# THE RECENT VIEW ON THE OBSTRUCTIVE SLEEP APNOEA SYNDROME IN CHILDREN – SHORT RÉSUMÉ

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**Abstract**

The authors present a recent overview of the common clinical manifestations, management, diagnostic criteria and currently accepted treatment approaches of children with obstructive sleep apnoea syndrome (OSAS). Paediatric OSAS has become widely recognized as a frequent disorder and as a major public health problem. Diagnosis of this problem is usually based on physical examination, history and clinical evaluation confirmed by the polysomnography (PSG). PSG is considering as a gold-standard test for establishing the presence and severity of sleep disordered breathing (SDB) in children. According to current understanding, OSAS is a dynamic process in which increased upper airway collapsibility is present resulting from a combination of structural and neuromotor abnormalities, rather than from structural abnormalities alone. In children the OSAS has completely different clinical features and requires different management strategy. Snoring, difficult breathing and apnoea during sleep, restless sleep, frequent awakening and behavioural disturbances are the typical symptoms usually present in children with OSAS. Nowadays, the classic presentation of child with OSAS as underweight child with adenotonsillar hypertrophy is being replaced by younger overweight or obese patients usually without the hypertrophied adenoids and tonsils. Recently it has been reported that delayed diagnosis of OSAS can lead to neurobehavioural consequences and even serious cardiorespiratory morbidity, metabolic complications, as well as an increase in insulin resistance, high blood pressure and the development of OSAS in adulthood. OSAS must be diagnosed and managed aggressively with having these new repercussions. Evidence suggests that the surgical intervention with removal of the tonsils and/or adenoids will lead to significant improvements in the most incomplicated cases, as recently reported from a meta-analysis. SDB and especially OSAS should be taken into serious consideration by pediatricians to prevent comorbidities in adulthood.

**Key words:** Obstructive sleep apnoea syndrome, snoring, polysomnography, adenotonsillectomy.

**Abbreviations:** AASM – American Academy of Sleep Medicine, AHI – Apnoe hypopnoe index, ALTE - Apparent life threatening events, AT – Adenotonsillectomy, BiPAP - Bilevel positive airway pressure, CPAP - Continuous positive airway pressure, CRP – C-reactive protein, EEG – Electroencephalogram, GERD - Gastroesophageal reflux disease, ICSD – 2 - International Classification of Sleep Disorders, the Second edition, NREM – Non rapid eye movement, OSAS – Obstructive sleep apnoea, PSG – Polysomnography, RDI – Respiratory disturbance index, REM – Rapid eye movement, SDB – Sleep disordered breathing, TST – Total sleep time, URI – Upper respiratory infection.

## INTRODUCTION

Sleep is the primary physiological phenomenon particularly during the childhood [1, 2]. The ontogenic development of sleep is dependent on age. Characteristics of normal sleep change dramatically from infancy through childhood to adolescence. The cycle of sleep/wake begins to evolve during fetal life. The first spontaneous movements have been noted by 10 weeks of gestation age. Electroencephalogram (EEG) reveals the patterns of active and quiet sleep, the forerunners of REM (Rapid eye movement) and NREM (Non-rapid

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eye movement) sleep during the 30 to 32 weeks of gestation. By term, 35-80% of total sleep time (TST) is spent in active sleep. By 12 months REM stage of sleep occupies 20-25% of TST, a proportion that remains through adulthood and sleep is no longer initiated by active sleep. The stages of NREM sleep (I.,II.,III. and IV.) are differentiated by 3 to 6 months of age [3, 4, 5]. An average daily sleep requirements for children shows Table 1.

**Table 1** Average daily sleep requirements for children (adapted from Pearl [3 ])

| Average daily sleep requirements for children | | |
|---|---|---|
| Age | Hours of sleep Total | Hours of sleep During the day |
| 1 week | 16 | 8 |
| 1 month | 15 | 7 |
| 6 months | 14 | 4 |
| 12 months | 14 | 3 |
| 2 years | 13 | 2 |
| 3 years | 12 | 1 |
| 5 years | 11 | 0 |
| 9 years | 10 | 0 |
| 14 years | 9 | 0 |
| Adult | 8 | 0 |

## SLEEP DISORDERED BREATHING

Sleep disordered breathing (SDB) is increasingly being recognised as a cause of morbidity even in children. The International Classification of Sleep Disorders, the Second edition (ICSD-2) by the American Academy of Sleep Medicine, 2005 (AASM) distinguishes five sleep disordered breathing:

– Central Sleep Apnea Syndrome
– Obstructive Sleep Apnea Syndrome
– Central Alveolar Hypoventilation Syndrome
– Central Alveolar Hypoventilation Syndrome associated with other Disorders
– Others Sleep Disordered Breathing [5, 6].

SDB is defined as a spectrum of sleep-related breathing disturbances, resulting from having a structurally narrow airway combined with reduced neuromuscular tone and increased airway collapsibility [7]. Clinical ranging from mild obstruction of upper airway producing primary snoring, through increased upper airway resistance syndrome (UARS) associated with globally rather normal oxygenation patterns, but evidence for increased respiratory - related arousals, sleep fragmentation to continuous events of complete upper airway obstruction producing obstructive sleep apnoea syndrome (OSAS) [8, 9, 10]. The spectrum of SDB occurs in children of all ages, from neonates through infants to adolescents. In prepubertal children there is no significant gender difference, while in teenagers exist a higher incidence of SDB in males [7, 8]. Snoring, the main indicator of increased upper airway resistance during sleep is a frequent symptom during childhood and it is occasionally reported in up to 27% of children. Primary (habitual) snoring is considered as a relatively benign condition and can be characterised as a noisy breathing caused by turbulent airflow through the upper airway without frequent arousals from sleep, events of obstructive apnoe or abnormality of gas exchange [8]. It is estimated to occur in 6 to 12 % of children [7, 9, 11].

## OBSTRUCTIVE SLEEP APNOEA SYNDROME

Obstructive sleep apnoea syndrome (OSAS) is a sleep disordered breathing characterized by a combination of repeated episodes of prolonged partial upper airway obstruction (obstructive hypopnoea) and/or intermittent complete obstruction (obstructive apnoea) that disturb normal sleep pattern, normal ventilation during sleep and resulting in disruption of normal gas exchange (intermittent hypoxia and hypercapnia) [8, 12-14]. Apnoea is a term which is usually used to describe pauses in breathing and is defined as a complete or near cessation of airflow for a minimum of 10 seconds with or without associated oxygen desaturation or sleep fragmentation. It is divided to the central, obstructive and mixed apnoea. An obstructive apnoea is scored when there is a ≥ 90% drop in the signal amplitude of airflow for ≥ 90% of the entire event, compared with the baseline amplitude. The event lasts for at least two breaths (or the duration of two baseline breaths) with continued inspiratory effort throughout the entire period of decreased airflow [15]. During the past decade OSAS has become widely recognized as a frequent and relatively common disorder with potentially serious clinical implications in childhood and has emerged as a major public health problem [8]. Prevalence of OSAS has been traditionally estimated to be 1% to 5% in the paediatric population [11, 16-18]. This syndrome was first described in 1976 in school children, but OSAS has existed for a long time without being widely recognised as a clinical syndrome. In 1889 Hill published an article on some causes of stupidity and backwardness in children and identified the main symptoms of OSAS [6, 11]. Since 1970s and 80s there has been a significant increase in the SDB in children [7]. Although the majority of cases in children population are associated with adenoid and tonsil hypertrophy, OSAS also occurs in children with obesity (13-78%), Down syndrome (57-100%), Prader-Willi syndrome (93%), neuromuscular disorders (Duchenne Muscular Dystrophy 53%), Chiari malformations and myelomeningocele (60%), craniofacial anomalies (achondroplasia 48%, Piere Robin sequence 76% and 50-91% of craniofacial dysostoses) [19]. Intermediate level of risk for OSAS have children with history of prematurity (7.3%), African American race, family history of SDB, allergic rhinitis and chronic upper and lower respiratory tract diseases [1, 12, 20]. Studies of family cohorts suggest that the genetic factors also play an important role in the pathophysiology of OSAS (affect anatomic features and ventilator drive) [10].

## PATHOPHYSIOLOGY

Although the pathophysiology of paediatric OSAS is still not well understood there are some elements such as anatomical structure, neuromotor tone and inflammation which seems to be important in the development of this syndrome. The etiology of OSAS is multifactorial. Pathophysiologic determinants include structural factors (in nonobese healthy children the hypertrophied adenoids and tonsils), which may lead to anatomical obstruction in the pharyngeal airway, role of upper neuromotor tone, genetic predisposition or combination of these factors [11,14]. The peak of occurring main symptoms is 2–8 years of age, when the tonsils and adenoids are the largest in relation to the underlying airway size [1]. In paediatric population with allergic rhinitis, asthma, upper airway respiratory infections, particularly viral and children exposed to cigarette smoke are shown disproportionate proliferation of the adenoids and tonsils [8]. Anatomical obstruction of the airway is especially important in young children who are usually obligate nose breathers. This may lead to chronic mouth breathing and anatomical changes in facial growth. Patients with SDB usually have a structurally narrow airway which predisposes them to having increased resistance and collapsibility when they asleep to lead reduced or absent airflow resulting in apnoeas/hypopnoeas [1, 11]. In the obese children an increase of adipose tissue in the neck, throat and chest wall creates an increase in the work of breathing and also increase in the upper airway resistance [13]. Systematic and local inflammation can also contribute to the

increased resistance at the adenotonsillar level in children. In paediatric population with SDB higher levels of cysteinyl leukotrienes, oedema and inflammatory cell infiltration have been found in tissue removed surgically. But the situation is more complex than simple obstruction by adenotonsillar hypertrophy because not all children with large lymphoid tissue have OSAS. It appears that paediatric OSAS is a dynamic process that involves interactions between sleep state, upper airway mechanics and respiratory drive. It is resulting from a combination of structural and neuromotor abnormalities, rather than from structural abnormalities alone [1, 8, 11].

Children with gastroesophageal reflux disease (GERD) are also at increased risk of developing SDB or vice versa, sleep disordered breathing can precipitate or worsen GER due to increased negative intrathoracic pressures. Apparent life threatening events (ALTE) can be another presentation of witnessed apnoeas [7]. Also increased CRP levels have been demonstrated in these patients. Obese subjects are predisposed to be snorers and to have other symptoms of sleep-related obstructed breathing, which are independently related to the development of hypertension, cardiovascular diseases, behavioral disorders, poor school performance in children, daytime sleepiness, and poor quality of life in adults. Nowadays the pathophysiological mechanism joining OSAS to insulin resistance and dyslipidemia is still not well understood. There is a growing evidence from studies suggesting that the metabolic abnormalities observed in OSAS are most likely the result of sleep disruption, intermittent hypoxia through alteration of the hypothalamic-pituitary-adrenal and autonomic axes, and potentiation of pathways involving inflammatory cytokines and adipokines [8].

## SYMPTOMS

Clinical manifestations, diagnostic criteria, pathophysiology, treatment approaches and PSG findings of paediatric OSAS differ significantly from adults [1, 8, 11]. The presentation in children is much more varied and often difficult to diagnose based on individual symptoms [7]. Excessive daytime sleepiness, the predominant features in adults, is not a significant symptom of paediatric OSAS [7, 11]. Only 7 % of children with sleep disorders breathing present to a physician with excessive sleepiness during the day. Another difference in children is that the symptoms change with age shows Table 2 [7]. Abnormal narrowing in the nose, nasopharynx, oropharynx and hypopharynx causes abnormal air exchange during sleep, which in turn leads to clinical symptoms.

We can divide the symptoms of OSAS in children into daytime and night time symptoms. Difficulty breathing and apnoeic pauses during sleep, snoring, restless sleep and frequent awakening are main nocturnal symptoms and the most common complaints of patients with OSAS. Children with OSAS often snore continuously and loudly. Parents describe episodes of retraction with increased respiratory effort and may be often anxious about their child's breathing during sleep [9, 11]. Other common nocturnal findings are frequently changing sleep positions, sleeping in a position with the head extended in an effort to open his or her airway and increased diaphoresis as a sign of increased effort of breathing at night and changed activity of autonomous system. Parents also reported secondary enuresis. Evidence also exist of an association of primary enuresis with habitual snoring from a population based study. Dry mouth is associated with mouth breathing at night and the patient often keeps water by the bedside. Obese children may prefer sleeping while sitting upright or propped on pillows [7, 9, 11]. These symptoms may exacerbate when the child has a respiratory tract infection [11]. Children with SDB are at increased risk for recurrent upper airway infections such as tonsillitis and otitis media [7]. The classic symptoms during the daytime are usually present as behavioural disturbances - from subtle impairments of learning, attention and behaviour to prominent neurobehavioral deficits that may mimic attention-deficit/hyperactivity disorder-ADHD or learning disabilities [10, 12, 16, 18]. The studies showed universally poorer concentration, attention, behaviour and quality of life in the

**Table 2** The main symptoms of sleep disorders breathing in children by age. Infants (3-12 months), Toddlers (1-3 years), Pre-school (3-5 years), School (5-18 years). Adapted from Sinha [7]

| Infants | Toddlers | Pre – school children | School children |
|---|---|---|---|
| Snoring | Snoring | Snoring | Snoring |
| Witnessed apnoeas | Witnessed apnoeas | Witnessed apnoeas | Witnessed apnoeas |
| Arousals | Arousals | Arousals | Arousals |
| Mouth breathing | Mouth breathing | Mouth breathing | Mouth breathing |
| Dry mouth | Dry mouth | Dry mouth | Dry mouth |
| Noctural sweating | Noctural sweating | Noctural sweating | Noctural sweating |
| Failure to thrive | Failure to thrive | Failure to thrive | Failure to thrive |
| Hyper extended neck | Hyper extended neck | Hyper extended neck | Hyper extended neck |
| Nasal congestion | Nasal congestion | Nasal congestion | Nasal congestion |
| Recurrent URI | Recurrent URI | Recurrent URI | Recurrent URI |
| Noisy breathing | Noisy breathing | Drooling | Nightmares |
| Poor suck | Sleep terrors | Sleep terrors | Sleeptalking |
| ALTE | Confusional arousal | Confusional arousal | Sleepwalking |
| Poor day/night cycle | Irritability | Sleep walking | Confusional arousal |
| Stridor | Daytime sleepines | Daytime sleepines | Daytime sleepines |
| Breath holding spells | Restless sleep | Persistent naps | Restless sleep |
| | | Restless sleep | Enuresis |
| | | Enuresis | Hyperactivity,inattention |
| | | Hyperactivity | Droolong |
| | | Difficulty waking up | Difficulty waking up |
| | | Morning headache | Morning headache |
| | | | Insomnia |
| | | | Learning difficulties |
| | | | Delayed puberty |
| | | | Crossbite,malocclusion |
| | | | Mood disturbance |
| | | | Depression |
| | | | Hypertension |

child and also in parents and family [11]. Mouth breathing is associated with structural changes in the face, known as “adenoid face”. More severe causes of OSAS may be associated with failure to thrive, somnolence, growth retardation and with many deadly diseases such as systemic hypertension, metabolic syndrome, cor pulmonale, pulmonary hypertension and developmental delay [8, 12, 21].

## DIAGNOSIS

OSAS in children has completely different clinical features and requires differ management strategy from adult OSAS [11]. The process of diagnose the paediatric OSAS continues to evolve as more morbidity is recognized and more precise diagnostic methodologies become available [9]. Nowadays, diagnostic approaches to paediatric OSAS vary widely. On one hand PSG has been recommended to establish the presence and severity of SDB in children population by the American Academy of Pediatrics (AAP) and the American Thoracic Society. On the other hand, some otolaryngologists decide to perform adenotonsillectomy based on the physical examination and personal history of the patient. Well-designed prospective cohort studies showed, that the clinical parameters such as history, physical examination, standardized questionnaires and audio or visual recording have poor positive value for OSAS in children and do not consistently identify the presence or absence of OSAS when compared with PSG. PSG parameters correlated best with a combination of symptoms

and signs rather than for any individual parameter. PSG in children is a reliable and valid measure of the presence of OSAS [11, 19]. In 2011 American Academy of Sleep Medicine (AASM) published the Practice Parameters for the Respiratory Indications for Polysomnography in children with SDB. It is reported that the evaluation for SDB mostly based only clinical evaluation alone does not have sufficient specificity and sensitivity to establish a diagnosis of OSAS. The gold standard for the diagnosis of SDB is overnight PSG. It also demonstrates the severity of OSAS, which is helpful in planning treatment and in postoperative shortand long-term management [22]. Nowadays, some questionnaires have been created and developed in an effort to find a simple screening instrument to identify subjects who are at high risk for OSA. The results of these questionnaires are still controversy [6, 15, 17].

A diagnosis of OSA is usually confirmed by a laboratory PSG revealing RDI (Respiratory disturbance index) > 5 respiratory events/hour, according to the AASM criteria [17]. Some experts use the AHI (apnoea hypopnoea index - the average number of apnoe and hypopnoea episodes per hour) as the main parameter; others use RDI while it includes minor respiratory events such as RERA (respiratory event related arousal) or changes in the pleural pressure [14]. AHI greater than 1 is abnormal in a child [10].

## DIAGNOSTIC PROCESS OF OSAS IN CHILDREN

The diagnostic recommendations to children with suspected OSAS are following:

1. Screening for OSAS. Every child should be screened for snoring, apnoe and arousals during sleep. If child or adolescent snores or presents another signs and symptoms of OSAS, clinicians should perform a more focused evaluation.
2. History (Family diseases, history of the present illness, past and acute medical history, review of systems, social history, gynecological history).
3. Physical objective examination.
4. Questionnaire.
5. Otorhinolaryngology.
6. The gold standard test is being considered nocturnal in-laboratory PSG.
7. If PSG is not available, other alternative diagnostic methods such as nocturnal oximetry, nocturnal video recording, daytime nap or ambulatory PSG are needed [22].

## TREATMENT

Interventions for paediatric OSAS are varied and often multidisciplinary. The goal of treatment is to restore optimal breathing during night and to relieve associated symptoms (improved daytime functioning and minimizing negative impact). The most common definitive treatment in children is surgical intervention with removal of the tonsils and/or adenoids (adenoidectomy, tonsillectomy or adenotonsillectomy), which provide more airway space. Outcome-based data report the effectives of adenotonsillectomy (AT) as the first line surgical procedure for uncomplicated OSAS in children with hypertrophy of lymphoid tissues [20]. The main risk of AT is postoperative haemorrhage and there is an increased risk of perioperative respiratory complication [6]. Nasal CPAP (Continuous positive airway pressure) or BiPAP (Bilevel positive airway pressure) are used for children who are not good surgical candidates, have SDB associated with major craniofacial deformities, residual OSAS after upper airway surgery or who have failed previous surgical treatment. This therapy is sometimes not good tolerated by young children and also by their parents. Important is training of the family and child, behavioural modification techniques, daytime training as well as finding the appropriate nasal interface. Various alternative treatments (craniofacial surgery or orthodontic treatment such as rapid maxillary distraction and oral appliances)

are used on an individualised basis that cannot underwear the two first-line therapies for sleep apnoea [18]. Tracheostomy is rarely indicated [12]. It is now being investigated the incorporation of nonsurgical approaches for milder forms of the disorder and for residual OSAS after AT. An intensive weight reduction program is an important first line step for obese or overweight paediatric patients, which can also lead to improvement in number of child with OSAS. Some recent randomized controlled studies have demonstrated beneficial effects and significant reduction of the RDI with using of intranasal steroids in paediatric patients with mild OSAS. It is reported that leukotriene receptor antagonists have also resulted improvements in sleep-related respiratory disturbance in children [8]. Good sleep hygiene is very important for any individuals [13].

There is still a continued debate whether AT should be routinely based on clinical suspicion of OSAS or whether objective confirmation of the diagnosis with PSG should be required before surgery. This question – selection of patient for treatment/clinical evaluation versus PSG remains still controversial. The arguments supported PSG before AT are following: it is a standard of care for adults with suspected OSAS, PSG can provide objective confirmation of the presence of OSAS, it can help predict, which children are at increased risk for postoperative airway complications after AT and other objective screening tools (oximetry, validated questionnaires) show much more limited specificity and sensitivity. Equally compelling arguments are against the requisite use of PSG. Many sleep children laboratories have limited experiences in doing and interpreting PSG in children, norms for interpretation of paediatric PSG are still not well established, the lengthy waiting lists can result to delayed treatment for some symptomatic children, routine PSG is not always sensitive to detective UARS and specialized technology for detection of these disturbances is not widely available [13].

## CONCLUSION

Paediatric sleep medicine is a dynamic, complex and multidisciplinary field of medicine. In the diagnosis and management of SDB in children has performance of nocturnal PSG strong clinical utility. Better understanding of individual risk factors and importance of sleep disturbances in children population, improved screening, diagnostic tools and methods of the patients suspected of SDB, using evidence-based treatment modalities seems to be important goals for future research [23]. Early identification of OSAS is desirable, because it is a highprevalence disorder and identification, accurate diagnosis and treatment can result in alleviation of current symptoms, improved quality of life, prevention of sequelaes, education of parents and decreased health care utilization [13, 22].

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