INTRODUCTION

Obstructive sleep apnea (OSA) is the most common sleep-associated breathing disorder in children and can be seen from infancy through adolescence. It is increasingly recognized in children, and is a distinct disorder from that seen in adults, especially in regards to risk factors, symptoms, diagnostic criteria on polysomnography, and management. This review will summarize the key features, diagnosis and management of OSA in children.

OSA is characterized by partial (obstructive hypopnea) or complete (obstructive apnea) obstruction of the upper airway. Usually this is associated with oxygen desaturations, gas exchange abnormalities and disrupted sleep.1 The gold standard test to diagnose OSA is with a polysomnogram (PSG).

The reported prevalence of OSA in children is 2-4%2,3 and occurs at a peak age of 2-8 years. However, snoring, which is a key symptom of OSA occurs habitually in approximately 3-12% of pre-school aged children,4 and this is referred to as primary snoring. Snoring may be defined as noisy breathing caused by turbulent airflow throughout the upper airway.

OSA may be even more prevalent in certain syndromes and malformations (see table 1), conditions which may also be associated with dental problems.

PATHOPHYSIOLOGY

During sleep it is normal for a child to have a decrease in minute ventilation and an increase in upper airway resistance. This results in a reduction in the partial pressure of oxygen in arterial blood by 2-5 mmHg and an increase in carbon dioxide by 3-7 mmHg.5 The pathophysiological characteristics that lead to obstruction may be divided into anatomical and neuromuscular factors.

ANATOMICAL FACTORS
The cross-sectional area of the upper airway tends to be smaller in children with OSA. In addition, there is a dynamic component, in that there is a change in the size of the airway between inspiration and expiration, especially at the level of the nasopharynx.6

The most common cause of upper airway narrowing is hypertrophy of the adenoids and tonsils, a significant risk factor for OSA. Other factors include increased fat tissue around the airway, tongue and pharynx in children who are obese. In a similar fashion, in children with mucopolysaccharidosis (MPS), accumulation of glycosaminoglycans in the soft tissues surrounding the airway causes airway narrowing. There are numerous craniofacial anomalies specifically associated with airway narrowing including midfacial hypoplasia, retrognathia, micrognathia, narrow maxillary arch, nasoseptal obstruction and macroglossia.7

NEUROLOGICAL FACTORS

Neuromuscular regulation of upper airway tone is an important factor in OSA and obstruction is seen during sleep (or sedation) because of loss of this upper airway tone.

The pharyngeal dilators involved in patency of the airway include genioglossus, the posterior arytenoids, tensor palatini and the alae nasae. They are innervated both centrally via the respiratory control centre of the medulla and locally via mechanoreceptor reflexes. Global alterations in muscle tone, for example in children with hypotonia or hypertonia, can affect the neuromuscular tone of the pharyngeal dilators. Any focal insult or space-occupying lesion affecting the brainstem can result in sleep-disordered breathing, most commonly seen with Arnold-Chiari malformations or brainstem tumors. Further, palatal muscle function may be abnormal in children with a cleft palate, even after surgery. Likewise, children with achondroplasia with a small foramen magnum have compression of the brainstem and upper cervical spinal cord which will also affect function of the airway.

Drugs and toxins may affect both neuromuscular tone and respiratory drive. Of particular concern are opiates, benzodiazepines, chloral hydrate, general anesthetics, and alcohol.

In the majority of otherwise well children there may be a single etiologic factor for OSA. This is usually adenotonsillar hypertrophy in younger children and obesity in teenagers. However in many complex or syndromic patients there are multiple anatomical and neuromuscular factors that interplay predisposing to OSA. A typical example would be a child with Down syndrome, who has adenotonsillar hypertrophy, macroglossia, midface hypoplasia and generalized hypotonia.

CONSEQUENCES OF UNTREATED OSA

Untreated OSA may lead to cardiovascular, metabolic and neurobehavioural complications. In the past children with severe OSA presented with failure to thrive, due to increased metabolic requirements and interruption of growth hormone production. OSA is an independent risk factor for cardiovascular and metabolic complications including hypertension,8,9 changes in left ventricular mass10 and the metabolic syndrome.11,12 Reported neurobehavioural consequences include poor school performance, learning deficits, change in mood, inattention and ADHD-like behaviors.

SYMPTONS AND SIGNS OF OSA

Parents and children may notice both daytime and night-time symptoms due to OSA (see tables 2 and 3). Symptoms may worsen during an upper respiratory infection. In the older child, whose sleep is not always witnessed by the parents, the predominant complaint might be daytime sleepiness.
Key features in the past medical history would include any risk factors for OSA, for example, the conditions listed in table 1. Children with OSA are at risk of upper airway problems when they receive sedation or muscle relaxants and so a history of anesthetic complications is important.

Several questionnaires addressing symptoms of OSA have been developed to try to identify which children who snore are likely to have OSA. Unfortunately these questionnaires are not able to reliably differentiate children with primary snoring from those with OSA.

**CLINICAL EXAMINATION**

Documentation of weight and height on a growth chart will identify failure to thrive and obesity. The World Health Organization growth charts adapted for Canada are recommended. Failure to thrive may be defined as when a child’s weight for age is below the 5th percentile, or when the weight crosses two major percentile lines. Obesity may be defined as body mass index (BMI) greater than the 95th percentile for age.

Examination of the face should include inspection for mid-face hypoplasia, retrognathia or micrognathia, and mouth breathing. Testing of nasal airflow indicates the degree of nasal patency, as well as inspection of the nasal mucosa for inflammation.

Examination of the oropharynx should include assessment of the size the tonsils (Figure 1). The Mallampati classification (Figure 2) is based on the structures visualized with maximal mouth opening and tongue protrusion in the sitting position. Cleft palate, particularly submucosal clefts and bifid uvula, should be identified. Cardiovascular, respiratory, abdominal and neurological examination should be completed but are usually normal.

**INVESTIGATIONS**

Polysomnography is the gold standard test for diagnosis of OSA and involves recording multiple variables during sleep, overnight in a sleep laboratory. In some children with symptoms and signs indicating adenotonsillar hypertrophy, a consultation by ENT may be appropriate prior to a PSG. PSG is clearly not possible in all patients with suspected OSA due to resource limitations and clinical criteria are often used to proceed to adenotonsillectomy in children with suspected OSA.

**POLYSOMNIOGRAPHY**

PSG recording requires a dedicated sleep laboratory with paediatric-trained sleep technologists, with or without respiratory therapists for management of non-invasive ventilation. Younger children are accompanied by a parent, who sleep in the same room.

**PSG MONITORING**

Sleep state is determined by the combination of EEG leads, eye movements and muscle tone. Respiratory recordings include chest and abdominal wall movement, airflow at the nose and mouth, oxygen saturations, and either transcutaneous or end-tidal carbon dioxide measurements. Heart rate is recorded using an EKG as arrhythmias may be associated with OSA. All children are monitored with a video enabling direct visualization of the head, neck and chest. A 2 minute screen of a PSG in a child is shown in Figure 3.

**PSG SCORING**
The principal respiratory events recorded are obstructive and central apneas.

An obstructive apnea (OA) event is scored when airflow decreases by at least 90% from baseline with chest and/or abdominal motion throughout the entire event; the duration of which is at least a minimum of two baseline breaths. An obstructive hypopnea (OH) event is scored when airflow decreases by at least 50% from baseline, the duration of which is at least a minimum of two baseline breaths. The OH event must be accompanied by either (i) a minimum 3% drop in oxygen desaturation, (ii) an arousal, or (iii) an awakening. A central apnea is defined as a cessation of airflow with an absence of respiratory and abdominal effort for a minimum of 20 s or of the duration of two prior baseline breaths in which case the event must be accompanied by (i) a minimum 3% drop in oxygen desaturation, (ii) an arousal, or (iii) an awakening. OSA severity is graded according to accepted clinical criteria; the obstructive apnea-hypopnea index (OAHI) is the number of obstructive apneas and hypopneas per hour during sleep. OAHI of <1.5 is considered normal, OAHI from 1.5 to <5 is mild OSA; OAHI from 5 to <10 is moderate OSA and OAHI >10 is considered severe OSA.1

**MANAGEMENT**

Management strategies for OSA depend on the underlying etiologies and may include medical, surgical and non-invasive mechanical ventilation treatments and/or non-invasive mechanical ventilation.

**SURGICAL OPTIONS**

Adenotonsillectomy has been shown to significantly improve OSA in many children and the American Academy of Pediatrics has recommended this as the first-line therapy. Adenotonsillectomy results in improvements in the frequency of obstructive events, oxygen saturations, night-time awakenings and sleep efficiency.

Despite the improvements seen, adenotonsillectomy is not successful in all children, as this is just one risk factor for OSA. Repeating a PSG after tonsillectomy should be considered, especially in children who are older, obese, asthmatic, or who have severe OSA.

**NON-INVASIVE VENTILATION**

Continuous positive airway pressure (CPAP) delivered through a nasal or facial mask is increasingly used for the treatment of OSA, especially when adenotonsillectomy has not been successful, or when there are other factors contributing to upper airway obstruction. Numerous studies have shown that CPAP is safe, well-tolerated and effective in all ages. For many obese patients CPAP is the main long-term therapy and is effective when used.

**MEDICAL THERAPY**

Nasal congestion or obstruction is a significant factor for OSA and treatment of allergic rhinitis may be effective in alleviating symptoms. Nasal steroids and oral montelukast (trade name singulair) may be effective in reducing symptoms in mild OSA.

**WEIGHT LOSS**

Increasingly, obesity is an important causative factor in OSA. Reduction of BMI is likely to result in improvement in OSA and these children should be enrolled in a multi-disciplinary weight reduction program.
However weight reduction may take place over the long-term and children with OSA may require treatment in the interim with CPAP.

RAPID MAXILLARY EXPANSION

Rapid maxillary expansion describes the use of a personalized oral jaw-positioning appliance for children with OSA and dental malocclusion. These devices apply pressure to widen the maxilla, increasing the size of the nasal cavity directly, and the oropharyngeal space by repositioning of the tongue. Small studies show improvement in symptoms and PSG indices.25

WHICH CHILDREN SHOULD BE REFERRED TO A SLEEP CLINIC?

OSA, ideally should be diagnosed based on clinical and PSG criteria. However, a healthy child with typical symptoms of OSA may be referred to ENT for an adenotonsillectomy, without necessarily obtaining a PSG. Health professionals should have a low threshold for referring children with Down syndrome, obesity, craniofacial malformation and other children deemed to be at high-risk for OSA.

SUMMARY

OSA is increasingly recognized in children (2-4%) and is seen in both otherwise healthy and medically complex children.

OSA can result in adverse neurobehavioral and cardiovascular consequences.

Polysomnography is required for diagnosis.

Treatments are effective and may include: adenotonsillectomy, non-invasive ventilation, medical therapies and rapid maxillary expansion.

Health professionals need to be aware of the signs and symptoms of OSA in children to ensure prompt diagnosis and treatment. OH

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Oral Health welcomes this original article

REFERENCES

1 Academy of Sleep Medicine. International classification of sleep disorders, 2nd ed, American Academy of Sleep Medicine, Westchester, 2005.


Photos

Larger photo & full caption
File size: 365.2 KB (900px X 814px)
Caption: Figure 1.

Larger photo & full caption
File size: 470.5 KB (900px X 1222px)
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