British Journal of Anaesthesia **111** (S1): i83–i95 (2013)
doi:10.1093/bja/aet371

BJA

PAEDIATRICS

Obstructive sleep apnoea in children: perioperative considerations

M. Patino, S. Sadhasivam and M. Mahmoud*

Department of Anesthesiology, Cincinnati Children's Hospital Medical Center, 3333 Burnet Ave, Cincinnati, OH 45229, USA
* Corresponding author. E-mail: mohamed.mahmoud@cchmc.org

Editor's key points

- Obstructive sleep apnoea (OSA) is common in children, and is a source of significant morbidity.
- Adenotonsillar hypertrophy is often associated with OSA, which can lead to perioperative complications.
- Optimal perioperative management of OSA includes preoperative snoring assessment, close respiratory observation and monitoring, and opioid-sparing anaesthetic and analgesic approaches.

Summary. Obstructive sleep apnoea (OSA) has become a major public health concern as its incidence and severity have increased in tandem with the obesity epidemic. In children, OSA is now recognized as a common disorder and can be associated with significant morbidity. OSA belongs to a spectrum of diagnoses known as sleep-related breathing disorders in which the airway is completely (apnoea) or partially (hypopnoea) occluded during sleep despite continued respiratory efforts. This airway obstruction can cause abnormal gas exchange leading to hypoxaemia, hypercapnia, sleep fragmentation, and their attendant physiological and behavioural consequences. The degrees of hypercapnia, hypoxaemia, and upper airway airflow reduction are the primary factors determining the severity of OSA. In young children, adenotonsillar hypertrophy is the most common anatomical abnormality associated with OSA, and adenotonsillectomy is, therefore, the most common surgical intervention. Perioperative complications associated with adenotonsillectomy are more common in children with severe OSA. A thorough understanding of the pathophysiology of OSA, careful and complete preoperative assessment, meticulous intraoperative and postoperative management, and early recognition of potential perioperative complications are essential to optimization of outcomes. The safe anaesthetic management of a child with OSA requires an anaesthetic technique tailored to the underlying aetiology and severity of OSA and the surgical procedure. This review focuses on the epidemiology, pathogenesis, and diagnosis of OSA, and the state-of-the-art and future directions in the perioperative management of children with OSA.

Keywords: adenoidectomy; analgesics, opioid; child; sleep apnoea obstructive; sleep apnoea syndromes; tonsillectomy

Sleep-related breathing disorders (SBD) are a continuum of disorders including primary snoring, upper airway resistance syndrome and obstructive sleep apnoea (OSA). The most severe form of SBD with the highest risk of perioperative complications is OSA.[1] The pathophysiology of paediatric OSA is often multifactorial, with significant contributions from adenotonsillar hypertrophy, obesity, and genetics. Polysomnography is currently the most widely accepted diagnostic modality for OSA, but restricted availability and high cost limit its routine use. Currently, there is no consensus among anaesthetists regarding the best and safest anaesthetic technique for children with significant OSA; there is also a lack of agreement among anaesthetists, surgeons and institutions on specific criteria to identify children with OSA who will benefit from admission to hospital and aggressive postoperative monitoring after surgery. The objectives of this review are to: (i) update anaesthetists with the latest on the epidemiology, pathophysiology, diagnosis, and treatment of OSA, (ii) outline current strategies for the perioperative management of children with OSA, and (iii) analyse current perioperative outcomes in children with OSA undergoing one of the most common surgical procedures in this patient population, adenotonsillectomy.

Epidemiology

In children, the prevalence of OSA is 1–4%; primary snoring is more common, with an incidence as high as 20%.[1] SBD are more common in boys and children who are obese.[2] The incidence of OSA peaks between ages 2 and 8 years and then declines in older children, although a second increase in the incidence of OSA seen in adolescence is associated with obesity.[3 4] This peak corresponds to the age range where adenotonsillar hypertrophy is most commonly observed in children. African-American children have a higher prevalence of OSA.[5 6] Infants whose family history includes OSA or multiple episodes of sudden infant death syndrome are more likely to be diagnosed with OSA than infants from families with no history of OSA

or a single episode of sudden infant death syndrome; this observation suggests a strong association between a family history of OSA and potentially life-threatening infantile OSA.[7]

Pathogenesis

Increased resistance in upper airway during sleep is an essential feature of OSA. Paediatric OSA is often a multifactorial disorder with overlapping contributions from airway narrowing (e.g. adenotonsillar hypertrophy), abnormal airway muscle tone, and genetics predisposing children to obstructed breathing during sleep.[4] Upper airway patency is determined by the interaction between respiratory dynamics, anatomic structures, and neuromotor tone; the degree of patency is determined by the balance between forces acting to collapse the airway (e.g. negative pressure during inspiration; size, shape, and floppiness of pharyngeal structures) and forces acting to maintain airway patency (e.g. pharyngeal dilator muscle tone, stiffness of pharyngeal structures). Suppression of pharyngeal dilator muscle tone is seen during sleep and anaesthesia/sedation; because of the imbalance of forces in the airway, this suppression leads to accentuated airway obstruction in patients with OSA. Therefore, airway obstruction in OSA can be relieved by arousal from sleep, and OSA is characterized by repeated episodes of airway obstruction relieved by arousal, resulting in restless sleep.[8 9] Children with habitual primary snoring often do not have apnoea, hypopnoea, respiratory effort-related arousals, and gas exchange abnormalities because of a compensatory neuromuscular mechanism preventing significant airway obstruction during sleep. The polysomnographic pattern in children with primary snoring shows obstructive hypoventilation and stable increased respiratory effort, but not frank apnoea, hypopnoea, or respiratory arousal.[10] Obstructive events during sleep in children with OSA have an inverse pattern in relation to sleep stage in comparison with that seen in adults with OSA. Eighty per cent of obstructive events in children with OSA occur during rapid eye movement (REM) sleep, whereas 80% of obstructive events in adults with OSA occur during non-REM sleep.[11 12] As a result, obstructive events in children are rare during slow wave sleep.[12] Non-REM obstructive events occur less often in children, but tend to increase in older children, in African-American children, in the lateral position and at low levels of oxyhaemoglobin saturation.[12] Non-REM obstructive episodes leading to arousal occur at higher oxygen saturation than events during REM sleep.[12] Body position during sleep seems to play a role in the degree of obstruction; the supine position is associated with more severe obstruction than the lateral or prone positions. The use of comprehensive respiratory obstruction event profiles may enhance our understanding of the pathophysiology and clinical manifestations, including adverse outcomes, of OSA in children.[12]

Accepted risk factors for OSA in children include adenotonsilar hypertrophy, craniofacial malformations, hypotonia, obesity, midface hypoplasia, macroglossia, retrognathia, micrognathia, and glossoptosis.[13] While adenotonsillar hypertrophy is the major structural factor contributing to the pathogenesis of OSA in younger children, obesity is increasingly recognized as an important contributing factor to OSA in adolescents.[14] The prevalence and severity of OSA are higher in obese children and adolescents, and the severity of OSA parallels the degree of obesity.[15] However, many obese adolescents do not develop OSA, despite having a narrow airway. Recently, Huang and colleagues speculated that obese adolescents without OSA maintain protective upper airway reflexes during adolescent development, whereas those who go on to develop OSA do not.[16] Obese patients experience alterations in respiratory dynamics resulting from reduced functional residual capacity, and also increased tissue mass and pressure in the neck and pharynx leading to airway narrowing. The increased respiratory effort required to initiate inspiration in the face of reduced functional residual capacity and also to overcome upper airway resistance because of narrowing of the pharynx results in a greater degree of negative pressure in the pharynx and upper airway and accentuates airway collapse in the obese patient.

A subgroup of children with OSA associated with Down syndrome, the mucopolysaccharidoses, craniofacial syndromes, and achondroplasia may have obstructions at several levels in the airway.[15 17] Children with craniofacial syndromes, especially those with midfacial hypoplasia, micrognathia, maxillary hypoplasia, or deformation of the cranial base (e.g. Crouzon, Apert and Pfeiffer syndromes), frequently have OSA, often associated with nasal and nasopharyngeal airway obstruction. In patients with Down syndrome, the upper airway obstruction occurs as a result of midface hypoplasia, macroglossia, and muscular hypotonia. In this subgroup of patients, adenotonsillectomy often alleviates obstruction because of adenotonsillar hypertrophy, but may fail to relieve the obstruction at other levels. These patients frequently require more complex and invasive airway surgery such as tongue reduction or lingular tonsillectomy.

Recurrent episodes of abnormal gas exchange resulting in hypoxaemia, hypercapnia, and acidosis in children with moderate and severe OSA can lead to haemodynamic co-morbidities such as pulmonary hypertension and cor pulmonale; these are usually reversible with resolution of OSA. Chronic hypoxaemia is an independent risk factor for left ventricular hypertrophy.[18] These cardiovascular manifestations arise from endothelial dysfunction from an increased sympathetic tone and inflammatory response facilitated by increases in levels of C-reactive protein.[19] In children with OSA, these inflammatory markers seem to be involved with metabolic and neurocognitive/neurobehavioural manifestations of OSA.[20]

Diagnosis

A description of the signs and symptoms of OSA was published in the *British Medical Journal* in the 19th century: 'the stupid-lazy child who frequently suffers from headaches at school, breathes through his mouth instead of his nose, snores and is restless at night, and wakes up with a dry mouth in the morning, is well-worthy of the solicitous attention of the school medical officer'.[21] While our medical and