Review Article

Obstructive Sleep Apnoea in Children

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Introduction

The chronic debilitating Obstructive Sleep Apnoea (OSA or OSAS) syndrome in adults is now increasingly recognized in children.OSA was described nearly a century ago but sleep apnoea in infants was first described in 1975 in relation to sudden infant death syndrome and OSA in school children was described in 1976. It is a disorder of breathing during sleep characterized by "prolonged partial upper airway obstruction and or intermittent complete obstruction that disrupts normal ventilation during sleep and normal sleep patterns"[1]. OSA is the most common type of sleep-disordered breathing (SDB). OSA is part of the spectrum of sleep disordered breathing and includes Primary Snoring [PS], Upper airway resistance syndrome [UARS], Obstructive hypoventilation [hypo-apnoea], sleep apnoea[2]. If unrecognized and untreated OSA can lead to impaired day time functioning as well as more serious complications such as heart failure, developmental delay, poor growth and death. The long term neurocognitive metabolic and cardiovascular complications and the fact that with early treatment, these morbidities can be prevented warrant early diagnosis[3]. Elio Lugaresi described that Sleep Related Breathing Disorder Continuum has its least severe condition i.e. primary snoring in lower end and severe OSA in the other end with upper airway resistance syndrome (UARS) occupying between them and overlapping severity. Validity of this observation in children is to be studied.

Prevalence

Obstructive sleep-disordered breathing is common in children. Though there are not real epidemiological studies for OSA, the few published reports indicate that PS occurs in 3 to 12 percent of children[4-6].OSA affects 2 percent of children diagnosed by usual adult polysomnography values. There is no difference in sex till adolescence when males tend to have more OSA.

Definition

The consensus is evolving for definition and many researches are based on their centers' own definition for OSA. Apnoea is defined by the American Academy of Sleep Medicine, as cessation of airflow for at least 10 seconds and may last for 30 seconds or even longer[7]. But the time limitation in definition cannot be applied for all age groups as breath rates differ in different age groups from 60 per minute to 12 per minute. To address this, International Classification of Sleep Disorders 2nd edition defined apnoea as a cessation of airflow over two or more respiratory cycles. Again this cannot be applied to neonates, whose two cycles takes only two seconds which is considered as normal in periodic breathing[8-11]. Hypo-apnoea is defined as a recognizable transient reduction of breathing for 10 seconds or longer, a decrease of greater than 50% in the amplitude of breathing, or a reduction in amplitude of less than 50% associated with oxygen desaturation of 4% or more. Arousal is not taken in to account. With above background definitions, when the apnoea - hypopnoea index greater than 1 in a child it is abnormal. The Apnoea Hypo-apnoea Index (AHI) helps grading and is defined as the total number of apnoeas and hypo-apnoeas that occur divided by the total duration of sleep in hour[s1]2

Pathophysiology

The transition from wake to sleep involves muscle relaxation and this includes the pharyngeal dilator, intrinsic and extrinsic tongue muscles, which usually stiffen and maintain the upper airway. This relaxation in healthy individuals results in collapsibility of the airway, leading to increased resistance to air flow resulting in increase of PCO_2 of 3-5mmHg.[13-16] In children with Sleep Disordered Breathing (SDB), there is compensatory increased tone of the muscle in stage 2 sleep, and compensation also occurs by increasing respiratory effort and results in an arousal.[16-18] But children with SDB also show poor ventilator responses to hypercapnia and higher end-tidal CO_2. In severe OSA, apnoea may occur frequently, sometimes 1-2 times per minute. Such apnoeas are accompanied by varying heart rate, desaturations, EEG arousal simultaneously with stertorous breathing sounds as air is exhaled when the critical pressure exceeds and airway reopens.

There are three elements which appear to contribute to the pathophysiology of Sleep Disordered Breathing (SDB) which are anatomy, neuromotor tone and inflammation.

In children, adenotonsillar hypertrophy is an important factor, and it is known that relative large adenotonsillar tissue is at its largest in the first few years of life and then involutes by adolescence[19-21]. In infants and young children who are obligate nasal breathers, nasal obstruction is a factor. Difficult nasal breathing, commonly due to large adenoids in children, leads to chronic mouth breathing and this can lead to anatomical changes in facial growth. Tongue is unable to mould the palate in mouth breathing and results in a narrow, high arched palate and poor maxillary growth, which can also result in narrow nasal passages, narrow sdental arches and an anterior cross-bite. Children who sleep supine tend to have a smaller maxillary width, possibly because lying supine causes the tongue to maintain a more posterior position[22]. Other changes are increased anterior face height and a retro-gnathia, shorter maxilla and mandibular, longer and thicker soft palate and a more inferiorly placed hyoid bone. Macroglossia, glossoptosis, hypopharyngeal collapse, tracheal stenosis, laryngomalacia and recurrent enlarged adenoids are the reasons for Down syndrome developing SDB. Muscular relaxation occurs and genioglossus tone has been shown to decrease more so in patients with SDB compared to controls, when transitioning from wake to sleep[23]. Local inflammation can result in increased resistance, particularly at the adenotonsillar level[24]

Etiology

Anatomical obstruction

Obesity because of the fatty infiltration reducing the airway and hypertrophy of tonsils and or adenoids are common causes in children for obstructive sleep apnoea [25]. But any anomaly of the upper airway may produce intermittent obstructive symptoms during sleep. Facial, oral, and throat anatomical and physiological variations occur in many congenital syndromes. Hypothyroidism, Down syndrome and Storage diseases, result in upper airway crowding due to a relative larger tongue mass compared to mouth size.

Inflammation

It is well known that allergy and chronic inflammation is known to cause obstructive sleep apnoea. Gastroesophageal reflux predisposes to development of SDB[23]

Neuromuscular dysfunction

Chiari malformations and Neuromuscular diseases contribute to obstructive sleep apnoea due to abnormal muscle dysfunction and tone in the pharyngeal constrictors, which are responsible for maintaining airway patency[26-28].

Diagnosis

As in many illnesses the paediatric presentations are different from adults. The symptoms are varied and a single symptom leads us to wrong diagnostic algorithms, but group of such symptoms suggests us Sleep Disordered Breathing. Children also do not exhibit day time sleepiness but are often hyperactive. Only snoring, sleep arousals and witnessed apnoeas are present in all ages.

Snoring, apnoeas noted by parents, frequent arousals, mouth breathing, nocturnal sweating, failure to thrive, nasal congestion, hyper extended neck , recurrent otitis media and upper Respiratory infection are common symptoms seen in infants, toddlers, preschool and school children. In infants the poor suckling, poor day and night cycle, breath-holding spells and noisy breathing are subtle symptoms which may indicate SDB. Day time sleepiness, confusional arousal and restless sleep are symptoms occurring in toddlers, preschool and school children should alert the possibility of SDB. School children exhibit night terror, sleep talking, day time inattention, and hyperactivity.

History, physical examination, abbreviated polysomnography, and full polysomnography are the tools to help in diagnosis, identify candidates for further investigations, identify the candidates who are at risk and identify the management required[12,29-31]. The tools also help in avoiding unwanted interventions.

A sleep history screening for snoring should be part of routine health care visits. In children, OSAS is very unlikely in the absence of habitual snoring. If a history of nightly snoring is elicited, a more detailed history regarding labored breathing during sleep, observed apnoea, restless sleep, diaphoresis, enuresis, cyanosis, excessive daytime sleepiness, and behavior or learning problem, attention-deficit hyperactivity disorder should be obtained. Findings on physical examination during wakefulness are often normal.Evidence of complications of OSAS like systemic hypertension, accentuated second heart sound indicating pulmonary hypertension, and poor growth or obesity may be present.

Children with OSAS experience obstruction primarily during rapid eye movement (REM) sleep, which occurs predominantly in the early morning hours when their parents are not observing. Some children have a pattern of persistent partial upper airway obstruction associated with gas exchange abnormalities, rather than discrete, cyclic apnoeas and do not show pauses and gasps in their snoring, and therefore, the condition may be misdiagnosed as PS[32].

Nocturnal polysomnography or sleep study is the gold standard diagnostic technique and quantifies ventilatory and sleep abnormalities in sleep-disordered breathing and can objectively determine the severity of OSAS and related gas exchange and sleep disturbances. Polysomnography can be performed satisfactorily in children of any age. Studies in children should be scored and interpreted using age-appropriate criteria as outlined in the American Thoracic Society consensus

statement on pediatric polysomnography which can distinguish PS from OSAS 12,[29-31].

Treatment

An AHI of 1-5 is very mildly increased, 5-10 is mildly increased, 10-20 is moderately increased, and greater than 20 severely abnormal[8-11]. Weight control i.e. weight maintenance for a growing child in obese child helps in managing SDB. First line treatment in children is tonsillectomy and adenoidectomy when SDB is diagnosed. Even children with relatively small tonsils or those at risk for SDB for other reasons, such as obesity or Down syndrome may benefit from tonsillectomy and adenoidectomy[33-37]. Treatment of enlarged turbinate is also important in increasing airway diameter by radiofrequency ablation or treatment of allergies with intranasal steroids and or immunotherapy. Continuous positive airway pressure (CPAP) is useful for children who are unable to have T&A or who have residual SDB post operatively[38]. Further long term studies are required for further insight on OSA in children.

OSA	-obstructive sleep apnoea
OSAS	-obstructive sleep apnoea syndrome
SDB	-Sleep disordered breathing
PS	-Primary snoring
UARS	-upper airway resistance syndrome
AHI	-apnoea-hypoapnoea index.

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Smoking Again!
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There is not a single redeeming feature to smoking. Every new study throws up a new evidence to damn it further. The latest is from Institute of Ophthalmology, Zhejiang University in China.Juan Ye and team did an extensive meta-analysis of 16 cohort and 8 case controlled studies done all over the world. They found that the risk of developing age related cataract increased in current and past smokers compared to non-smokers. The types of cataract associated with smoking were found to be nuclear cataract and subcapsular cataract. But, no association was found between cortical cataract and smoking. The results are reported in the latest issue of Investigative Ophthalmology & Visual Science.(J. Ye, J. He, C. Wang, H. Wu, X. Shi, H. Zhang, J. Xie, S. Y. Lee. Smoking and Risk of Age-Related Cataract: A Meta-Analysis. Investigative Ophthalmology & Visual Science, 2012; 53 (7): 3885 DOI:10.1167/iovs.12-9820)

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