Obstructive Sleep Apnea

Developed by Steffany Charles and Dr. Joanna MacLean for PedsCases.com.
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Hi, my name is Steffany Charles, and I am a medical student at the University of Alberta. This podcast will review the diagnosis and management of obstructive sleep apnea in children. I’d like to thank the co-author of this podcast: Dr. Joanna MacLean, a pediatric Respirologist and Sleep Specialist at the Stollery Children’s Hospital and the University of Alberta, in Edmonton, Canada. For a broader overview of sleep problems in children, please listen to our PedsCases podcast on Sleep Disorders!

After listening to this podcast, the learners should be able to:
1. Understand the pathophysiology of OSA and common causes in the pediatric population
2. Learn the steps to evaluating OSA in a pediatric patient
3. Evaluate the role of a PSG (sleep study) in diagnosing OSA
4. Explain the treatment options for OSA

Case Presentation

Let’s start with a case…
Nathan is a previously healthy 9-year-old boy, who presents for his yearly check-up at a community pediatrician’s office. His parents comment that they have noticed him snoring during sleep. He has also started taking naps during the day and has had difficulties concentrating in school. His parents are concerned about his quality of sleep and are wondering if you have any suggestions of what to do next. How do you proceed?

Introduction

Obstructive sleep apnea (abbreviated as OSA) is a common issue in children. Obstructive sleep apnea is the most common sleep related breathing disorder. It is characterized by intermittent complete or partial upper airway obstruction, prolonged partial upper airway obstruction, or both intermittent and prolonged obstruction that disrupts normal sleep, breathing or both. Upper airway resistance syndrome, a term used to describe snoring with no identifiable airflow obstruction but increasingly negative esophageal pressure resulting in arousal from sleep, is included under the term OSA as the pathophysiology does not appear to differ from OSA. While snoring and mouth breathing during sleep are hallmarks of OSA, most children with these...
symptoms do not have OSA so it is important to understand the clinical manifestations of OSA and how it is diagnosed in children. Differences in the pathophysiology of OSA in children also means that the treatment approach to OSA in children is different than that for adults.

**Why does OSA occur and who is at risk?**

Let's start with some definitions….

A Apnea is the cessation of breathing, defined in pediatric sleep medicine as two or more missed breaths. Hypopneas are partial cessations in breathing where the amplitude of breathing decreases but breathing does not stop. During obstructive apneas and hypopneas, airflow stops or is reduced while inspiratory effort continues but is ineffective due to obstruction of the upper airway. This is in contrast to central apneas and hypopneas where airflow is stopped or reduced as is inspiratory effort.

OSA results from complete or partial collapse or obstruction of the upper airway during sleep, eventually leading to periods of cessation of airflow with on-going breathing effort. Upper airway obstruction can be due to a variety of structural and neuromotor abnormalities. In addition, muscle tone in the upper airways is decreased during sleep, particularly during rapid eye movement or REM sleep. These obstructive events rouse the patient from sleep. Frequent disruptions through the course of the night impair sleep quality and can lead to fatigue. Long-standing, severe OSA can lead to serious problems for children including failure to thrive due to increased energy requirements during sleep, systemic hypertension and pulmonary hypertension. These complications further emphasize the importance of appropriate evaluation and management of this condition.

The prevalence of OSA in the pediatric population is 1 to 4 percent. The incidence is equal among males and females. OSA can present at any age, however the peak incidence is commonly seen in children between two and six years of age due to the rapid growth of tonsil and adenoid tissues compared to the underlying airway, resulting in a relative size discrepancy. In children who are otherwise healthy, adenotonsillar hypertrophy and obesity remain the two major risk factors for OSA. Other risk factors for OSA include a family history of OSA as well as conditions that are associated with a smaller airway (like craniofacial syndromes), conditions where muscle tone is lower (like neuromuscular disease or Down syndrome), conditions associated with altered response to respiratory gases (like Prader-Willi syndrome) or where more than one of these risk factors are present.

**Screening & Diagnosis**

Routine screening for OSA should be done during healthy visits to a doctor’s office. This can include questions about snoring to find out if this occurs consistently at least 3 times per week. A questionnaire, such as The Pediatric Sleep Questionnaire, published by Chervin et al may also be helpful as a screening tool.

If OSA is suspected in a child, then appropriate diagnostic evaluation should be conducted. This should include:

- A focused history
• Appropriate physical examination, including evaluation of the upper airways
• Consideration for referral for a polysomnography (overnight sleep study).

**Focused History**

Your general patient history should include questions focused on the child’s normal bedtime routine and sleeping environment. This includes the typical number of hours of sleep per night, daytime sleep, and things that disrupt sleep like caffeine intake and electronics in the bedroom.

It is important to ask parents about their child’s night time symptoms. The most common symptom is snoring, however the majority of children who snore do not have OSA. Other nocturnal symptoms include witnessed apneas, mouth breathing, restless/agitated sleep, diaphoresis (or excessive sweating), and sleeping in unusual positions, such as with their neck extended. Older children may present with primary or secondary nocturnal enuresis (or bedwetting) and excessive sleep need.

In addition, children could be experiencing daytime symptoms as a consequence of poor sleep. These include: morning headaches, daytime somnolence, poor functioning at school or behavioural concerns, such as irritability and inattentiveness.

Finally, questions about the child’s past medical history, including craniofacial or cardiopulmonary syndromes, developmental concerns as well as a family history of obstructive sleep apnea are of relevance.

**Physical Exam**

Most children who present with OSA have normal physical exam findings. Begin with overall general inspection and vital signs, specifically assessing the child’s blood pressure for evidence of systemic hypertension. Next assess the child’s growth, through plotting their height and weight on a standard growth chart. Chronic and severe OSA can limit normal growth and obesity is a major risk factor for OSA.

Inspection of the head and neck region can reveal craniofacial abnormalities that impact the size of the upper airways. This includes a small chin, or micrognathia, which is best assessed from a side view of the face. Mouth-breathing, a resting mouth open posture, and hyponasal speech, may be signs of adenotonsillar hypertrophy. It is important to examine the nares for signs of mucosal irritation or swelling which can indicate chronic nasal congestion. Examination of the oropharynx should include assessment of tonsil-size, specifically noting the degree of oropharyngeal crowding. The Brodsky score is used to assess tonsil size relative to the space of the oropharynx. It is a graded score from 0-4: Grade 0 is tonsils absent; Grade 1: Tonsils hidden within tonsil pillars; Grade 2: Tonsils extending to the pillars; Grade 3: Tonsils are beyond the pillars; Grade 4: Tonsils extending to midline (often described as tonsils kissing). Significant tonsillar hypertrophy is present with grade 2 and above. Of note, this scale only assesses tonsils in the lateral dimension. Tonsillar extension in the anterior-posterior dimension and assessment of adenoidal obstruction may require endoscopy.

Finally, a neurological examination is prudent given the role that hypotonia can play in contributing to OSA symptoms.

Polysomnography

An overnight attended PSG, also known as a sleep study, remains the gold standard for diagnosing OSA. It definitively identifies obstructive events and can quantify the severity of OSA. A sleep study is performed in a sleep laboratory where multiple sensors are used to monitor the child for sleep-disordered breathing. These include EEG and chin EMG to define sleep, and nasal and oral airflow sensors, snoring microphone, respiratory effort signal, pulse oximetry, and CO2 monitoring to define breathing. Body position and leg movements can also be monitored.

Respiratory events are then scored, which include:

- Apneas: defined as > 90% decrease in airflow signal
- Hypopneas: defined as > 30% decrease in airflow signal
- Oxygen desaturation events; defined as a 3% or greater drop in oxygen saturation

Apneas and hypopneas are combined to calculate the apnea-hypopnea index (AHI), which is the total number of apneas and hypoapneas per hour of sleep.

Other parameters observed during a sleep study also include:

- Oxygen parameters including minimum, mean, maximum and percent time <90% saturation.
- Carbon dioxide parameters including hypoventilation defined by CO2 greater than 50 mmHg for greater than 25% of sleep time.

The diagnostic criteria for pediatric OSA requires both clinical and sleep study findings; defined by the American Academy of Sleep Medicine (AASM).

- **Clinical Criteria:** Presence of 1 or more of:
  1. Snoring
  2. Laboured, paradoxical or obstructed breathing during sleep
  3. Sleepiness, hyperactivity, behavioural or learning issues

- **Sleep Study Criteria:** Presence of one or both of the following:
  1. AHI > 2 events/hour
  2. Pattern of obstructive hypoventilation: at least 25% of total sleep time with PaCO2 > 50 mm Hg with snoring, flattening of nasal pressure, or paradoxical thoracoabdominal motion

Severity is determined by the AHI. A higher AHI indicates more severe OSA. A rough guide that may not relate to complications or treatment response is:

- **Mild:** AHI 1–4.9 events/h
- **Moderate:** AHI 5—9.9 events/h
- **Severe:** AHI > 10 events/h

Who gets a sleep study? Ideally, every child with clinically suspected OSA should receive a PSG (sleep study), as it remains the gold standard for diagnosing OSA. The importance of objective testing through a sleep study also allows for better prediction of high risk groups where adenotonsillectomy may not alleviate OSA symptoms and surgery would be inappropriate.

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These include children who have disrupted oxygen saturation, a greater risk of complications related to general anesthetic or other reasons for their snoring. However, current infrastructure remains inadequate to allow every child access to a sleep study. Hence, the use of other screening tests may be used in the interim, as these tests are a better alternative to no objective testing at all.

**Alternative Screening Tests**

Clinical history is not a good predictor of OSA so when a sleep study is not readily available, some form of objective testing, is better than none. Options currently available for OSA in children include the use of questionnaires, continuous overnight oximetry, and finally ambulatory limited channel monitoring also referred to as home sleep apnea testing (or HSAT). None of these options should be used to exclude the diagnosis of OSA but their use, along with a clinical history, may increase the correct identification of those children with OSA.

There are two questionnaires that have been validated against polysomnography for the diagnosis of OSA in children. The Pediatric Sleep Questionnaire, was developed by Chervin and colleagues. A study of this scale found that a positive screen correctly classified approximately 85% of a sample of children 2-18 years of age. A 3-item scale developed by Brouilette and colleagues (often referred to as the Brouilette score), correctly classified approximately 95% of children 1-10 years of age. The validation of both scales included a relatively small sample of children and did not include many children with comorbidities or complex medical illness.

Nocturnal oximetry measures both heart rate and oxygen saturation in the blood during sleep. It is an accessible and relatively inexpensive test that can be done overnight at home to detect episodes of oxygen desaturation events that occur throughout the night. The baseline O2 saturation along with the pattern i.e. clustering of desaturation events can be suggestive of a pattern consistent with OSA. However, oximetry alone remains inadequate for a true diagnosis of OSA as it holds a poor sensitivity and specificity compared to a sleep study. This may be due to movement artifact during sleep and OSA symptoms that result in arousals and sleep fragmentation but little oxygen desaturation.

Home sleep apnea test monitors generally include 3 or more cardiorespiratory signals with no direct measure of sleep. This test is valid for use in adults with suspected OSA as a replacement for a sleep study. Studies do show it is technically feasible in school-aged children; however, comparison with a sleep study do not support home sleep apnea testing as routine use in the place of a sleep study in children.

**Treatment**

The treatment for OSA is determined on a case-by-case basis and will depend on the severity of the OSA symptoms, co-morbidities, and patient or family preference. Preventative measures should be taken such as avoidance of pollutants, smoke and other air irritants in the household to minimize any exacerbating factors.

Treatment options include watchful waiting, medical management, surgery and non-invasive ventilation.

First we will consider watchful waiting. A recent large randomized-controlled trial of adenotonsillectomy showed that 50% of children 5-9 years of age who had OSA showed resolution of OSA on a follow-up polysomnography 9 months later with no treatment in the interim. This supports that in many cases, only observation is needed, and OSA may get better on its own. When you decide on watchful waiting, you should reassess symptoms on a regular basis and consider repeat polysomnography.

Next is medical management. Medication for OSA include intranasal steroids and/or a leukotriene receptor antagonist aimed at reducing inflammation in the upper airway as well as potentially reducing adenoid and tonsillar size. While these have been reserved for mild OSA, there is sufficient data to support their use in uncomplicated OSA of greater severity. A trial of 2-3 months is generally sufficient to assess response and determine if additional treatments are needed.

Next we will discuss different surgical options. Adenotonsillectomy remains the first line treatment for children with adenotonsillar hypertrophy. This procedure is not curative in all children however the majority of children will derive some benefit and it may improve the effects of other treatments for OSA post-operatively. Usually adenotonsillectomy is a day procedure, however children at high risk of post-operative complications may require post-operative monitoring in an intensive care environment. These include children less than 2 years of age and those with severe OSA and/or hypoventilation. Other surgical interventions appropriate for select patients include mandibular advancement, maxillary advancement, and supraglottoplasty. Tracheostomy is reserved as a last resort option, primarily in children with severe neurologic problems, or upper airway malformations.

The last option is non-invasive ventilation in the form of CPAP or BPAP. Continuous positive airway pressure (CPAP) therapy, where air is blown into the airway through a mask worn over the nose or face to keep the airway open, is a first line therapy for some children and can also be used if other therapies have failed to improve OSA symptoms. It is highly effective but does require long-term compliance with equipment use. Adjustment in mask and settings are needed with on-going facial change and growth. If high pressures are needed to keep the airway open, CPAP, which delivers a single continuous pressure, may be switch to bi-level positive airway pressure or BPAP where 2 pressures allow a lower pressure during exhalation.

**Conclusion**

*Now, back to the case with Nathan, the 9-year-old boy with a history of snoring during sleep. After a thorough sleep history, you find that he often likes to play on his iPad just before sleep each night and that his father uses a CPAP machine at home during sleep. On exam, he has a BMI greater than the 95th percentile and he has 2+ tonsils on the Brodsky scale. The rest of his examination is unremarkable and he is otherwise healthy.*

*You proceed to counsel Nathan and his family on maintaining good sleep hygiene habits, including minimizing electronic screen-time before sleep. You discuss referral to a local weight management program for children and families with for Nathan and his family. You decide that his presentation makes him a likely a candidate for adenotonsillectomy and so a referral is also made to ENT. Once seen by ENT, an overnight oximetry will be ordered as part of his pre-
operative assessment. In the interim, you decide to have the patient start a trial of intranasal steroids and to follow-up with you in 2-3 months to see if his symptoms have improved.

Take Home Points:
To conclude this podcast, let’s review a few key take-home points:

1. Obstructive sleep apnea is the most common sleep related breathing disorder in children.
2. The most common symptom of OSA is snoring, however the majority of children who snore do not have OSA. Other important symptoms include daytime somnolence and behavior problems. These can be evaluated using a screening questionnaire.
3. Polysomnography is the gold standard for diagnosing OSA.
4. If access to polysomnography is not possible or limited, there is a role for other forms of objective testing such as questionnaires, overnight oximetry, or home sleep apnea testing. Some form of objective testing is likely better than none.
5. The treatment of OSA is determined on a case-by-case basis and includes medications such as intranasal steroids or leukotriene receptor antagonists, surgery such as adenotonsillectomy, and home CPAP.

Thanks for listening!

References:


The A to Zs of Obstructive Sleep Apnea. Presentation presented by Dr. Sherri Katz at CHEO, University of Ottawa. 2017.

