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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.

Keywords: Pediatric; Obstructive sleep apnea syndrome; Ambulatory adenotonsillectomy; Anesthesia

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 preoperative 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].

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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 airway 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 neuromuscular 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 sequalae 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 [44]. 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 1 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 cardiorespiratory 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 apneae 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 92% 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.

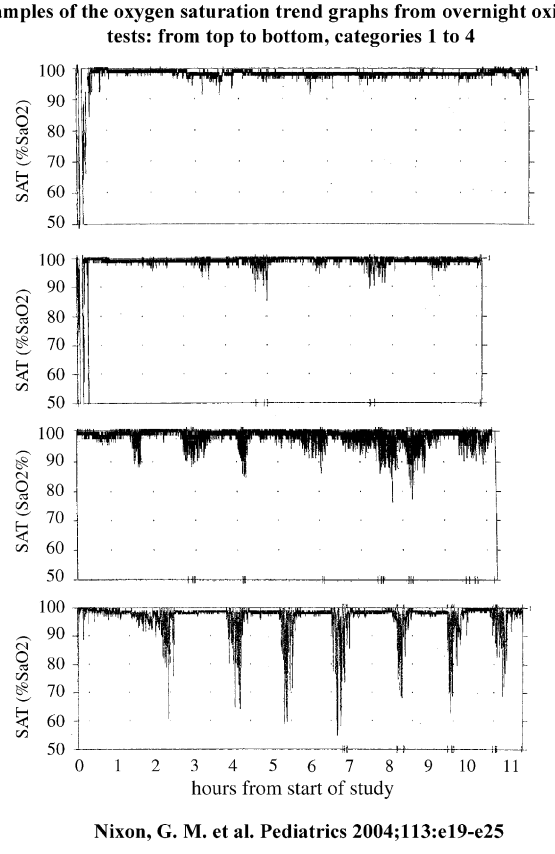

Fig. 1. Representative nocturnal oximetry trend traces for McGill oximetry scores 1–4 (top to bottom). McGill oximetry score of 1 is an inconclusive study. McGill oximetry scores 2, 3 and 4 are distinguished by the nadir desaturation which are 90%, 85% and 80%, respectively. Reproduced with permission 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 [1,9].

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 perioperative 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 H_2O. 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].

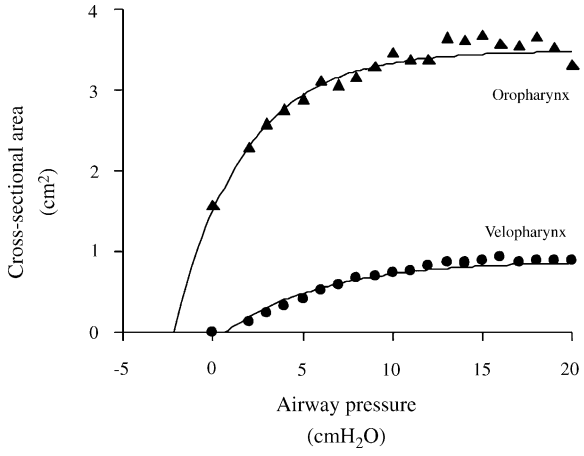

Fig. 2. The influence of positive pressure on the pharyngeal cross-sectional area for both the velo- and oro-pharynx. Reproduced with permission from J Appl Physiol 95:2257–64, Copyright 2003 by the American Physiology Society.

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 maybe sufficient to exclude the child from ambulatory programs. Prospective studies on cost effective risk reduction strategies following adenotonsillectomy for OSAS are urgently needed.

References

[1] Marcus CL, England SE, Annett RD, Brooks LJ, Brouillette RT, Carroll JL, et al. Cardiorespiratory sleep studies in children. Am J Respir Crit Care Med 1999;160:1381–7.

[2] Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea. Am J Respir Crit Care Med 2002;165:1217–39.

[3] Guilleminault C, Eldridge FL, Simmons FB, Dement WC. Sleep apnea in eight children. Pediatrics 1976;58:23–31.

[4] Franco RT, Rosenfeld RM, Rao M. Quality of life for children with obstructive sleep apnea. Otolaryngol Head Neck Surg 2000;123:9–16.

[5] Benumof JL. Obstructive sleep apnea in the adult obese patient: implications for airway management. J Clin Anesth 2001;13:144–56.

[6] Rothschild MA, Catalano P, Biller HF. Ambulatory pediatric tonsillectomy and the identification of high-risk subgroups. Otolaryngol Head Neck Surg 1994;110:203–10.

[7] Weatherly RA, Mai EF, Ruzicka DL, Chervin RD. Identification and evaluation of obstructive sleep apnea prior to adenotonsillectomy in children: a survey of practice patterns. Sleep Med 2003;4:297–307.

[8] Pratt LW, Gallagher RA. Tonsillectomy and adenoidectomy: incidence and mortality, 1968–1972. Otolaryngol Head Neck Surg 1979;87:159–66.

[9] American Society of Anesthesiologists Task Force on Perioperative Management of Patients with Obstructive Sleep Apnea. Practice guidelines for the perioperative management of patients with obstructive sleep apnea. 2005; p. 1–47 [www.asahq.org/publicationsServices].

[10] Richmond KH, Wetmore RF, Baranak CC. Postoperative complications following tonsillectomy and adenoidectomy—who is at risk? Int J Pediatr Otorhinolaryngol 1987;13:117–24.

[11] Berkowitz RG, Zalzal GH. Tonsillectomy in children under 3 years of age. Arch Otolaryngol Head Neck Surg 1990;116:685–6.

[12] Kendrick D, Gibbin K. An audit of the complications of paediatric tonsillectomy, adenoidectomy and adenotonsillectomy. Clin Otolaryngol 1993;18:115–7.

[13] McColley SA, April MM, Carroll JL, Naclerio RM, Loughlin GM. Respiratory compromise after adenotonsillectomy in children with obstructive sleep apnea. Arch Otolaryngol Head Neck Surg 1992;118:940–3.

[14] Rosen GM, Muckle RP, Mahowald MW, Goding GS, Ullevig C. Postoperative respiratory compromise in children with obstructive sleep apnea syndrome: can it be anticipated? Pediatrics 1994;93:784–8.

[15] Helfaer MA, McColley SA, Pyzik PL, Tunkel DE, Nichols DG, Baroody FM, et al. Polysomnography after adenotonsillectomy in mild pediatric obstructive sleep apnea. Crit Care Med 1996;24:1323–7.

[16] Tom LWC, DeDio RM, Cohen DE, Wetmore RF, Handler SD, Potsic WP. Is outpatient tonsillectomy appropriate for young children? Laryngoscope 1992;102:277–80.

[17] Wilson K, Lakheeram I, Morielli A, Brouillette RT, Brown K. Can assessment for obstructive sleep apnea help predict postadenotonsillectomy respiratory complications? Anesthesiology 2002;96:313–22.

[18] Brown K, Morin I, Hickey C, Manoukian JJ, Nixon GM, Brouillette RT. Urgent adenotonsillectomy: an analysis of risk factors associated with postoperative respiratory morbidity. Anesthesiology 2003;99:586–95.

[19] Nixon GM, Kermack AS, Davis GM, Manoukian JJ, Brown KA, Brouillette RT. Planning adenotonsillectomy in children with obstructive sleep apnea: the role of overnight oximetry. Pediatrics 2004;113:e19–25.

[20] Cote CJ, Sheldon SH. Obstructive sleep apnea and tonsillectomy: do we have a new indication for extended postoperative observation? Can J Anaesth 2004;51:6–12.

[21] Rosen G. Identification and evaluation of obstructive sleep apnea prior to adenotonsillectomy in children: is there a problem? Sleep Med 2003;4:273–4.

[22] Redline S, Tishler PV, Hans MG. Racial differences in sleep-disordered breathing in African-Americans and Caucasians. Am J Respir Crit Care Med 1997;155:186–92.

[23] Palmer LJ, Buxbaum SG, Larkin EK, Patel SR, Elston RC, Tishler PV, et al. Whole genome scan for obstructive sleep apnea and obesity in African-American families. Am J Respir Crit Care Med 2004;169:1314–21.

[24] Stepanski E, Zayyad A, Nigro C, Lopata M, Basner R. Sleep-disordered breathing in a predominantly African-American pediatric population. J Sleep Res 1999;8:65–70.

[25] Marcus CL, Keens TG, Bautista DB, Von Pechmann WS, Davidson Ward SL. Obstructive sleep apnea in children with down syndrome. Pediatrics 1991;88:132–9.

[26] Crysdale WS, Djupesland P. Nasal obstruction in children with craniofacial malformations. Int J Pediatr Otorhinolaryngol 1999;49:S63–7.

[27] McGowan FX, Kenna MA, Fleming JA, O'Connor T. Adenotonsillectomy for upper airway obstruction carries increased risk in children with a history of prematurity. Pediatr Pulmonol 1992;13:222–6.

[28] Redline S, Tishler PV, Schluchter M, Aylor J, Clark K, Graham G. Risk factors for sleep-disordered breathing in children: associations with obesity, race, and respiratory problems. Am J Respir Crit Care Med 1999;159:1527–32.

[29] Wing YK, Hui SH, Pak WM, Ho CK, Cheung A, Li AM, et al. A controlled study of sleep related disordered breathing in obese children. Arch Dis Child 2003;88:1043–7.

[30] Mitchell RB, Kelly J. Adenotonsillectomy for obstructive sleep apnea in obese children. Otolaryngol Head Neck Surg 2004;131:104–8.

[31] Nixon GM, Brouillette RT. Sleep and breathing in Prader–Willi syndrome. Pediatr Pulmonol 2002;34:209–17.

[32] Marcus CL, Curtis S, Koerner CB, Joffe A, Serwint JR, Loughlin GM. Evaluation of pulmonary function and polysomnography

in obese children and adolescents. Pediatr Pulmonol 1996;21:176–83.
[33] Guilleminault C, Pelayo R, Leger D, Clerk A, Bocian RCZ. Recognition of sleep-disordered breathing in children. Pediatrics 1996;98:871–82.
[34] Guilleminault C, Korobkin R, Winkle R. A review of 50 children with obstructive sleep apnea syndrome. Lung 1981;159:275–87.
[35] Gozal D. Sleep disordered breathing and school performance in children. Pediatrics 1998;102:616–20.
[36] Row BW, Kheirandish L, Neville JJ, Gozal D. Impaired spatial learning and hyperactivity in developing rats exposed to intermittent hypoxia. Pediatr Res 2002;52:449–53.
[37] O'Brien LM, Mervis CB, Holbrook CR, Bruner JL, Smith NH, McNally N, et al. Neurobehavioral correlates of sleep-disordered breathing in children. J Sleep Res 2004;13:165–72.
[38] Wiatrak BJ, Myer III CM, Andrews TM. Complications of adenotonsillectomy in children under 3 years of age. Am J Otolaryngol 1991;12:170–2.
[39] Carroll JL, McColley SA, Marcus CL, Curtis S, Loughlin GM. Inability of clinical history to distinguish primary snoring from obstructive sleep apnea syndrome in children. Chest 1995;108:610–8.
[40] van Someren V, Burmester B, Alusi G, Lane R. Are sleep studies worth doing? Arch Dis Child 2000;83:76–81.
[41] Messner AH. Evaluation of obstructive sleep apnea by polysomnography prior to pediatric adenotonsillectomy. Arch Otolaryngol Head Neck Surg 1999;125:353–6.
[42] Brouillette RT, Hanson D, David R, Klemka L, Szatkowski A, Fernbach S, et al. A diagnostic approach to suspected obstructive sleep apnea in children. J Pediatr 1984;105:10–4.
[43] Amin RS, Kimball TR, Bean JA, Jeffries JL, Willging JP, Cotton RT, et al. Left ventricular hypertrophy and abnormal ventricular geometry in children and adolescents with obstructive sleep apnea. Am J Respir Crit Care Med 2002;165:1395–9.
[44] Tal A, Leiberman A, Margulis G, Sofer S. Ventricular dysfunction in children with obstructive sleep apnea: radionuclide assessment. Pediatr Pulmonol 1988;4:139–43.
[45] Konno A, Hoshino T, Togawa K. Influence of upper airway obstruction by enlarged tonsils and adenoids upon recurrent infection of the lower airway in childhood. Laryngoscope 1980;90:1709–16.
[46] Strauss SG, Lynn AM, Bratton SL, Nespeca MK. Ventilatory response to CO_2 in children with obstructive sleep apnea from adenotonsillar hypertrophy. Anesth Analg 1999;89:328–32.
[47] Waters KA, McBrien F, Stewart P, Hinder M, Wharton S. Effects of OSA, inhalational anesthesia, and fentanyl on the airway and ventilation of children. J Appl Physiol 2002;92:1987–94.
[48] Davidson SL, Marcus CL. Obstructive sleep apnea in infants and young children. J Clin Neurophysiol 1996;13:198–207.
[49] Goh DYT, Galster P, Marcus CL. Sleep architecture and respiratory disturbances in children with obstructive sleep apnea. Am J Respir Crit Care Med 2000;162:682–6.
[50] Jacob SV, Morielli A, Mograss MA, Ducharme FM, Schloss MD, Brouillette RT. Home testing for pediatric obstructive sleep apnea syndrome secondary to adenotonsillar hypertrophy. Pediatr Pulmonol 1995;20:241–52.
[51] Hamilton GS, Solin P, Naughton MT. Obstructive sleep apnoea and cardiovascular disease. Intern Med J 2004;34:420–6.
[52] Nixon GM, Brouillette RT. Diagnostic techniques for obstructive sleep apnoea: is polysomnography necessary? Paediatr Respir Rev 2002;3:18–24.
[53] Marcus CL, Omlin KJ, Basinski DJ, Bailey SL, Rachal AB, Von Pechmann WS, et al. Normal polysomnographic values for children and adolescents. Am Rev Respir Dis 1992;146:1235–9.
[54] Rosen CL, D'Andrea L, Haddad GG. Adult criteria for obstructive sleep apnea do not identify children with serious obstruction. Am Rev Respir Dis 1992;146:1231–4.
[55] Isono S, Suzukawa M, Sho Y, Ohmura A, Kudo Y, Misawa K, et al. Preoperative nocturnal desaturations as a risk factor for late postoperative nocturnal desaturations. Br J Anaesth 1998;80:602–5.
[56] Brouillette RT, Morielli A, Leimanis A, Waters KA, Luciano R, Ducharme FM. Nocturnal pulse oximetry as an abbreviated testing modality for pediatric obstructive sleep apnea. Pediatrics 2000;105:405–12.
[57] Bonsignore G, Marrone O, Macaluso C, Salvaggio A, Stallone A, Bellia V. Validation of oximetry as a screening test for obstructive sleep apnoea syndrome. Eur Respir J 1990;11(Suppl):542s–4s.
[58] Monahan KJ, Larkin EK, Rosen CL, Graham G, Redline S. Utility of noninvasive pharyngometry in epidemiologic studies of childhood sleep-disordered breathing. Am J Respir Crit Care Med 2002;165:1499–503.
[59] Gozal D, Burnside MM. Increased upper airway collapsibility in children with obstructive sleep apnea during wakefulness. Am J Respir Crit Care Med 2004;169:164–7.
[60] Marcus CL, McColley SA, Carroll JL, Loughlin GM, Smith PL, Schwartz AR. Upper airway collapsibility in children with obstructive sleep apnea syndrome. J Appl Physiol 1994;77(2):918–24.
[61] Isono S, Shimada A, Utsugi M, Konno A, Nishino T. Comparison of static mechanical properties of the passive pharynx between normal children and children with sleep-disordered breathing. Am J Respir Crit Care Med 1998;157:1204–12.
[62] Berthon-Jones M, Sullivan CE. Time course of change in ventilatory response to CO_2 with long-term CPAP therapy for obstructive sleep apnea. Am Rev Respir Dis 1987;135:144–7.
[63] Krieger J, Grucker D, Sforza E, Chambron J, Kurtz D. Left ventricular ejection fraction in obstructive sleep apnea. Chest 2005;100:917–21.
[64] Sullivan CE, Issa FG, Berthon-Jones M, Eves L. Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. Lancet 1981;317:862–5.
[65] Kravath RE, Pollak CP, Borowiecki B. Hypoventilation during sleep in children who have lymphoid airway obstruction treated by nasopharyngeal tube and T and A. Pediatrics 1977;59:865–71.
[66] Nixon GM, Brouillette RT. Obstructive sleep apnea in children: do intranasal corticosteroids help? Am J Respir Med 2002;1:159–66.
[67] Koomson A, Morin I, Brouillette RT, Brown KA. Children with severe OSAS who have adenotonsillectomy in the morning are less likely to have postoperative desaturation than those operated in the afternoon. Can J Anaesth 2004;51:62–7.
[68] Coleman CG, Lydic R, Baghdoyan HA. M2 muscarinic receptors in pontine reticular formation of C57BL/6J mouse contribute to rapid eye movement sleep generation. Neuroscience 2004;126:821–30.
[69] Garrigue S, Bordier P, Jais P, Shah DC, Hocini M, Raherisson C, et al. Benefit of atrial pacing in sleep apnea syndrome. N Engl J Med 2002;346:404–12.
[70] Liu X, Sood S, Liu H, Horner RL. Opposing muscarinic and nicotinic modulation of hypoglossal motor output to genioglossus muscle in-vivo. J Physiol 2005;565:965–80.
[71] Guilleminault C, Stoohs R. From apnea of infancy to obstructive sleep apnea syndrome in the young child. Chest 1992;102:1065–71.
[72] Isono S, Tanaka A, Nishino T. Dynamic interaction between the tongue and soft palate during obstructive apnea in anesthetized patients with sleep-disordered breathing. J Appl Physiol 2003;95:2257–64.
[73] Eastwood PR, Szollosi I, Platt PR, Hillman DR. Comparison of upper airway collapse during general anaesthesia and sleep. Lancet 2002;359:1207–9.
[74] Brown KA, Laferrière A, Moss IR. Recurrent hypoxemia in young children with obstructive sleep apnea is associated with reduced opioid requirements for analgesia. Anesthesiology 2004;100:806–10.
[75] Sutters KA, Miaskowski C, Holdridge-Zeuner D, Waite S, Paul SM, Savedra MC, et al. A randomized clinical trial of the effectiveness of a scheduled oral analgesic dosing regimen for the management of postoperative pain in children following tonsillectomy. Pain 2004;110:49–55.

[76] Elhakim M, Ali NM, Rashed I, Riad MK, Refat M. Dexamethasone reduces postoperative vomiting and pain after pediatric tonsillectomy. Can J Anaesth 2003;50:392–7.
[77] Korpela R, Korvenoja P, Meretoja OA. Morphine-sparing effect of acetaminophen in pediatric day-case surgery. Anesthesiology 1999;91:442–7.
[78] Goldbart AD, Goldman JL, Li RC, Brittian KR, Tauman R, Gozal D. Differential expression of cysteinyl leukotriene receptors 1 and 2 in tonsils of children with obstructive sleep apnea syndrome or recurrent infection. Chest 2004;126:13–8.
[79] Bean-Lijewski JD. Glossopharyngeal nerve block for pain relief after pediatric tonsillectomy: retrospective analysis and two cases of life-threatening upper airway obstruction from an interrupted trial. Anesth Analg 1997;84:1232–8.
[80] Weinberg S, Kravath R, Phillips L, Mendez H, Wolf GL. Episodic complete airway obstruction in children with undiagnosed obstructive sleep apnea. Anesthesiology 1984;60:356–8.
[81] Katz ES, Greene MG, Carson KA, Galster P, Loughlin GM, Carroll J, et al. Night-to-night variability of polysomnography in children with suspected obstructive sleep apnea. J Pediatr 2002;140:589–94.
[82] Nixon GM, Kermack AS, McGregor CD, Davis GM, Manoukian JJ, Brown KA, et al. Sleep and breathing on the first night after adenotonsillectomy for obstructive sleep apnea. Pediatr Pulmonol 2005;39:332–8.
[83] Spector A, Scheid S, Hassink S, Deutsch ES, Reilly JS, Cook SP. Adenotonsillectomy in the morbidly obese child. Int J Pediatr Otorhinolaryngol 2003;67:359–64.