
THOMAS E HAVAS

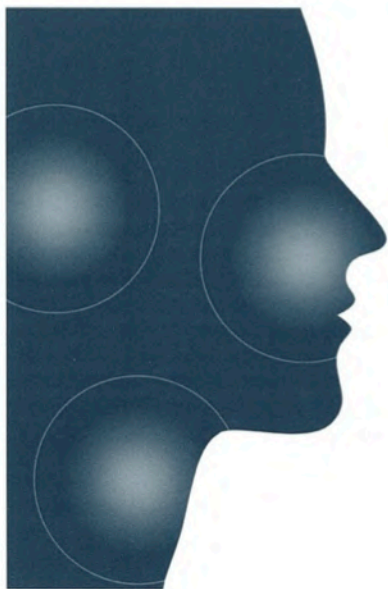
MBBS(SYD) MD(UNSW) FRCSE, FRACS, FACS

CONJOINT ASSOCIATE PROFESSOR UNSW

OTOLARYNGOLOGY HEAD AND NECK SURGERY

TREATMENT UPDATE

OBSTRUCTIVE SLEEP – APNOEA IN CHILDREN



HAVAS ENT CLINIC

Bondi Junction • Kogarah • Crows Nest

Tel **02 9387-7360**

Fax **02 9369-4463**

Email **thavas@ozemail.com.au**

Web **www.earnosethroat.com.au**

INTRODUCTION

Obstructive sleep apnoea/hypopnoea syndrome (OSAHS) is a common and serious condition occurring during childhood. It is estimated to affect 1 – 2% of all children with the incidence peaking during early childhood between the ages of 3 and 8 years. Untreated OSAHS is associated with serious cognitive neurobehavioural and cardiopulmonary dysfunction.

The pathophysiology of OSAHS remains poorly understood. Although it is related to adeno-tonsillar hypertrophy this is not likely to be the sole cause of sleep disordered breathing in the paediatric age group. Rather large tonsils and adenoids appear to precipitate OSAHS in children with underlying abnormalities of upper airway function. Normal children with relatively narrow upper airways maintain airway patency during sleep because of increased upper airway neuromotor tone and an increase in central ventilatory drive. Children with OSAHS appear to be lacking appropriate compensatory upper airway neuromuscular responses leading to both increased upper airway collapse and a lack of central respiratory drive.

HISTORY

Childhood obstructive sleep apnoea was first described in medical literature by William Osler in 1892. However Guilleminault et al published the first scientific case series in 1976.

PATHOPHYSIOLOGY OF CHILDHOOD OSAHS

Adenotonsillar hypertrophy clearly plays a role in the pathogenesis of childhood OSAHS. Other causes include obesity, craniofacial disorders and neuromuscular disorders. Childhood OSAHS is clearly not due to adeno-hypertrophy alone. A number of studies suggest that a combination of structural abnormalities such as adenotonsillar hypertrophy, narrow highly arched palate, turbinate hypertrophy and neuromuscular and central abnormalities must be present for OSAHS to occur. Most obvious is the fact that patients with OSAHS do not obstruct during wakefulness when tone of the upper airway muscles is increased. ***Studies have shown no correlation between upper airway or tonsillar size and frequency or severity of OSAHS.***

PREVALENCE

The prevalence of OSAHS is 1-2% of all children.

PEAK INCIDENCE

The peak incidence is between the ages of 3 and 8 years, mirroring the typical period of tonsil and adenoid enlargement. 50% of resistance to breathing occurs in the nose and TURBINATE ENLARGEMENT plays a large role in the aiteology of upper airway resistance syndrome.

HIGH RISK GROUPS

The prevalence of OSAHS is increased in children with

- Morbid obesity
- Cranio-facial syndrome
- Neuromuscular disorder

There may be a predisposition for increased prevalence in certain racial (African American) and socio-economic (low socio-economic) groups.

SEQUALE

Untreated OSAHS can result in serious morbidity including:

- Failure to grow
- Core pulmonale
- Neuro cognitive disorders
- Adverse consequences on academic achievement

There is also a strong association between OSAHS and Attention Deficit Hyperactivity Disorder (ADHD).

DIAGNOSIS

Diagnosis is made on formal overnight polysomnography. The threshold for diagnosing OSAHS has evolved and changed over the last 5 years; early studies used the criteria of five significant events per hour, but the currently accepted threshold is one significant event per hour. It is important to note that the technology of polysomnography (PSG) has also evolved over the years and it is likely that currently employed PSG equipment has enhanced sensitivity to detect OSAHS.

CLINICAL DIAGNOSIS OF OSAHS

There is in fact poor correlation between oropharyngeal examination and formal PSG due to:

- The multi factorial aetiology of the condition
- The important role of neuromuscular tone and central respiratory drive in maintaining a patent paediatric airway.

FOR OPTIMAL DIAGNOSTIC ACURACY CLINICANS MUST HAVE A LOW THERSHOLD FOR THE USE OF FORMAL POLYSOMNOGRAPHY.

POLYSOMNOGRAPHY

PSG has been underutilised in the diagnosis of OSAHS in children for two main reasons;

- Firstly, it was felt that clinical diagnosis correlated well with moderate to severe OSAHS.
- Secondly, formal PSG, although the gold star standard, has been associated with delays in treatment, increased cost and there was limited access in some areas.

POLYSOMNOGRAPHIC DIFFERENCES BETWEEN ADULTS AND CHILDREN WITH OSAHS

- Children appear to have clinical sequelae associated with milder forms of OSAHS than adults.
- Fewer and shorter obstructive apnoeas cause significant symptoms.
- Children have less fragmented sleep than adults.
- Children may show a pattern of persistent upper airway obstruction associated with hypercapnia and/or hypoxia rather than cyclic discrete obstructive episodes.
- In children, obstructive sleep apnoea is predominately a rapid eye movement (REM) phenomenon.
- Children have much less daytime somnolence than adults. Sleep deprivation / disturbance is more likely to be associated with hyperactivity than with somnolence.

THE ASSOCIATION BETWEEN SNORING AND OBSTRUCTIVE SLEEP APNOEA.

Normal children snore less than adults and rarely have obstructive apnoeas because the upper airway in children is less collapsible than it is in adults. The prevalence of snoring in young children varies between 3% and 27 % between the ages of 2 and 8 years. The prevalence of OSAHS is between 1 and 2% in that same age group, so clearly the vast majority of children who are snorers, even very loud snorers, appear not to have OSAHS.

SMOKING AND SNORING

An interesting albeit somewhat anticipated finding is that there is an increased frequency of snoring in smoking households.

OSAHS AND ATTENTION DEFICIT HYPERACTIVITY DISORDER (ADHD)

The association between sleep disordered breathing and ADHD is well documented in the literature lending support to the concept that the behavioural disturbances in at least some of the ADHD children may be resolved by addressing and treating their sleep disturbance. There is an increasing trend towards performing formal polysomnography in all children with ADHD to ensure that this important underlying cause is not missed.

TREATMENT

Primary treatment of choice remains adenotonsillectomy, with an anticipated success rate of between 80 and 95%. Children with OSAHS are at increased risk of post operative surgical complications including:

- Upper airway oedema
- Increased secretions
- Respiratory depression associated with analgesia and anaesthetic agents
- Post operative pulmonary oedema

Children with OSAHS should preferably undergo adenotonsillectomy in a tertiary referral paediatric hospital with access to appropriate intensive care facilities, the provision of CPAP overnight if required and as a routine should have pulse oximetry for the first 12 hours following operation. It is important to note that adenotonsillectomy is not 100% successful in the treatment of OSAHS in the paediatric age group. Children in high risk groups or with persisting symptoms after surgery, should have follow up PSG performed.

SUPPLEMENTARY TREATMENTS

Recent studies on children with persisting OSAHS following adenotonsillectomy identified two subsets with surgical problems:

- significant turbinate hypertrophy
- significant residual adenoids (with or without adenoid extension through the posterior nasal choanae into the nose).

The most commonly performed supplementary surgical procedure involves endoscopic turbinate reduction together with removal of any residual adenoid tissue. As awareness of these two conditions increases, more and more centres are incorporating turbinate surgery with adenotonsillectomy as their first line of surgical treatment.

USE OF NASAL STEROIDS IN THE TREATMENT OF OSAHS

Several recent studies have shown that prolonged use of intranasal steroid spray have no effect on OSAHS syndrome in children.

CONCLUSIONS

- OSAHS is a common disorder in childhood peaking in prevalence between the ages of 3 and 8 years.
- It affects 1-2% of the paediatric population.
- It is more common in children who are obese, with craniofacial syndromes or with a strong family history of obstructive sleep apnoea.
- The prevalence of OSAHS is increasing.
- The diagnosis of OSAHS requires formal polysomnography because the clinical correlation between examination of the oropharynx and the presence and severity of the condition is poor.
- High risk groups should have post operative polysomnography.
- Children with ADHD should routinely have polysomnography performed to exclude OSAHS as a cause of their hyperactivity.
- Primary surgical management remains adenotonsillectomy but increasingly turbinate surgery is being added to the first line of treatment.
- Children with OSAHS are at increased risk of perioperative complications, require careful monitoring and are optimally operated on in a tertiary referral paediatric hospital.