSAS. These children had significantly enlarged tonsils and adenoids leading to smaller upper airway volumes as compared with controls. In addition positive correlation was seen when percent difference of combined tonsil and adenoid volume between each subject and matched controls was plotted against apnea-hypopnoea index. Soft palate volume was also found to be more in subjects with OSA. Children with OSA have hyperplasia of lymphoid tissue in regions other than Waldeyers ring also. Kun-Tai kang et al reviewed 495 children with OSA and studied the contribution of adenoid and tonsil size in childhood OSA and interactions between adenotonsillar hypertrophy, age, and obesity. They found that the effect of adenoid size on OSA decreased in adolescence. Moreover, both adenotonsillar hypertrophy together increased OSA risk more than tonsil or adenoid hypertrophy alone.
Adenoidal- nasopharyngeal space is narrowest at 4.5 years of age and adenoidal mass reaches greatest size at 7-10 years of age when facial framework rapidly develops. This space gradually decreases until 12 years of age. Thus the influence of adenoid size decreases as the child reaches adolescence. However, there is no such correlation between age, tonsil size and OSA in children.
Craniofacial factors include hypoplasia or retro-positioning of maxilla or mandible, large or retro-positioned tongue. Evaluation of children with craniofacial anomalies is directed towards assessing skull base shape, maxillary size and shape, tongue size and support and mandibular size and shape. OSAS in such children persists even after adenotonsillectomy and additional surgical as well as non-surgical therapies are generally required. Infants usually require tracheostomy. Skeletal dysplasias such as achondroplasia may lead to ‘mixed apnea syndrome’. Brainstem or cervico-medullary compression results in abnormal ventilatory drive and central apnea. Adenotonsillar enlargement and craniofacial factors cause Obstructive sleep apnea.
Control of airway patency
Anatomical factors are not the only determining factor leading to OSA in pediatric age group. Children with large tonsils may not have OSA. Since upper airway is a collapsible tube, Marcus et al proposed the ‘Starling Resistor Model’ of the upper airway. According to this model maximum inspiratory flow through a collapsible upper airway is determined by upstream (nasal) pressure changes and pressure changes surrounding the collapsible segment. This airflow is not dependent on the tracheal pressure generated by the diaphragm. The pressure outside the airway is determined by the activity of the airway dilator muscles. The pressure at which airway collapses is termed as critical closing pressure (Pcrit) and this is a measure of airway collapsibility. Children with OSA have more airway collapsibility (Pcrit was higher or less negative) than children with primary snoring.
Obesity
With increasing epidemic of obesity in children especially in developed countries and now in the developing countries too, more and more children presenting with OSA are observed to be obese. Classical presentation of a child with adenotonsillar hypertrophy and failure to thrive is now being replaced with an obese child presenting with OSA. These children may or may not have adenotonsillar hypertrophy. They usually present at a later age and the clinical profile resembles adult OSA phenotype. A percentage of this group of children undergoing adenotonsillectomy (AT), still have residual OSA following surgery.
Possible mechanisms causing OSA in childhood obesity are as follows.7,9
- There are alterations in mechanisms regulating upper airway patency and increased airway collapsibility in obese children.
- Central obesity reduces functional residual capacity by limiting diaphragmatic descent, more during supine position. Moreover, these
children have decreased lung compliance leading to hypoventilation, atelectasis and ventilation-perfusion mismatch.

- OSA can induce leptin resistance and increase ghrelin levels both of which can increase obesogenic behaviour.

Three subtypes of childhood OSA have been identified:

Type I marked increased lymphoid tissue in upper airways in absence of obesity

Type II milder lymphoid hypertrophy associated with obesity

Type III Children with syndromic OSAS

Conditions associated with obstructive sleep apnea in children

- Adenotonsillar hypertrophy
- Obesity
- Allergic rhinitis
- Mucopolysaccharidoses/metabolic storage diseases
- Macroglossia
- Laryngomalacia

- Conditions with reduced upper airway patency

- Conditions with abnormal muscle tone

- Craniofacial syndromes

- Neurologic disorders

OSA and Inflammation

OSA leads to elevation of CRP. An animal model of intermittent hypoxia and hypercapnia lead to elevation of interleukin-6 (IL-6) level, which is a precursor of CRP. Some studies demonstrated increased levels of proinflammatory cytokines TNF-α, IL-6 and IL-1α from OSA derived tonsils. They postulated that recurrent vibrations of the upper airway during snoring, promotes localized inflammation. Goldbart et al showed higher levels of leukotriene B4 and cysteinyl leukotriene in children with OSA. Even sputum from children with OSA exhibits neutrophilia as compared with controls.

Sequelae of OSA

OSA causes intermittent hypoxemia and subsequent sleep fragmentation which induces local and systemic inflammation. The combination of these inflammatory cascades and oxidative stress mechanisms lead to cell injury, dysfunction and cell death affecting various targeted organs.

Cardiovascular system

OSA can promote cardiovascular disturbances in blood pressure regulation, ventricular remodelling and endothelial dysfunction. Children can have a vast variety of cardiovascular symptoms including systemic and pulmonary hypertension, and cor pulmonale with heart failure. Majority of children, show significant improvement in endothelial function after treatment of OSA with adenotonsillectomy. Children with OSA have a higher diastolic BP during sleep as compared with primary snorers though elevations in systemic BP were noted even in children with primary snoring.

Amin et al demonstrated overnight greater changes in brain natriuretic peptide (BNP) in children with moderate to severe OSA as a result of frequent negative intrathoracic pressure swings. This accounts for the ventricular dysfunction in these children. Endothelial dysfunction is known to be a precursor of atherosclerosis. Children with concomitant obesity and OSA have a greater degree of endothelial dysfunction as compared to children with only OSA. OSA severity is associated with a decrease in T regulatory lymphocytes (Tregs) in peripheral blood of children with OSA. Tregs have shown to inhibit development and progression of atherosclerosis. Gozal et al stated that OSA in children is strongly related to changes in Tregs and their function. This in turn contributes to cardiovascular morbidity in children.

C-reactive protein (CRP), an acute phase reaction
protein has recently emerged as one of the powerful independent predictors of risk for future cardiovascular morbidity and is now widely used to stratify risk for ischemic heart disease. Increased CRP levels have been demonstrated in children with OSA and the level of rise in CRP levels is proportionate to the severity of OSA. However, there is significant reduction in these levels following effective treatment.6,9 Polverino and associates studied 101 children and found that AHI (Apnea- Hypopnea index) was significantly associated with Hs-CRP (high sensitivity CRP). Hs-CRP was significantly higher in children with OSA. Children with OSA and raised CRP levels are found to be at a greater risk for the development of long term cardiovascular complications.14

Behavioural and neurocognitive impairment
SDB is associated with poor learning, poor school performance, attention deficits, concentration difficulties, hyperactivity and impulsivity. This is secondary to fragmented, non-restorative sleep with intermittent hypoxia and its effect on the development of prefrontal cortex. Prefrontal cortex is responsible for behavioural control, working memory, organisation, analysis and self-regulation of motivation.2,15 ‘Memory consolidation’ occurs during REM sleep whilst growth hormone is produced in slow wave sleep. Thus sleep fragmentation occurring in OSA affects both cognition and interferes with growth. Even children with ‘primary snoring’ (without gas exchange abnormalities) or mild OSA can present with neurobehavioral changes.12 Maria et al concluded in their study that executive dysfunction is related to nocturnal hypoxemia rather than daytime sleepiness.15

Gozal in 1998 performed a study in children whose school performance was in the lowest 10th percentile of their class. There was a marked prevalence of OSA in these children. Moreover, children who were treated (adenotonsillectomy) showed significant improvement in school grades.2,9,12 Khadra et al hypothesized that neurocognitive impairment occurs as a result of changes in cerebral blood flow during sleep.9 It is further emphasized that not all children with OSA exhibit behavioural and cognitive deficits. Both genetic and environmental factors play a major role in phenotypic expression of these deficits. Following treatment of OSA improvement occurs in behaviour and cognition. However, early diagnosis and prompt treatment is advised as some neurocognitive changes are only partially reversed if left too long.

Metabolic sequelae
OSA has been associated with failure to thrive. Children with OSA and primary snorers have disruption of slow wave sleep, during which growth hormone (GH) and insulin-like growth factor (IGF-1) are secreted. Failure to thrive results from reduction of IGF-1 or insulin-like growth factor binding proteins (IGFBPs) which significantly reverses following adenotonsillectomy (AT).9 Furthermore, there may be dysphagia and increased energy expenditure leading to lesser intake.4 In contrast to the earlier presentation of failure to thrive, more and more children presenting with OSA are now obese. Sleep fragmentation and intermittent hypoxia is associated with reduced insulin sensitivity and dyslipidemia in obese children. Fatty liver disease has been demonstrated in children with OSA and obesity. Treatment with AT followed by continuous positive airway pressure led to improvement in liver serum aminotransferases.9 Rise in low-density lipoprotein (LDL) cholesterol along with lowering of high-density lipoprotein (HDL) cholesterol is seen in OSA children irrespective of the presence or absence of obesity.9

Diagnosis
Clinical manifestations
Early diagnosis and prompt management of OSAS, results in decreased morbidity and reversal of most of the sequelae of OSA. Primary snoring and OSA cannot be differentiated by history and examination alone. Symptoms in pediatric age group are dependent on the age of the child. Nocturnal symptoms noticed by parents are seen in all age groups. They include loud, frequent snoring, choking, breathing pauses, restless sleep, arousals and nocturnal enuresis. These children
usually have unusual sleeping postures. They keep their necks hyperextended to maintain patency of upper airway. They are also known to present with paradoxical breathing, mouth breathing, nocturnal sialorrhea, nocturnal sweating, parasomnia, and bruxism.2, 6, 7 (see box 2)

Most of the time daytime symptoms are more pronounced and apparent in older children. Excessive daytime sleepiness (EDS) is seen only in 7-10% of the cases. EDS is seen in children with severe OSA and obesity and is associated with higher incidence of complications (40-50%). More commonly pediatric OSA presents as hyperactivity and inattention during the day, moodiness, poor learning in school as explained earlier.2, 9

<table>
<thead>
<tr>
<th>Symptoms of pediatric OSA 2, 8 (Box 2)</th>
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<tbody>
<tr>
<td><strong>Daytime symptoms</strong></td>
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<tr>
<td>• Mouth breathing</td>
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<tr>
<td>• Poor appetite/difficulty in swallowing</td>
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<tr>
<td>• Morning headache</td>
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<tr>
<td>• Nasal speech</td>
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<tr>
<td>• Attention deficit/aggression/moodiness</td>
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<tr>
<td>• Hyperactivity</td>
</tr>
<tr>
<td>• Chronic nasal congestion/rhinorrhoea</td>
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<tr>
<td><strong>Nocturnal symptoms</strong></td>
</tr>
<tr>
<td>• Snoring</td>
</tr>
<tr>
<td>• Choking/gasping</td>
</tr>
<tr>
<td>• Paradoxic breathing</td>
</tr>
<tr>
<td>• Retractions (cervical or costal)</td>
</tr>
<tr>
<td>• Abnormal sleep positions</td>
</tr>
<tr>
<td>• Frequent awakenings</td>
</tr>
<tr>
<td>• Enuresis</td>
</tr>
<tr>
<td>• Sweating</td>
</tr>
<tr>
<td>• Dry mouth</td>
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<tr>
<td>• Bruxism</td>
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<td>• Parasomnia</td>
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Examination
First step to examining a child suspected to have OSA is to note the height and the weight of the child, because growth can be impaired in the child. A full ororhinolaryngologic examination must be conducted to establish the site of static or dynamic airway obstruction.4, 6, 7, 12, 16

Child suspected to be suffering from OSA will have a typical ‘long face syndrome’. These mouth breathers have altered dento-alveolar morphology. The characteristic features include high arched palate, increased lower facial height, narrow maxilla and retrognathia.5 (see box 3)

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<th>Physical examination in pediatric obstructive sleep apnea syndrome 2 (Box 3)</th>
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<tr>
<td><strong>General</strong></td>
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<tr>
<td>• Obesity</td>
</tr>
<tr>
<td>• Failure to thrive</td>
</tr>
<tr>
<td>• Sleepiness</td>
</tr>
<tr>
<td>• Increased neck circumference</td>
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<tr>
<td><strong>Head and neck</strong></td>
</tr>
<tr>
<td>• Tonsillar hypertrophy</td>
</tr>
<tr>
<td>• High arched palate</td>
</tr>
<tr>
<td>• High/ large tongue position</td>
</tr>
<tr>
<td>• Overbite</td>
</tr>
<tr>
<td>• Posterior buccal cross bite</td>
</tr>
<tr>
<td>• Elongated soft palate</td>
</tr>
<tr>
<td>• Long face syndrome</td>
</tr>
<tr>
<td>• Midfacial hypoplasia</td>
</tr>
<tr>
<td>• Micrognathia/retronathia</td>
</tr>
<tr>
<td>• Deviated septum</td>
</tr>
<tr>
<td>• Swollen nasal mucosa</td>
</tr>
<tr>
<td><strong>Cardiovascular</strong></td>
</tr>
<tr>
<td>• Systemic/pulmonary hypertension</td>
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Polysomnography
Overnight polysomnography in a sleep laboratory is the gold standard for diagnosis of pediatric OSAS and is the best technique to differentiate between primary snoring and OSA.7 Accurate diagnosis of the severity of OSA will ensure the proper treatment in children and will defer unnecessary surgeries where ever not required.

American Academy of Thoracic Society, Standards and Indications of Cardiopulmonary Studies in children recommends measurement of the following parameters: 7
1. Sleep State: Electroencephalogram, electromyogram, electro-oculogram.
2. Respiratory parameters: abdominal and chest wall movements, oronasal airflow, end tidal CO2,
oxygen saturation with pulse oximetry
4. Audio video recording

Sleep related upper airway obstruction is diagnosed in children who have evidence of significant obstructive hypoventilation or more than one obstructive apneic episode per hour. Obstructive apneic episodes in children often are shorter than 10 seconds as compared with adults where they last for more than 10 seconds. Two or more consecutive breaths with obstructive apneas or hypopneas are considered abnormal in children.7

Obstructive apnea is defined as near complete cessation of airflow despite on-going respiratory effort. Obstructive hypopnea is defined as partial upper airway obstruction resulting in greater than 50% reduction in airflow associated with either an arousal or desaturation of 3% or greater from baseline. Apnea-hypopnea index (AHI) reflects the number of discrete obstructive events per hour. There is no international consensus for AHI cut off values to initiate treatment in children. The current accepted arbitrary cut off for AHI is > 3 standard deviations beyond mean of normative AHI in healthy children. Children with an AHI <1/ hour total sleep time (TST) do not have significant OSA. On the other hand a child with AHI>5/ hour TST requires treatment.9 However, as there is no evidence based cut-off some children with AHI<5 may be symptomatic and require intervention.2 The American Society of Anesthesiologists guidelines defines severe OSA as AHI of 10 or more.2

It is seen that only 10% of children with habitual snoring referred for adenotonsillectomy actually undergo overnight sleep study. AAOHNS published clinical practice guidelines for use of PSG before tonsillectomy in children (see box 4).17 Many factors like the cost, inconvenience to the child and parents, expertise in pediatric sleep study and interpretation make most of the physicians consider PSG not necessary for diagnosis.8

Conventional numerical measures (eg. obstructive apnea-hypopnea index, arousal index, oxyhemoglobin desaturation index etc.) are poor indicators of morbidity in children.17 Additionally, many morbidities in children present after a long period of time. Children who are symptomatic, may show ‘normal PSG’ in the presence of habitual snoring and conversely, relatively asymptomatic snoring children, may have severe respiratory disturbances in their NPSG.

<table>
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<th>AAOHNS guidelines for use of PSG before tonsillectomy (SDB)2 (box 4)</th>
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<tbody>
<tr>
<td>• Obesity</td>
</tr>
<tr>
<td>• Downs syndrome</td>
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<tr>
<td>• Craniofacial abnormalities</td>
</tr>
<tr>
<td>• Neuromuscular disorders</td>
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<tr>
<td>• Sickle cell disease</td>
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<tr>
<td>• Mucopolysaccharidoses</td>
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To overcome the drawbacks of using as only NPSG as a diagnosing tool for the severity of OSA an

Other studies 4,7,11

In view of the expertise and resources required to perform a PSG several alternative studies under evaluation are:

1. Nap PSG can be helpful if positive but it has a high false-negative rate attributed to shorter total sleep time and lesser proportion of REM sleep in daytime nap.
2. Unsupervised overnight pulse oxymetry gives information about desaturation but cannot rule out OSA if negative. Associated recording of end-tidal CO2 provides additional information of cessation of airflow.
3. Video and audio recordings
4. Multichannel devices for home recordings offer cost effective details on sleep information in an unsupervised setting.
5. Flexible nasendoscopy can be used to evaluate the level of airway obstruction, lymphoid tissues and post nasal space. Sleep endoscopy- fibre optic endoscopy is performed under artificially induced sleep to determine the site of obstruction and plan the treatment subsequently.

Treatment

Surgical

Adenotonsillectomy (AT) is the first line of treatment in children with OSA and adenotonsillar hypertrophy. Combination of adenoid and tonsil surgery is considered superior and more effective in treating OSA than both alone.8,12
As compared to watchful waiting, surgical treatment of OSA improved symptoms, behavioural changes and quality of life according to a recently published Childhood adenotonsillotomy trial (CHAT). AT reduces AHI to < 1 event/hour in 25% to 71% children with OSA. Significant improvements are seen in quality of life, attention span, growth, behaviour, school performance and cognition.

Surgery is associated with complications which are more common in high risk group. The risk factors include children younger than 3 years, severe OSAS (apnea-hypopnea index more than or equal to 10 and/or oxygen saturation nadir less than or equal to 80%), failure to thrive, obesity, cardiac complications of OSAS, craniofacial and neuromuscular disorders and current respiratory infections. Careful pediatric intensive care monitoring is recommended for all high risk children in the post-operative period.

According to a retrospective study, although majority of the children showed marked improvements following AT, residual OSA was prevalent in a large subset of cases. Residual OSA was seen in severe OSA cases (AHI > 20/ hour TST), children older than 7 years, asthmatics, positive family history of OSA, African American race, high Mallampati score, craniofacial abnormalities, chromosomal defects and neuromuscular disorders and other obstructive causes like enlarged turbinates, deviated septum. Residual OSA is seen in >40% of children. A repeat sleep study is recommended 6-8 weeks post-surgery in these high risk children for recurrence or persistence of OSA.

Coblation assisted tonsillectomy is a new technique used at present, demonstrating decreased intraoperative blood loss and markedly reduced postoperative oedema and pain. Conventional adenoid curettage is now being replaced increasingly with endoscopic microdebrider assisted adenoidectomy. Since the adenoid removal is done under direct vision, it allows complete removal of both choanal and tubal lymphoid tissue causing obstruction.

Other surgical options

Palatal surgery (Uvulopalatopharyngoplasty, UPPP) is indicated in complicated OSAS in obese children, cerebral palsy, Down syndrome and children with craniofacial anomalies and neurologic impairments. Kershner and colleagues demonstrated a modest improvement in oxygen saturation nadir on PSG following UPPP. Tongue base procedures: Genioglossal advancement, radiofrequency ablation or coblation assisted tongue base reduction, partial midline glossectomy and lingual tonsillectomy are a list of a few procedures recommended in children with tongue base obstruction. Tongue base obstruction leading to OSA is typically seen in children with Down syndrome and Beckwith-Wiedmann syndrome.

Other craniofacial procedures include distraction osteogenesis and mandibular distraction in children with mandibular hypoplasia and retrognathia. Tracheostomy is reserved for severe OSAS in children who have failed other medical and surgical therapy, children with anatomic and neuromotor issues. Nasal surgeries like septum correction and turbinectomy are very rarely conducted in children.

Positive Airway Pressure (PAP)

This is often considered as second line therapy in children with OSAS in the following clinical situations:

• Persistence of symptoms (in children with other risk factors like obesity)
• Recurrence of symptoms after AT
• AT not performed or contraindicated
• Before surgery in severe OSAS

PAP involves delivery of pressurized air by an electronic device via a nasal or face mask acting as a pneumatic stent of the airway. PAP can be delivered as continuous (CPAP) or as bilevel pressure (BIPAP). Home nasal CPAP has been used in infants, prepubertal and pubertal children. Even though this therapy is highly efficacious adherence is particularly challenging in children. Complications of CPAP or BIPAP use in children, are global nasal flattening, midfacial hypoplasia, local discomfort like eye irritation, skin ulceration, rhinorrhea.

Other surgical options

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Pharmacologic Therapies

Medical therapy is considered as the first line option for children with mild OSA or as an adjunct to treatment of severe OSA.
Nasal steroids
Intranasal steroids have been known to be effective in residual lymphoid tissue or adenoidal regrowth after adenotonsillar hypertrophy or in situations where surgery is not performed. Nasal corticosteroids exert lympholytic action; reduce inflammation and upper airway edema. A small study with children younger than 10 years found that use of nasal fluticasone propionate decreased AHI from 10.7±2.6 to 6.8±2.2.10

Leukotriene receptor antagonists
Leukotrienes regulate inflammation in respiratory system. Both leukotrienes and their receptors are increased in adenotonsillar tissue and exhaled condensate of children with OSAS. Leukotriene receptor antagonists like montelukast treat OSA through anti-inflammatory action on this pathway. Concomitant use of both montelukast and and nasal budesonide for 12 weeks in children who had residual mild OSA after AT lead to significant improvement in AHI, respiratory arousal index and nadir oxygen saturation.9

Other Non-Surgical Therapies
Rapid maxillary expansion devices are used to widen hard palate by opening mid palatal suture and enlarging nasal cavities in prepubertal children. After 4 months of therapy, nasal resistance decreases and there is significant improvement in OSA symptoms in children with maxillary constriction. Oral appliances are also used which can be worn during sleep. They advance the mandible or the tongue increasing the size of the upper airway. Mandibular advancement and nasopharyngeal airways are other options in children with dysgagmia and hypotonia respectively.

Future Developments
Even though sleep studies provide an objective measure of sleep disturbances, the parameters used are not predictive of OSA associated morbidities. Home based studies and limited multichannel studies may provide a more economical option. Moreover, identification and use of biomarker approaches requires further exploration. Some data has shown a strong association between paediatric OSA and nocturnal rise in urinary neurotransmitters. Episodic hypoxemia and arousals result in increase in sympathetic activity causing rise in urinary epinephrine and norepinephrine. Overnight changes in 3 neurotransmitters: gamma - aminobutyric acid (GABA), decrease in taurine and decrease in beta-phenyl ethylamine (PEA) are postulated to differentiate children with OSA with neurocognitive defects from those without.

Summary
OSA is common in children and early recognition and referral is helpful in preventing longterm morbidity related to neuro development and pulmonary hypertension. Risk factors such as craniofacial abnormalities, Downs Syndrome, neuromuscular abnormalities should be identified early and addressed appropriately. A thorough evaluation of growth and development and otolarynologic evaluation, Investigations such as Sleep study (polysomnography), nasendoscopy are required before considering surgical options. Night CPAP or BIPAP therapy is also becoming more available in Indian setups for resistant cases of OSA not amenable to surgical correction. A clinical approach to any child with persistent snoring or episodes of apnea or hypopnea is shown in the flow diagram.

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