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Childhood Obstructive Sleep Apnoea: What Parents Want to Know

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Abstract This article is the result of five years of accumulating knowledge, experience gained from our clinical work and research carried out by our team on childhood obstructive sleep apnoea syndrome (OSAS). It is very much a review of our personal practice and we try to provide as much evidence based materials as possible though available data is often limited. There is no doubt that a large gap is still waiting to be filled regarding this important and common condition. We hope this article would help practicing clinicians to provide answers to questions commonly asked by parents of children suspected of having OSAS.

Key words Child; Obstructive sleep apnoea; Polysomnography

How Common Is It?

Snoring is the hallmark symptom of obstructive sleep apnoea syndrome (OSAS) and its prevalence in local school children has been found to be >15% in recently published studies.[1,2] There is however, limited local data available on the prevalence of OSAS. Wing et al[1] in their study involving obese children and randomly selected normal weight controls found the prevalence of OSAS among normal children to be between 2.3-4.5%. Unfortunately, this study only involved 44 normal healthy children. Overseas studies have reported the estimates to be between 0.7-10.3% but it is important to note that these studies had methodological flaws and may not be a true representation for our locality.[3-7] Therefore a well-designed population-based cohort study with an adequate sample size is urgently required to answer the question of how common OSAS is among our local children.

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What Are the Symptoms and Signs?

We carried out a retrospective analysis of the presenting symptoms of 50 polysomnography (PSG) confirmed OSAS children who presented to our unit over a 12-month period.[8] Their mean age was 6.5 years (standard deviation 2.7) and there were 35 boys. The summary of their presenting symptoms is shown in the following figure (Figure 1). Forty-four (88%) children snored every night and the snoring could at least be heard in the same room for all of them. Other common presenting night-time symptoms included struggling to breathe during sleep (74%), restless sleep (74%), increased night sweats (58%) and sleeping with the neck extended or in the prone position (54%). Twelve (24%) children had apneoic episodes during sleep witnessed by their parents but only four required stimulation to make them breathe again. Four (8%) children had nocturnal enuresis. The most common daytime symptom was mouth breathing (86%) and daytime somnolence was a feature in only 6 children (12%).

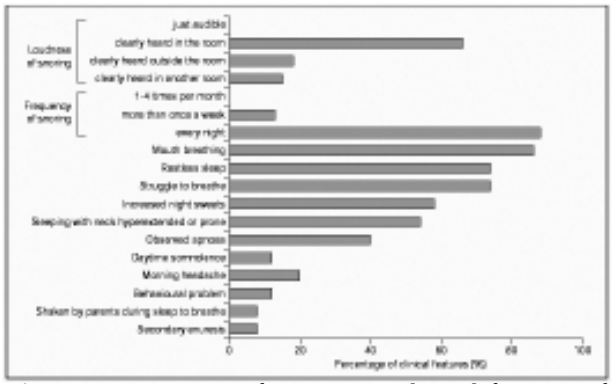

Figure 1 Frequency of presenting clinical features of obstructive sleep apnoea syndrome (n=50).

What Are the Causes of OSAS?

The most common cause of OSAS in children is adenotonsillar hypertrophy and most cases of which are amenable to surgical treatment.[9] The peak incidence occurs between 3 and 6 years of age as that corresponds to the age range when upper airway lymphoid tissue enlargement, relative to craniofacial size, is greatest.[10] However, the size of tonsils on direct inspection does not bear any relationship with the presence and severity of OSAS.[11] Obesity is another important factor for OSAS in children; obese children are 10 times more likely than normal weight controls to suffer from the condition.[1] Other causes for OSAS include Down syndrome, craniofacial anomalies, neuromuscular disorders and laryngomalacia.

Will My Child Die from OSAS?

Adults with OSAS have been reported to be at an increased risk of dying early, compared to the general population, mainly from respiratory and cardiovascular complications. Hypertension, obesity, age between 30 and 50 years and a severe degree of OSAS increase the risk of early death in these patients.[12] The mortality rate in children with OSAS is unknown. Deaths thought to be secondary to OSAS in children occurred during the time when childhood OSAS was relatively unrecognised and were attributed to perioperative cardiorespiratory failure in children with associated craniofacial and neurological disorders.[13] Definitive causal relationship between sudden infant death and OSAS has not been established. An association between sleep-disordered breathing and apparent life-threatening events has however, been suggested.[14]

Does It Run in the Family?

Studies using familial correlation and segregation analysis have provided data to support that inheritable factors contribute to the development of OSAS and that maternal components may be more important than paternal ones.[15,16] However, the exact pattern of inheritance and whether this genetic predisposition is modifiable by other environmental factors is still unknown. The individual craniofacial skeleton maybe an important component of the final end product of this genetic predisposition.

What Are the Complications?

Neuro-cognitive abnormalities – In contrast to adult patients, affected children tend to have preserved sleep architecture and therefore excessive daytime sleepiness (EDS) is not usually a predominant feature of childhood OSAS.[17] Despite the relative absence of EDS, OSAS and even snoring appear to be associated with significant behavioural and learning problems, poor attention span, hyperactivity and even lower than average intelligence quotient.[18-24] There is some evidence to suggest reversibility of such neuro-cognitive dysfunction following treatment.[22-24]

Growth failure – Marcus et al[25] evaluated 14 prepubertal children with a mean age of 4 years who had OSAS documented by overnight PSG. Caloric intake and sleeping energy expenditure as well as anthropomorphic measurements were taken before and after adenotonsillectomy. Average sleeping energy expenditure decreased, and mean weight Z score increased postoperatively without any change in caloric intake. We had carried out a case-control resting energy expenditure study and found a trend towards higher energy consumption in the OSAS group but we could not establish a correlation between severity of OSAS and energy expenditure.[26] Bar et al[27] evaluated changes in growth and also measured insulin-like growth factor (IGF)-I before and 18 months after adenotonsillectomy. There was a statistically significant increase in weight and IGF-I levels at 18 months. The pathogenesis associated with failure to thrive in children with OSAS is likely to be an inter-play between various mechanisms. Luckily we are seeing less of this complication as people are becoming more aware of this condition and tend to seek medical intervention early.

Cardiovascular abnormalities – In adults, the presence of OSAS is without doubt associated with an increased risk of systemic hypertension.[28] Systemic blood pressure (BP)

was evaluated in a study of children referred for PSG.[29] Higher diastolic pressures (adjusted for body mass index and age) were found in children with OSAS, compared with those with primary snoring. However, the response after adenotonsillectomy was not reported. A local study demonstrated abnormal arterial distensibility and blood pressure even in primary snorers.[30] A well-designed long-term prospective study is needed to assess whether any cardiac abnormalities detected are precursor for cardio-vascular disease in adults.

How Do You Make the Diagnosis?

OSAS is unlikely in the absence of habitual snoring. Symptoms on their own however, have a poor diagnostic yield.[31] Findings on physical examination are often normal. Size of tonsils and adenoids on direct inspection are not related to the presence or severity of OSAS.[11]

The "gold standard" for the diagnosis of OSAS is overnight polysomnography (PSG) even though it has not been well standardised in its performance or interpretation especially among the paediatric population. The most acceptable diagnostic cutoff for childhood OSAS as proposed by Marcus et al[32] is an obstructive apnoea index of greater than 1. However, this criterion does not take into account episodes of hypopnoea that are important in children with OSAS. The same group recently proposed an apnoea-hyponoea index of greater 1.5 as diagnostic for OSAS.[33]

In adults, incorrectly diagnosing OSAS as primary snoring after a single night recording has been reported in some studies.[34-36] A disturbed sleep pattern which is known as the "first night effect" caused by artificial sleep laboratory environment and continuous visual surveillance is well described and may influence the result reliability of a single night study.[36-39] First night effect is known to reduce the amount of rapid eye movement (REM) sleep and with childhood OSAS being more severe during REM sleep, a single night PSG may be expected to generate a high rate of false negative studies. We recently carried out a study looking at the feasibility of a single night study in the assessment of OSAS in children. The first night PSG would have correctly identified 84.6% of OSAS cases and all cases missed by the first night study had only borderline PSG abnormality. Thus a single night sleep study is adequate and more cost effective in assessing for childhood OSAS (unpublished findings).

A variety of screening tests have been proposed, including pulse oximetry, abbreviated sleep study, audiotaping and radiography. They were all compared to polysomnography as the gold standard with its inherent problems as mentioned above. Most tests were found to have acceptable positive predictive value but less than desirable specificity and negative predictive value.

What Treatments Are Available?

Medical treatment – Nasal corticosteroids have recently been examined as an alternative to adenotonsillectomy in otherwise healthy children with OSAS. Brouillette et al[40] in a prospective, randomised, double-blinded study treated children with mild to moderate OSAS with a 6-week course of either nasal corticosteroids or placebo. The authors were able to demonstrate a moderate improvement in cases treated with nasal corticosteroids. The apnoea hypopnoea index decreased from 11/hr to 6/hr. This was associated with concomitant decreases of about 50% in both the desaturation index and the movement arousal index. In contrast, the placebo group did not show improvement. Wheeler et al[41] found significant improvement in symptom scores of 40-50% in subjects treated with 4 months of nasal fluticasone at a dose of 200 micrograms once daily, but formal assessment using overnight polysomnography was not mentioned. Kiely et al[42] were also able to demonstrate beneficial effect in patients with OSAS and rhinitis treated with a 4-week course of intranasal fluticasone. Topical intranasal steroids may become the alternative therapy for some children with mild OSAS. This is equally important if individuals also suffer from allergic rhinitis that causes nasal oedema and hence increases nasal airflow resistance. Treating the underlying rhinitis will lessen the severity of obstruction.

The importance of weight reduction by means of dietary intervention and increased physical activity should be emphasised in cases where obesity is the main aetiological factor. In our clinical experience, however, the majority of patients have difficulty achieving significant weight loss through such means.

Surgical treatment – A few studies provide evidence that adenotonsillectomy is superior to either adenoidectomy or tonsillectomy alone.[43,44] Summarising results from all published studies, it appears that adenotonsillectomy is curative in around 85% of children. Some children with persistent disease after adenotonsillectomy may benefit from uvulopalatopharyngoplasty, maxillary or mandibular surgery, or tracheostomy.[45]

Mechanical treatment – In childhood OSAS, nasal continuous positive airway pressure (CPAP) or bi-level positive airway pressure (BiPAP) have established themselves as the second line treatment or in cases where adenotonsillectomy is contraindicated.[46-48] CPAP provides positive pressure to the lumen of the airway, and decreases airway collapsibility. Bi-level positive airway pressure may be more comfortable to use, especially with higher pressures. BiPAP also allows setting of a backup rate, and provides some ventilatory assistance. This is especially important for patients with sleep-related hypoventilation caused by muscle weakness, neurological disease, or obesity.

Will It Recur After Treatment?

It is our practice to review patients who have undergone adenotonsillectomy at 4-6 months after the operation. If they remain symptomatic, a repeat PSG would be offered and mechanical intervention would be instituted if residual OSAS is demonstrated. The following group of patients have been identified to be at a higher risk for residual problem; initial severe OSAS, obesity and those with a positive family history of OSAS.[49] In a study by Contencin et al,[50] 8.5% of children who had undergone adenotonsillectomy experienced residual or recurrent symptoms of OSAS at 3 years following the operation. In a recently published local study,[51] boys undergoing surgical adenotonsillectomy at an early age (<5 years) were found to be at a higher risk for residual OSAS.

As our knowledge on this condition increases, more questions are going to be generated. Further advancement in this important field of paediatric medicine can only be made with collaborative research. We hope this article would provide clinicians with up-to-date information to deal with commonly asked questions regarding childhood OSAS.

References

1. Wing YK, Hui SH, Pak WM, et al. A controlled study of sleep related disordered breathing in obese children. Arch Dis Child 2003;88:1043-7.
2. Chau KW, Ng DK, Kwok CK, Chow PY, Ho JC. Clinical risk factors for obstructive sleep apnoea in children. Singapore Med J 2003;44:570-3.
3. Ali NJ, Pitson DJ, Stradling JR. Snoring, sleep disturbance, and behaviour in 4-5 year olds. Arch Dis Child 1993;68:360-6.
4. Gislason T, Benediktsdottir B. Snoring, apneic episodes, and nocturnal hypoxemia among children 6 months to 6 years old. An epidemiologic study of lower limit of prevalence. Chest 1995;107:963-6.
5. 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(5 Pt 1):1527-32.
6. Brunetti L, Rana S, Lospalluti ML, et al. Prevalence of obstructive sleep apnea syndrome in a cohort of 1,207 children of southern Italy. Chest 2001;120:1930-5.
7. Rosen CL, Larkin EK, Kirchner HL, et al. Prevalence and risk factors for sleep-disordered breathing in 8- to 11-year-old children: association with race and prematurity. J Pediatr 2003; 142:383-9.
8. Li AM, Hui S, Wong E, Cheung A, Fok TF. Obstructive sleep apnoea in children with adenotonsillar hypertrophy: prospective study. Hong Kong Med J 2001;7:236-40.
9. Hui S, Wing YW. Obstructive sleep apnoea syndrome in children. Hong Kong Med J 1997;3:419-426.
10. Jeans WD, Fernando DC, Maw AR, Leighton BC. A longitudinal study of the growth of the nasopharynx and its contents in normal children. Br J Radiol 1981;54:117-21.
11. Li AM, Wong E, Kew J, Hui S, Fok TF. Use of tonsil size in the evaluation of obstructive sleep apnoea. Arch Dis Child 2002; 87:156-9.
12. Lavie P, Herer P, Peled R, et al. Mortality in sleep apnea patients: a multivariate analysis of risk factors. Sleep 1995;18: 149-57.
13. Lauritzen C, Lilja J, Jarlstedt J. Airway obstruction and sleep apnea in children with craniofacial anomalies. Plast Reconstr Surg 1986;77:1-6.
14. Guilleminault C, Pelayo R, Leger D, Philip P, Ohayon M. Sleep-disordered breathing and upper-airway anomalies in first-degree relatives of ALTE children. Pediatr Res 2001;50:14-22.
15. Holberg CJ, Natrajan S, Cline MG, Quan SF. Familial Aggregation and Segregation Analysis of Snoring and Symptoms of Obstructive Sleep Apnea. Sleep Breath 2000;4: 21-30.
16. Buxbaum SG, Elston RC, Tishler PV, Redline S. Genetics of the apnea hypopnea index in Caucasians and African Americans: I. Segregation analysis. Genet Epidemiol 2002;22: 243-53.
17. Goh DY, Galster P, Marcus CL. Sleep architecture and respiratory disturbances in children with obstructive sleep apnea. Am J Respir Crit Care Med 2000;162(2 Pt 1):682-6.
18. Gozal D. Sleep-disordered breathing and school performance in children. Pediatrics 1998;102(3 Pt 1):616-20.
19. Guilleminault C, Korobkin R, Winkle R. A review of 50 children with obstructive sleep apnea syndrome. Lung 1981;159:275-87.
20. Weissbluth M, Davis AT, Poncher J, Reiff J. Signs of airway obstruction during sleep and behavioral, developmental, and academic problems. J Dev Behav Pediatr 1983;4:119-21.
21. Chervin RD, Dillon JE, Bassetti C, Ganoczy DA, Pituch KJ. Symptoms of sleep disorders, inattention, and hyperactivity in children. Sleep 1997;20:1185-92.
22. Ali NJ, Pitson D, Stradling JR. Sleep disordered breathing: effects of adenotonsillectomy on behaviour and psychological functioning. Eur J Pediatr 1996;155:56-62.
23. Blunden S, Lushington K, Kennedy D, Martin J, Dawson D.

Behavior and neurocognitive performance in children aged 5-10 years who snore compared to controls. J Clin Exp Neuropsychol 2000;22:554-68.
24. Goldstein NA, Post JC, Rosenfeld RM, Campbell TF. Impact of tonsillectomy and adenoidectomy on child behavior. Arch Otolaryngol Head Neck Surg 2000;126:494-8.
25. Marcus CL, Carroll JL, Koerner CB, Hamer A, Lutz J, Loughlin GM. Determinants of growth in children with the obstructive sleep apnea syndrome. J Pediatr 1994;125:556-62.
26. Li AM, Yin J, Chan D, Hui S, Fok TF. Sleeping energy expenditure in paediatric patients with obstructive sleep apnoea syndrome. Hong Kong Med J 2003;9:353-6.
27. Bar A, Tarasiuk A, Segev Y, Phillip M, Tal A. The effect of adenotonsillectomy on serum insulin-like growth factor-I and growth in children with obstructive sleep apnea syndrome. J Pediatr 1999;135:76-80.
28. Peppard PE, Young T, Palta M, Skatrud J. Prospective study of the association between sleep-disordered breathing and hypertension. N Engl J Med 2000;342:1378-84.
29. Marcus CL, Greene MG, Carroll JL. Blood pressure in children with obstructive sleep apnea. Am J Respir Crit Care Med 1998; 157(4 Pt 1):1098-103.
30. Kwok KL, Ng DK, Cheung YF. BP and arterial distensibility in children with primary snoring. Chest 2003;123:1561-6.
31. 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.
32. Marcus CL, Omlin KJ, Basinki DJ, et al. Normal polysomnographic values for children and adolescents. Am Rev Respir Dis 1992; 146(5 Pt 1):1235-9.
33. Witmans MB, Keens TG, Davidson Ward SL, Marcus CL. Obstructive hypopneas in children and adolescents: normal values. Am J Respir Crit Care Med 2003;168:1540.
34. Bliwise DL, Benkert RE, Ingham RH. Factors associated with nightly variability in sleep-disordered breathing in the elderly. Chest 1991;100:973-6.
35. Mosko SS, Dickel MJ, Ashurst J. Night-to-night variability in sleep apnea and sleep-related periodic leg movements in the elderly. Sleep 1988;11:340-8.
36. Aber WR, Block AJ, Hellard DW, Webb WB. Consistency of respiratory measurements from night to night during the sleep of elderly men. Chest 1989;96:747-51.
37. Le Bon O, Hoffmann G, Tecco J, et al. Mild to moderate sleep respiratory events: one negative night may not be enough. Chest 2000;118:353-9.
38. Agnew HW Jr, Webb WB, Williams RL. The first night effect: an EEG study of sleep. Psychophysiology 1966;2:263-6.
39. Bliwise DL, Carey E, Dement WC. Nightly variation in sleep-related respiratory disturbance in older adults. Exp Aging Res 1983;9:77-81.
40. Brouillette RT, Manoukian JJ, Ducharme FM, et al. Efficacy of fluticasone nasal spray for pediatric obstructive sleep apnea. J Pediatr 2001;138:838-44.
41. Wheeler AJ, van Someren V. Fluticasone for obstructive sleep apnea. J Pediatr 2002;140:489.
42. Kiely JL, Nolan P, McNicholas WT. Intranasal corticosteroid therapy for obstructive sleep apnoea in patients with co-existing rhinitis. Thorax 2004;59:50-5.
43. Zucconi M, Strambi LF, Pestalozza G, Tessitore E, Smirne S. Habitual snoring and obstructive sleep apnea syndrome in children: effects of early tonsil surgery. Int J Pediatr Otorhinolaryngol 1993;26:235-43.
44. Nieminen P, Tolonen U, Lopponen H. Snoring and obstructive sleep apnea in children: a 6-month follow-up study. Arch Otolaryngol Head Neck Surg 2000;126:481-6.
45. Cohen SR, Holmes RE, Machado L, Magit A. Surgical strategies in the treatment of complex obstructive sleep apnoea in children. Paediatr Respir Rev 2002;3:25-35.
46. Marcus CL, Ward SL, Mallory GB, et al. Use of nasal continuous positive airway pressure as treatment of childhood obstructive sleep apnea. J Pediatr 1995;127:88-94.
47. Waters KA, Everett FM, Bruderer JW, Sullivan CE. Obstructive sleep apnea: the use of nasal CPAP in 80 children. Am J Respir Crit Care Med 1995;152:780-5.
48. Guilleminault C, Nino-Murcia G, Heldt G, Baldwin R, Hutchinson D. Alternative treatment to tracheostomy in obstructive sleep apnea syndrome: nasal continuous positive airway pressure in young children. Pediatrics 1986;78:797-802.
49. Schechter MS; Section on Pediatric Pulmonology, Subcommittee on Obstructive Sleep Apnea Syndrome. Technical report: diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics 2002;109:e69.
50. Contencin P, Guilleminault C, Manach Y. Long-term follow-up and mechanisms of obstructive sleep apnea (OSA) and related syndromes through infancy and childhood. Int J Pediatr Otorhinolaryngol 2003;67 Suppl 1:S119-23.
51. Chau KW, Ng KK, Kwok KL, Cheung MY. Survey of children with obstructive sleep apnea syndrome in Hong Kong of China. Chin Med J (Engl) 2004;117:657-60.