**Sleep Disordered Breathing (SDB) in Childhood**

 Ahmareen Oneza (SpR), Burke David (final year medical student) & Greally P.

Department of Respiratory Medicine

National Children’s Hospital Tallaght, Dublin 24

The American Academy of Paediatric’s clinical diagnosis and management Guidelines defines SDB ‘as a disorder of breathing during sleep characterised by prolonged partial upper airway obstruction and/or intermittent complete obstruction (obstructive apnoea) that disrupts normal ventilation during sleep and normal sleep patterns, leading to symptoms and signs**1**.

**Epidemiology**

The reported prevalence of SDB or what was formerly described as obstructive sleep apnoea syndrome has increased in paediatric populations in the developed world. This trend largely reflects the increasing prevalence of childhood overweight/obesity. We recently reported a prevalence of overweight/obesity in 25% of randomly selected healthy Irish school children.

The reported prevalence varies between 2-6% in school aged children. OSAS has a peak incidence in 2-8 years, which mirrors the peak in growth of the palatine tonsils and adenoids. However, it may affect children at any age, particularly obese adolescents**2,3**. Predisposing factors are listed in Table 1 **4**.

**Table 1.** Predisposing factors for SDB

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| --- |
| Adenotonsillar HypertrophyIncreased BMICraniofacial Syndromes (Pierre-Robin, Apert’s)African-American RaceUpper Respiratory Tract DiseaseHistory of low birth weight**5,6**Mucopolysaccharidoses (e.g. Hurler and Hunter syndrome)Orthodontic ProblemsFamily history of Obstructive sleep apnoea **7,8**  |

Individuals with any condition that gives rise to a less capacious nasopharynx are at higher risk (e.g. cranio-facial abnormalities, adeno-tonsillar hypertrophy, increased para pharyngeal adipose tissue, Down’s Syndrome, Prader\_willi Syndrome) . OSAS is commoner in children with neuromuscular disease where arousal responses are impaired and there may be increased hypotonia of the upper airway (e.g. muscular dystrophy, cerebral palsy, Arnold- Chiari malformation). OSAS may be familial and is commoner in African-Americans where co-morbidity with Sickle Cell Disease may provide particular challenges from both the diagnostic and therapeutic perspective.

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**Clinical Presentation**

History

The presenting complaint to the GP may depend on the age of the child. Parents of preschool children may complain of their snoring at night. Habitual snoring in the paediatric population may be high as 12%, but does not always mean a child suffers from OSAS**12**.Conversely, the absence of snoring does not necessarily exclude OSAS**13**. Other common symptoms include mouth breathing, sweating, restlessness, frequent awakenings at night and witnessed apnoeic episodes. Symptoms are often worse in the supine position. School-age children with OSAS may present with secondary enuresis, behavioural problems, attention deficit and failure to thrive. Older children and teenagers may complain of excessive daytime sleepiness.

Chervin et al**14** , interviewed the parents of 866 children about their sleep behaviour. They specifically assessed the presence of attention/hyperactivity disorder. As many as 22% of habitual snorers reported attention deficit and/or hyperactivity compared to only 12% of non-snorers**14**. Table 2 below outlines the signs and symptoms highlighted by the American Academy of Paediatrics clinical guidelines.**1**

**Table 1.** American Academy of Paediatrics clinical guidelines.**1**

|  |  |
| --- | --- |
| **History*** Frequent snoring (≥3 nights/week)
* Laboured breathing during sleep
* Gasps/ snorting noises/observed episodes of apnoea
* Sleep enuresis (especially secondary enuresis)
* Sleeping in a seated position or with unusual sleeping posture
* Cyanosis
* Headaches on awakening
* Daytime sleepiness
* Attention-deﬁcit/hyperactivity disorder
* Learning problems
 | **Physical examination*** Underweight or overweight
* Tonsillar hypertrophy
* Adenoidal facies
* Micrognathia/retrognathia
* High-arched palate
* Failure to thrive
* Hypertension
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Physical Examination

Affected children may speak with hyponasal speech or rely on mouth breathing. Some children may be obese whereas others may fail to thrive**16,17**. It is hypothesised that the latter is explained by the increased work of breathing associated with abrupt arousal from apnoeic states depletes the child of necessary energy for growth and development**16**.These children have been shown to thrive following adeno-tonsillectomy for OSAS **6, 18.**

A thorough head and neck exam is an essential part of the evaluation in a child with suspected OSAS. The child should be inspected for craniofacial anomalies such as retrognathia, micrognathia; adenoidal facies (decreased nasal airflow, mouth breathing); nasal congestion. Children should be evaluated for oropharyngeal crowding; noting tonsil size and using tools such as the Mallampati score in Figure 2**19**.

**Figure 2: The Mallampati classification for difficult laryngoscopy and intubation**



OSAS is associated with systemic hypertension and less frequently with pulmonary hypertension, warranting a blood pressure measurement and cardiac evaluation in each case of suspected OSAS**20, 21** .

Radiographs of the posterior nasal space may reveal the degree of adenoidal enlargement. However, these X-rays are often taken in the standing position in the awake state and take no account of the dynamic changes in airway tone during sleep, especially REM.

Polysomnography

Polysomnography (PSG) is the gold standard investigation for the diagnosis of sleep disordered breathing. Full PSG requires elective admission to hospital with an overnight-PSG is the preferred choice, as greater possibility of sufficient duration of sleep to occur, with a likelihood of capturing periods of rapid eye movement (REM) where hypoventilation is most likely to occur. Multiple channels record variables including video montage, nasal and oral airflow, abdominal and chest wall movements, end-tidal CO2, oxyhaemoglobin saturation, continuous EEG, continuous ECG and a snore microphone. The evaluation permits the detection apnoeic and hypopnoeic events, hypoventilation, arousals, snoring, alteration in body postion and limb movements. Montage is scored using the American Acaedemy of Sleep Medicine Criteria (2007).

The most commonly quoted variable is the apnoea-hypopnea index (AHI) which consists of all the scored apnoeic and hyponoeic events divided by the total sleep time and is expressed in events per hour. An AHI of less than 1/hr is normal. Those between 1-5/hr are termed equivocal and will comprise of some patients who will require intervention. Values greater than 5/hr are abnormal and many of these patients will require intervention. An AHI of greater than 10/hr is considered severe.

**Management**

The decision to initiate treatment is on an individual basis based upon the clinical picture and the risks and benefits involved. Important determinants include age, sleep study abnormalities and any underlying medical conditions related to OSAS**1**.

Adenotonsillectomy is the first line therapy in children who suffer from OSAS with adenotonsillar hypertrophy. Adeno-tonsillectomy may also be the initial treatment for children with multifactorial OSAS who present with sizeable adenotonsillar tissue hypertrophy. While not correcting the main aetiologies, clinically it has been shown to improve obstructive signs in certain cases**23**.

Continuous positive airway pressure [CPAP] or bilevel positive airway pressure [BPAP] can be used where adeno-tonsillectomy is contraindicated or has failed as a primary treatment**1**. The main inhibitory factor in the use of positive airway pressure lies in the lack of compliance**24**. Education, training, and close follow-up may tackle this issue.

Other therapies

Avoidance of environmental pollutants and allergens is advocated in the presence of allergic rhinitis causing nasal congestion and increased airway resistance**1**. Weight loss is recommended in obese children but there have been no studies in the paediatric population advocating this; thus we must rely on evidence, based on adult OSAS studies. In selected cases of OSAS associated with maxillary contraction (high arched palate, crossbite) orthodontic interventions have been successful in reducing the apnoea-hypopnoea index in affected individuals but further trials are required to identify the appropriate treatment group**25**. Intranasal corticosteroids have demonstrated effective treatment response in children with moderate to severe OSAS unsuitable for adenotonsillectomy as well as in mild OSAS where adenotonsillectomy is not warranted**26**. In a randomised double blind trial of six weeks administration of intranasal budesonide versus placebo in children with mild OSAS, there was significant improvement in PSG measurements, with a sustained benefit lasting 8 weeks post therapy discontinuation**27**. Small studies have evaluated leukotriene receptor antagonist and have concluded that montelukast either alone or in conjunction with intranasal corticosteroids modestly decreases AHI.

**Conclusion**

Adeno-tonsillectomy has been the main stay in treatment for OSAS and associated adeno-tonsillar hypertrophy. In a multicentre retrospective study in 2006, where 578 children were evaluated post-adenotonsillectomy, 90% of the cohort showed a decreased apnea-hypopnea index. However, only 28% had normal apnea-hypopnea index at follow up. The study determined that age (>7 years old) and BMI were principal determinants for persisting OSA, highlighting the importance of careful review of all possible causes of OSAS**28**. Positive airway pressure therapy has been evaluated by several studies and has shown improvement in signs and symptoms as well as improvement in sleep study results in at least 85% of children**29,30,31,32**.

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