The impact of pediatric obstructive sleep apnea on ambulatory surgery

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Abstract

The purpose of this paper is to review pediatric obstructive sleep apnea syndrome (OSAS) with an emphasis on ambulatory adenotonsillectomy. Difficulties in establishing a diagnosis by clinical criteria alone are discussed. Diagnostic tests to establish a diagnosis of OSAS are discussed. The child with severe obstructive sleep apnea is at increased risk for post-adenotonsillectomy respiratory morbidity. The perioperative management with a focus on the ambulatory candidate is discussed.

The child with OSAS presents a challenge to ambulatory surgery because of the high prevalence of OSAS, difficulty in establishing a diagnosis of OSAS and the increased risk of respiratory morbidity.

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Obstructive sleep apnea syndrome (OSAS), affecting 1–3% of children, is characterised by sleep fragmentation and nocturnal episodic asphyxia [1]. The prevalence may be as high as 10–20% in children who habitually snore. The peak incidence of childhood OSAS occurs between 2 and 5 years coinciding with maximal adenotonsillar hypertrophy [2]. In children, as in adults, OSAS has a negative impact on quality of life, somatic growth, cardiovascular health, neurocognitive function and behaviour [3,4]. The impact of adult OSAS on the practice of anesthesia has recently been reviewed [5]. Whereas it is predicted that the next decade will see a 5- to 10-fold increase in adults with OSAS undergoing surgery [5], the last two decades have already seen a dramatic change in the indication for adenotonsillectomy from one of chronic tonsillitis to obstructive breathing. Today, 80% of children presenting for adenotonsillectomy have a history of sleep associated obstructive breathing [6,7]; an impressive statistic when one considers the annual caseload. This review discusses the impact of OSAS on the practice of pediatric anesthesia with an emphasis on the management of ambulatory adenotonsillectomy.

Although there is no centralized reporting mechanism to record mortality following adenotonsillectomy, mortality following adenotonsillectomy in children is estimated to be 1 per 16,000 or 0.6 per 10,000 [8]. Consensus opinion [1,9] suggests that a significant morbidity following adenotonsillectomy for OSAS exists. The risk of post-adenotonsillectomy respiratory morbidity in the unselected pediatric population is less than 1% [10–12]. A diagnosis of severe OSAS increases the risk of respiratory morbidity following adenotonsillectomy by a factor of at least 20 [13–19]. The potential risk in children with severe OSAS has provoked quite a lot of controversy. The root of this controversy lies in the difficulty in making a diagnosis of OSAS and establishing its severity and the cost implications to the health care providers of both pre-operative screening and post-operative care, given the magnitude of the annual pediatric caseload for adenotonsillectomy. Recent editorials suggest that anesthesiologists should assume a leading role in resolving this controversy [20,21].
1. Establishing a clinical diagnosis of OSAS

Most of the published literature has focused on identification of high-risk patients with OSAS, in order to exclude them from ambulatory programs. Many patient characteristics predispose the child to OSAS. African-Americans have a three-fold higher incidence of OSAS [22-23]. In addition during awake obstructive events, the African-American child desaturates more profoundly compared with Latino and Caucasian children with OSAS [24]. Unlike the adult presentation, there is no gender difference in the incidence of OSAS in children. Many coexisting medical conditions are associated with OSAS including Achondroplasia, Down syndrome and other craniofacial syndromes characterized by micrognathia or maxillary hypoplasia, chromosomal abnormalities; hypotonia and neurologic disorders; asthma; prematurity and obesity [1,25–30]. The Prader–Willi syndrome presents the dual problems of obesity and hypotonia [31].

The trilogy of sleep fragmentation, nocturnal intermittent hypoxia and episodic hypercapnea which is characteristic of OSAS affects multiple organ systems. Somatic growth is affected and either failure to thrive or obesity may be exist [30,32,33]. Neurocognitive dysfunction and a history of daytime sleepiness, behaviour problems and poor school performance support a diagnosis of OSAS [3,34,35]. Evidence for a link between learning and OSAS is found in the experimental rat model. Intermittent hypoxia is associated with long-term sequelae on the hippocampal functions of learning and memory. In addition, the arousal index has a significant influence on the prefrontal cortical functions influencing behaviour and attention [36,37].

It is well known that young age less than 3 years, is an independent risk factor for airway complications following adenotonsillectomy [38]. The combination of young age, an associated medical condition and OSAS results in a high risk of post-adenotonsillectomy respiratory morbidity [13,14,17,18].

Although a history of an associated medical condition, obesity, failure to thrive, behavioural problems, poor school performance and sleep disordered breathing suggest a diagnosis of OSAS, the fact remains that the majority of patients, including children, with OSAS are undiagnosed [2,5]. The OSA-18 clinical score is a questionnaire that could be administered in advance of the surgical date. Developed in 60 non-obese children, it presents a scoring system to diagnose OSAS by clinical criteria [4]. It assesses the frequency of symptoms known to be associated with OSAS including sleep disturbance, physical symptoms, emotional distress, daytime dysfunction and parental concern. When applied to a non-obese, pediatric population, there was a high correlation between the total score and the respiratory distress index, analogous to the apnea hypopnea index (AHI), and with the degree of adenotonsillar hypertrophy. The practice guidelines for perioperative management of patients with obstructive sleep apnea [9] propose a clinical scoring system based on physical characteristics, sleep disordered breathing and daytime somnolence. However, it is important to recognize that a diagnosis based on clinical scores alone will correlate poorly with findings on polysomnography [33,39–42]. Scoring systems based on clinical criteria will result in both false positive and negative diagnoses for OSAS.

Severe OSAS is associated with important cardiovascular pathophysiology including pulmonary and systemic hypertension, both right and left ventricular dysfunction [43,44], recurrent pulmonary aspiration [45] and abnormalities of ventilatory control [46] including an increased sensitivity to opioid respiratory depression [47]. These sequelae of OSAS are difficult to diagnose in the pre-operative examination. Conventional clinical assessment in a group of children with sleep disordered breathing and witnessed apnea did not suggest right ventricular dysfunction. However a reduced right ventricular ejection fraction was present in one-third of the children [48]. At present less than 10% of children are being tested for OSAS prior to adenotonsillectomy [7]. The challenge is to identify the otherwise well apparent-American Society of Anesthesiology (ASA) class I child with obstructed breathing during sleep who has severe OSAS.

2. Diagnostic testing for OSAS

2.1. Testing during sleep

The gold standard for diagnosis of OSAS is polysomnography involving the assessment of sleep and breathing from a computerized recording of electroencephalographic and cardiorespiratory signals with the simultaneous video recording in the sleep laboratory. The polysomnogram in OSAS shows episodic obstructive respiratory events which, in children, typically cluster during active, rapid eye movement (REM) sleep. Unlike adult OSAS, sleep architecture is preserved and REM content is normal [48,49]. Polysomnography is expensive, labour intensive and the waiting lists are prohibitive. Abbreviated cardiopulmonary recordings at home present a less costly option [50–52].

The diagnostic thresholds for OSAS by sleep laboratory criteria are based on statistical deviation from normative sleep parameters. Sleep data recorded in an asymptomatic, normative population of children indicates that an apnea index greater than 1 apnea per hour is abnormal [1,53], a threshold which is lower than the diagnostic threshold of 5–10 apneas per hour in adult sleep laboratories [53,54]. A nadir saturation, defined as the lowest saturation recorded during the night’s sleep, which is below 95% is statistically abnormal in a normative, asymptomatic population of children. This value is similar to the diagnostic threshold of 90% in adult sleep laboratories [53]. The upper airway resistance syndrome (UARS) is associated with long periods of hypoventilation during sleep and is considered an OSAS equivalent in children [33,48]. An AHI in excess of 10 events per hour and
a nadir saturation less than 80% are the thresholds which are predictive of increased risk for post-operative respiratory morbidity [13,14,17–19,55]. These thresholds are consistent with a diagnosis of severe OSAS.

We [17,19,52,56] and others [55,57] have explored the diagnostic potential of oximetry because it is widely available and pre-operative desaturation during sleep correlates with the AHI. Nocturnal oximetry has a high specificity albeit imperfect sensitivity for a diagnosis of OSAS by polysomnographic criteria [57] and is also a good predictor of peri-operative risk [17,19]. The McGill oximetry score was developed to stratify OSAS severity (Fig. 1). A positive McGill oximetry score demonstrates at least three clusters of desaturation below 90% during sleep. This pattern in addition to the nadir saturation has a positive predictive value for a diagnosis of OSAS in otherwise healthy children. The McGill oximetry score is also predictive of post-operative risk. Oximetry reporting by trend analysis is easily learned [19]. Furthermore, trend reporting of computerized records is amenable to electronic reporting from telehealth systems.

Fig. 1. Representative nocturnal oximetry trend traces for McGill oximetry scores 1–4 (top to bottom). McGill oximetry score of 1 is an inconclusive study. McGill oximetry score 2, 3 and 4 are distinguished by the nadir desaturation which are 90%, 85% and 80%, respectively. Reproduced with permission from Pediatrics 113:19–25, Copyright 2004 by the American Academy of Pediatrics.

2.2. Testing during wakefulness

Two tests are available to diagnose OSAS in awake children. The presence of a compensatory metabolic alkalosis with or without hypercarbia on a capillary blood sample drawn during wakefulness is consistent with recurrent nocturnal hypercarbia and supports a diagnosis of severe OSAS [17,19].

Acoustic pharyngometry is a non-invasive test to measure the cross-sectional area of the pharynx in awake children. In addition, the collapsibility of the pharynx can be assessed by measuring the cross-sectional area before and after the application of topical anesthesia. Pharyngeal cross-sectional area is smaller in children with OSAS compared with controls [58,59]. Whereas pharyngeal cross-sectional area decreased 5% in a control population, in children with OSAS, it decreased 40%, supporting the notion of increased upper airway collapsibility, in OSAS [59–61].

3. Anesthetic management of children with OSAS

3.1. Pre-operative preparation

The single most important pre-operative preparation is to identify the child with severe OSAS, since this diagnosis will exclude the child from ambulatory programs. However the reality is that only a minority of children are tested for nocturnal desaturation prior to adenotonsillectomy [7].

Given the poor specificity for OSAS severity of the clinical scoring systems, and the endorsement, by the American Society of Anesthesiologists, of the clinical diagnostic criteria within the context of practice guidelines for OSAS [9], pre-operative testing for OSAS may in future prove cost effective.

Treatment of OSAS is associated with an improvement in systemic hypertension, myocardial function, and normalization of carbon dioxide responsiveness [34,44,51,62–65]. However there is no review of the role of pre-operative optimization in children with OSAS. The child who has a worsening of obstructed breathing due to an upper airway infection presents a dilemma. These children may have life threatening airway obstruction with profound desaturation during sleep. To proceed with adenotonsillectomy increases the risk of serious respiratory morbidity [19]. An alternate option is to treat pre-operatively with antibiotics, steroids [66] and the insertion of nasal trumpets during sleep while hospitalized in an intensive care setting. Our experience with one child managed in this fashion reported an improvement in the airway obstruction, resolution of the respiratory acidosis and an uncomplicated adenotonsillectomy [17]. Some of these children may be candidates for delayed adenotonsillectomy following a course of antibiotics and steroids at home. However, our experience indicates that there may be a risk that some may be lost to follow-up until the next infectious process compromises respiration.
3.2. Conduct of anesthesia

The influence of anesthetic technique on reducing peri-operative respiratory morbidity post-adenotonsillectomy for OSAS is largely unreported. The risk of post-operative desaturation may decrease if surgery is performed in the morning [67]. Additionally administration of intra-operative atropine decreases the risk of post-adenotonsillectomy desaturation [18]. This decreased risk may involve cholinergic mechanisms which may play a role in both sleep regulation [68], sleep disordered breathing [69] and the function of the genioglossus musculature [70]. The recent trend away from the routine administration of atropine in pediatric anesthesia may need further thought in the management of children with severe OSAS.

First described in 1981, continuous positive airway pressure (CPAP) therapy during sleep normalizes sleep architecture and abolishes nocturnal asphyxia in OSAS [64]. CPAP therapy remains the mainstay of OSAS treatment in the adult population. The use of CPAP is also extremely useful to manage the airway in a child with OSAS during anesthesia [71], principally because it acts to increase the cross-sectional area of the pharynx [61,72]. Fig. 2 shows the relationship between airway pressure and the cross-sectional area of the pharynx. Fifty percent of the maximal pharyngeal area is achieved at 5 cm H2O. The closing pressure at which the pharynx collapses correlates with the severity of OSAS [60,61] and the closing pressure during anesthesia may identify the patient with OSAS [73].

3.3. Analgesic management

Children with untreated OSAS demonstrate a blunted responsiveness to hypercarbia and greater respiratory depression with opioids [46,47]. Morphine consumption following adenotonsillectomy is decreased in children with OSAS who demonstrate recurrent episodic desaturation during sleep [74], and therefore the child with severe OSAS may require much less opioid for analgesia. An increased opioid sensitivity in children with severe OSAS is not a small issue for children who will be discharged home with opioids [20,75].

Morphine sparing adjuncts including dexamethasone [76] and acetaminophen [77] may be useful. The anti-inflammatory properties of dexamethasone may confer additional benefit since the nasal exudate in children with OSAS had a higher content of the inflammatory mediator leukotriene than children with chronic tonsillitis [78].

Sensory nerve blockade including glossopharyngeal nerve block should be used with caution in severe OSAS since topical anesthesia provokes an eight-fold greater decrease in pharyngeal cross-sectional area in the child with OSAS [59,79].

3.4. Discharge practice

Although adenotonsillectomy is curative in the majority of children with OSAS, the first post-operative night may be problematic [13,14,17,19,80]. Implicit in safety of ambulatory programs is the notion that a responsible, informed parent is a suitable caregiver at home, in the post-operative period. However the parent of a child with OSAS has feared for the child’s sleep disordered breathing for months. For this parent, the baseline pattern of breathing is one which includes restless sleep, airway obstruction, apnea and desaturation during sleep and poor rousability [3,4,71,81]. This parent is ill-prepared to recognize a deterioration in respiratory status in the post-operative period.

The onset of respiratory distress may be delayed until a time remote from surgery. One third of desaturation following adenotonsillectomy presented more than 8 h after surgery [17,67]. One-third of children who experienced major respiratory compromise presented 1–8 h after adenotonsillectomy [19]. Two prospective studies reported that the majority of desaturations following adenotonsillectomy on the first post-operative night, were associated with obstructive apneas [15,82]. Furthermore parameters of sleep disordered airway obstruction and desaturation worsen in the early morning hours [49]. A delayed onset of respiratory compromise is problematic for ambulatory programs and may require a prolonged period of observation and overnight monitoring during sleep in some children with OSAS. Supportive measures in the post-operative period may require the insertion of nasal airways, CPAP support, reintubation, ventilation and the administration of furosemide, salbutamol, racemic epinephrine, heliox and dexamethasone [13,14,17,18,83]. This level of pediatric respiratory support, in many hospitals, is only provided in an intensive care setting.

The implications of OSAS to ambulatory adenotonsillectomy programs is sobering, given the widespread prevalence of OSAS. A diagnosis of OSAS identifies a patient population at increased risk for peri-operative cardiorespiratory compli-
cations in a time when the majority of pediatric adenotonsillectomy in North America is performed through ambulatory programs [20] and the most common indication for adenotonsillectomy is obstructive breathing [7]. In a health care system designed to manage adenotonsillectomy through ambulatory programs, risk reduction strategies for OSAS will remain focused on case finding of high-risk children and exclusion of these children from ambulatory programs. It is the otherwise well apparent-ASA 1 child with undiagnosed severe OSAS who poses the greatest challenge to ambulatory programs. Establishing a diagnosis of OSAS has been hampered by the limited resource of sleep laboratories. Although the clinical scoring system to establish a diagnosis of OSAS recently published in the ASA’s Practice Guidelines for the Perioperative Management of Patients with Obstructive Sleep Apnea has not been formally validated, the guidelines have empowered clinical criteria to both establish a diagnosis of OSAS and stratify its severity [9]. The clinical risk score combined with the surgical risk score yield an overall peri-operative OSAS risk score. A child with moderate to severe OSAS who requires adenotonsillectomy totals a risk score of 5–6 (maximum of 6). This risk score may be sufficient to exclude the child from ambulatory programs. Prospective studies on cost effective risk reduction strategies following adenotonsillectomy for OSAS are urgently needed.

References


