



OBSTRUCTIVE SLEEP APNEA SYNDROME IN HONG KONG CHILDREN

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Introduction

The patency of the upper airway, from the nasopharynx to the hypopharynx, depends on the dilator muscles that counteract the collapsing force generated during inspiration. During sleep, especially during rapid eye movement (REM) phase, the upper airway usually decreases in diameter, because of sleep-induced decrease in reflex responsiveness of the pharyngeal dilator, e.g. genioglossus muscle, to negative pressure during inspiration. This decrease is more prominent in those with obesity, micrognathia, neuromuscular diseases or allergic rhinitis. Significant decrease in the upper airway patency results in sleep-disordered breathing (SDB). SDB affects the cognitive functions and stresses the cardiovascular system and results in obstructive sleep apnea syndrome (OSAS). Sleep polysomnography is important in the diagnosis of SDB.

Spectrum of Sleep-Disordered Breathing (SDB)

Snoring is a common symptom in patients with SDB that includes primary snoring (PS), upper airway resistance syndrome (UARS), obstructive hypoventilation and obstructive sleep apnea (OSA). Primary snoring refers to snoring without any other pathology, i.e. apnea, hypoventilation, hypoxaemia, hypercarbia, sleep disturbance and daytime symptoms.

UARS refers to frequent subtle arousal associated with laboured breathing secondary to upper airway obstruction. However, the frequent nocturnal arousals lead to daytime symptoms, e.g. inattentiveness, hyperactivity. UARS is diagnosed when successive breaths of increased inspiratory efforts end with an arousal (i.e. respiratory event related arousal, RERA). Increasing inspiratory effort can be measured by esophageal pressure (P_{es}) and increasing P_{es} more than 10 cm H_2O for 4 to 5 successive breaths is usually seen. In the absence of esophageal pressure monitor, UARS can be inferred from the presence of paradoxical breathing followed by arousal during sleep in those older than 3-year. Paradoxical breathing is the asynchronous movements of the chest and abdominal effort and it can be detected by piezo crystal sensor belts, quantum belts or respiratory inductive plethysmography.¹

Presence of daytime sleepiness would support the diagnosis of UARS.

OSA refers to episodes of apnea and hypopnea during sleep, usually associated with a reduction in oxyhaemoglobin or/and hypercarbia. The main controversy about definition of OSA lies with the normal upper limit of obstructive apnea/hypopnea index (AHI). Marcus *et al.*² reported the statistical upper limit of obstructive apnea not including obstructive hypopnea to be one and a re-analysis of the same data reported the upper limit of AHI to be 1.5.³ However, different studies used different values of AHI, from 5 to 30, as indicative of OSAS.⁴ Obstructive hypoventilation refers to episodes of elevated arterial carbon dioxide, often assessed by end-tidal CO_2 ($ETCO_2$), as a result of partial upper airway obstruction. Obstructive hypoventilation is diagnosed when $ETCO_2$ is more than 55 mmHg for more than 10% of total sleep time or $ETCO_2$ is more than 45 mmHg for more than 60% of total sleep time despite respiratory effort.

Primary snoring, UARS, obstructive hypoventilation and OSA are thought to lie on a continuum, with primary snoring on one end and OSAS on the other. Thus, there is a great deal of symptoms overlap between these four entities. One patient could have all four phenomena in the same night.

Hong Kong Data

A community-based survey in students, from 6 to 12 years old, showed that habitual snoring occurred in 11%, which is lower than that in outpatient population, and the prevalence of witnessed obstructive sleep apnea was 1.5%.⁵ In the outpatient population, the prevalence of habitual snoring was 14.5%.⁴ Chau *et al.*⁶ reported that OSAS was diagnosed in 25% of children who underwent sleep polysomnography (PSG) and boys were more likely to have moderate to severe OSAS than girls and the most common symptoms included snoring (100%), sweating (81%) during sleep and nasal blockage (61%) and sleepiness (34%) during daytime. Li *et al.*⁷ found that the common night-time symptoms of children with OSAS included restless sleep (69%), struggle to breathe during sleep (63%), increased night sweat (49%), sleep with the

neck extended or in the prone position (49%) and the commonest daytime symptom was mouth breathing (74%). Chau *et al* reported that 'snoring every night' (i.e. habitual snoring) was an important risk factor for OSAS in children with sensitivity of 91% and specificity of 75%.⁸ A study comparing children with OSAS and normal children reported that children with OSAS had higher oxygen desaturation index (9.8 vs 1.2), arousal index (6.6 vs 4.7) and sleep energy expenditure (44.83 vs 40.71 Kcal/kg/day).⁹

Obesity is a predisposing factor for OSAS in children because of the mass loading of upper airway and respiratory muscles as well as impaired ventilatory control.¹⁰ In the same study, 32.6% of obese children and 4.5% of normal weight children was found to have obstructive sleep apnea (OSA), defined as apnea hypopnea index more than or equal to 5 per hour.

Treatment of Sleep-Disordered Breathing (SDB)

Initial evidence suggested that symptoms of SDB could be relieved by treatment of allergic rhinitis with topical steroid.¹¹ Demain *et al.*¹² reported reduction of adenoidal hypertrophy with intranasal steroid and this suggested that the therapeutic effect of intranasal steroids in children with SDB might be due to reduction of adenoid size. Another study reported the relative abundance of steroid receptors in excised adenotonsillar tissue from subjects with obstructive sleep apnea.¹³ Initial evidence reported by Goldbart *et al.*¹⁴ suggested that daily oral montelukast treatment for 16 weeks could provide significant reduction in obstructive sleep apnea and hypopnea index by one episode per hour in children with SDB. However, trials of topical steroid were of limited sample size.¹¹⁻¹⁴ Further large scale study is warranted and required to assess the roles of intranasal corticosteroids and montelukast in childhood with SDB.

Tonsillectomy and adenoidectomy (T&A) is the commonest treatment for sleep disordered breathing. Other surgical procedures include uvulopalatopharyngoplasty (UP3), laser or radio-frequency ablation of redundant tissue, maxillo-mandibular advancement and tracheostomy. However, these procedures are rarely used in children and should only be employed selectively after extensive investigations. Continuous positive airway pressure (CPAP) is an option for those who are not suitable for surgery. Weight reduction is also important in obese children and may obviate surgery for those successful cases.¹⁵

Follow-up After T&A

Eight to 10% of children with OSAS had recurrence of OSAS in follow-up for 1 to 3 years.¹⁶ Both the male gender and T&A earlier than 5-year of age was found to be risk factor for OSAS recurrence after T&A.¹⁶

Snoring may recur in puberty particularly in boys because of the increase in testosterone. The increased testosterone production leads to enlargement of muscles, including upper airway muscle mass. This can lead to further

narrowing of the upper airway. Hence, regular follow-up is important for children and adolescents with SDB. Post-operative PSG is useful for children after T&A if snoring recurs.¹⁷

Sleep PSG Service in Hong Kong

Sleep PSG is currently the only investigation that could exclude OSAS in children with snoring and there are significant differences between the standards of sleep PSG for children and adults in terms of longer time of preparation because of co-operation problem, smaller nostrils, smaller chest and hypoventilation without apnea.¹⁸ In Hong Kong, a population of almost 7 million and about 1 million are younger than 15 years old, there are currently five paediatric sleep laboratories in Duchess of Kent Children Hospital, Kwong Wah Hospital, Pamela Youde Nethersole Eastern Hospital, Prince of Wales Hospital and United Christian Hospital with total 7 beds. In Kwong Wah Hospital, two beds can be offered for full-sleep PSG study, including routine end-tidal CO₂. However, the laboratory only operates four nights per week because of manpower constraint. For urgent cases, the waiting time for full-sleep PSG study is 6 months. Otherwise, the time between initial routine referral and sleep clinic appointment currently is 18 months.¹⁹ In contrast, the waiting time of using sleep PSG in private hospital is usually 1 to 2 weeks, but the utilization rate is low. The reasons are probably under-diagnosis and recognition by primary care doctors and paediatricians, cost of PSG and lack of a standardized protocol for referral and management of patients.²⁰ Also many children with snoring underwent adenotonsillectomy without PSG preoperatively. There should be a closer co-operation between public and private hospitals in using sleep PSG for shortening the waiting time. A guideline for management of childhood snoring may also be a helpful tool to facilitate this co-operation and the Hong Kong Society of Paediatric Respiriography is currently working on this guideline.

Links

1. Hong Kong Society of Paediatric Respiriography (HKSPR) – <http://www.hkspr.org/>
2. American Academy of Sleep Medicine (AASM) – <http://www.aasmnet.org/>
3. American Thoracic Society (ATS) – <http://www.thoracic.org/>
4. European Respiratory Society (ERS) – <http://www.ersnet.org/ers/default.aspx>

Conclusion

Frequent snoring is a common symptom in children and a sign of sleep-disordered breathing. Sleep polysomnography is the diagnostic test of choice. Successful treatment is readily available. The long waiting time for sleep PSG in the public sector poses a problem that could be solved by a closer collaboration between the private and public sector.

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