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Review

Obstructive Sleep Apnoea: Children are not little Adults

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EDUCATIONAL AIMS

- To highlight the importance of the physiological interactions between sleep and breathing and the impact of sleep on respiration in children.
- To illustrate the unique clinical manifestations of OSA in children, which will enable practitioners to recognize the symptoms of OSA in this group of patients.
- To describe the differences between the overnight polysomnographic characteristics of children and adults.
- To provide updates on the treatment modalities for OSA in children, as well as their outcomes.

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SUMMARY

During a child's development, several important developmental physiological sleep processes occur, and, occasionally, pathological disorders occur, which results in differences between obstructive sleep apnoea (OSA) in adults and children. There are major differences in sleep and respiratory physiology as well as OSA symptoms and treatment options between children and adults. Many practitioners do not realize these differences, which results in delays in the diagnosis and treatment of OSA in children. The treatment options for OSA in children are markedly different compared with adults, effective in most children. The use of positive airway pressure (PAP) therapy delivered through continuous or bi-level positive airway pressure modes is successful in children and even in infants; however, there are several challenges facing parents and practitioners to achieve good compliance. The early recognition and treatment of paediatric OSA are essential to prevent deleterious consequences. This article discusses the major differences between paediatric and adult OSA and demonstrates why children are not little adults.

INTRODUCTION

Sleep is a major physiological drive. While newborns sleep as much as 16 hours per day, an average child spends almost half of his or her life asleep. During this long sleep period, several physiological processes occur, and, occasionally, pathological disorders are manifested. Obstructive sleep apnoea (OSA) is one of the major sleep disorders of childhood. Some respiratory disorders worsen or may only occur during sleep. Therefore, it is essential for clinicians to understand the interaction between sleep and breathing and the impact of sleep on respiratory disorders in children. There are major differences in sleep and respiratory physiology as well as OSA symptoms and treatment options between children and adults. Many practitioners do not realize these differences, which results in delays in the diagnosis and treatment of OSA in children [1]. In this review, we discuss the major differences between paediatric and adult OSA and demonstrate why children are not little adults.

WHY ARE CHILDREN NOT 'LITTLE ADULTS'?

Sleep in Infants

In the newborn, the daily sleep duration ranges from 14 to 16 hours, and newborns spend two-thirds of their total sleep time

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in active sleep (AS), which is equivalent to rapid eye movement (REM) sleep in older infants, children and adults. Subsequently, the time spent in AS decreases to 20-25% at the age of 5 years to become similar to the percentage of REM sleep in adults. As the child grows, the sleep duration decreases. In newborns and infants, sleep is divided into multiple periods of sleep. At 5-6 months of age, nocturnal sleep consolidation occurs and the infant sleeps at night more than during the day, with at least one nap per day.

Normal Physiology during Sleep

During development and maturation, several physiological changes occur during sleep, which are described below.

Changes in Respiratory Function

In adults, minute ventilation decreases during sleep by approximately 13-15% compared to the awake state. The ventilatory drive primarily diminishes during REM sleep in sleeping humans [2]. The respiratory rate does not change substantially because the reduction occurs mainly in the tidal volume [3]. By contrast, studies in children revealed a decrease in their respiratory rate during sleep [4]. However, there are limited studies that assessed the tidal volume and minute ventilation in healthy children [5,6]. During sleep, the functional residual capacity decreases and the upper airway resistance doubles in normal humans [7,8].

While respiration is regular during NREM sleep, REM sleep is characterized by irregular breathing in terms of the variable respiratory rate and tidal volume. In children, the suppression of the tonic muscle activity of the intercostal muscles leads to a further decrease in functional residual capacity, while the concurrent activity of the diaphragm muscle remains stable. This mechanism results in paradoxical chest and abdominal movements during sleep that usually disappear by 3 years of age [9]. Because children have a high percentage of REM sleep, they are prone to REM-associated sleep disorders such as OSA.

Thoracic Mechanics Development

There are several chest wall and upper airway mechanical changes that occur during infancy and childhood to adapt to the physiological needs of the developing child. In infancy, chest wall compliance is nearly triple the lung compliance, which results in a paradoxical inward rib cage motion during REM sleep. In normal children, high chest wall compliance continues until 3 years of age, and by the second year of life, the chest wall stiffness increases gradually until the chest wall and lungs have almost equal compliance, as in adulthood [10,11]. Moreover, the shape of the rib cage changes during infancy and early childhood. In infants, the chest is barrel-shaped with more horizontally placed ribs, resulting in limitations of the potential increase in thoracic expansion, particularly with the smaller zone for the apposition of the diaphragm. Therefore, the shape of the rib cage movement contributes to tidal breathing during quiet sleep(QS) and is only one-third in neonates compared with two-thirds during NREM sleep in older children [12].

The muscle mass in infants and young children is very low compared with older children and adults [13]. Therefore, when infants require high inspiratory pressure, the diaphragm works at a level close to the diaphragmatic fatigue threshold. Thus, infants are prone to decompensate if they develop OSA or respiratory tract infections [14].

Upper Airway Development

Because of increased chest wall compliance in infancy, infants have a lower functional residual capacity and are more prone to atelectasis compared with older children and adults. To actively increase their Functional Residual Capacity(FRC), infants utilize a mechanism termed laryngeal braking until 6 to 12 months of age(active laryngeal narrowing during expiration) [15]. Moreover, the cephalic location of the larynx during infancy allows the epiglottis to overlap the soft palate and thus enables infants to make a better seal for suckling [16]. However, this mechanism makes them at risk for upper airway obstructions if the nasopharynx is partially obstructed.

During the prepuberty period, there is no gender difference in the incidence of OSA; however, after puberty, the incidence increases in boys more than in girls. Theoretically, this difference could be explained by the testosterone-induced changes in upper airway morphology, as testosterone flow in boys during puberty leads to muscle mass enlargement. This difference may increase the risk for OSA in adolescent and adult males compared with females [17,18]. The increase in muscle mass could explain the reappearance of OSA in teenagers with relatively small upper airways. However, not all children with abnormal craniofacial morphometric features will develop OSA as teenagers [17].

Other maturational changes include an increase in the lymphoidal tissues of the upper airway from birth until the age of 12 years, accompanied by the gradual growth of the skeletal boundaries of the upper airway [19].

Upper airway patency is affected by a balance between the structural and neuromuscular factors. Imbalance of these factors will lead to an increased upper airway collapsibility and hence result in OSA. In response to upper airway obstruction, children maintain better patency of the upper airway compared with adults due to increased neuromuscular activation of the upper airway during sleep that is secondary to the increased central ventilatory drive [20]. Although obese adolescents have vigorously active upper airway reflexes during sleep, which help maintain upper airway patency, these reflexes normally decline with age and patients may end with OSA as they enter adulthood [21]. As the upper airway compensatory response to subatmospheric pressure loading decreases with age rather than degree of pubertal development, changes in sex hormones are unlikely to be the primary modulator of upper airway function during the transition from childhood to adulthood and that changes in upper airway function with age could be due to the depressant effect on ventilatory drive, leading to a decrease in upper airway neuromotor tone [22]. Nevertheless, obese adolescents without OSA have narrowed upper airway secondary to adipose tissue; however, they are protected from OSA by upper airway neuromotor activation. Thus neither neck circumference nor visceral adipose tissue measurements in obese adolescent are beneficial predictors of upper airway collapsibility without taking into consideration neuromotor factors during sleep [23].

Developmental Changes in Ventilatory Controllers

Almost all functions of the respiratory control system undergo postnatal changes in maturation. In the neonatal period, there is an increase in the sensitivity of the arterial chemoreceptors to hypoxia, which is known as the "resetting" of carotid and aortic chemoreceptors [24]. Compared with adults, infants have an extreme response to hypoxia, which is initially expressed as increased ventilation, followed by a reduction of ventilation below the baseline, which may lead to apnoea [25].

Laryngeal chemoreceptors (LCRs) located in the surrounding airway epithelium respond to hypochloraemic or strong acidic solutions by reflexes such as rapid swallowing, laryngeal constriction, apnoea, hypertension and bradycardia [9]. LCRs exhibit peak activity in the postnatal period compared with later infancy and childhood. In addition, the pattern of responses changes significantly with maturation. In the neonatal period, the reflexes appear in the form of swallowing and apnoea, whereas in

older infants and children, the reflexes present in the form of a cough and laryngeal constriction [26,27].

In addition to the anatomical factors, functional factors such as abnormal ventilatory drive and neuromotor tone contribute to the pathophysiology of OSA. Previous studies demonstrated that the central ventilatory response to hypoxia and hypercapnia are normal during wakefulness and sleep in OSA [28,29]. Other studies have shown that children with OSA have depressed spontaneous ventilation under anesthesia and have diminished ventilatory response to carbon dioxide (CO_2). Yuan et al, demonstrated that the ventilatory response to hypercapnia during wakefulness is higher in obese adolescents with or without OSA compared to lean controls [30]. However, obese adolescents with OSA have blunted ventilatory response to CO_2 during sleep [30]. Moreover, obese adolescents with OSA do not have a compensatory prolongation of inspiratory time, despite having normal CO_2 responsivity during wakefulness, which indicates that abnormalities in central ventilatory drive may play a role in the pathophysiology of OSA in obese adolescents [30].

Arousal

Arousal is a vital protective mechanism against apnoea. Arousal could be "spontaneous" as a part of the normal sleep, or as a response to apnoeas, hypopneas and external stimuli [31]. Autonomic activation and the ventilation changes in response to arousal occur in all sleep stages; nevertheless, the magnitude is greater in NREM sleep in normal humans [32]. Parslow et al. demonstrated that infants are less arousable to mild hypoxia during QS than during AS, and the younger the child, the higher the arousal threshold [33,34]. In general, children have a higher arousal threshold than adults. In children, the major stimuli for arousal are respiratory load and hypercapnia. By contrast, hypoxia is a weaker stimulus to arousal. Ward et al. showed that only 32% of infants were aroused in response to hypoxia [35]. The arousal threshold in response to upper airway obstruction is low during REM and stage N2 sleep and the greatest during stage N3 sleep [36]. van der Hal AL et al. showed that healthy infants and infants with apnoea of infancy aroused to hypercapnia; however, healthy infants aroused at a lower inspired PCO_2 (inspired PCO_2 40.1 +/- 2.6 mm Hg) than those with apnoea of infancy (inspired PCO_2 46.9 +/- 1.5 mm Hg, P less than .05) [37]. In general, children have lower spontaneous arousal than adults. It has been postulated that infants with failure to arouse from sleep are prone to develop a sequence of events leading to sudden infant death syndrome(SIDS) [38].

Apnoeas

Apnoea is the cessation of respiratory airflow and has three major types: central, obstructive or mixed. Central sleep apnoea results from absent respiratory drive from breathing centers in brain stem during sleep. The duration criterion required to score an apneic event has obviously changed over the last few decades. It was 2 minutes in 1956, then 1 minute in 1959, followed by 30 seconds in 1970, and finally 20 seconds or shorter if associated with bradycardia or cyanosis in 1978 [39–42]. Brief episodes of apnoea is a normal occurrence in infants, but prolonged apnoeic episodes may lead to morbidity and rarely mortality [43]. Thus the progressive reduction of the duration criterion in the definition of apnoea indicates doctors' desire to interfere early enough to avoid systemic consequences. The diagnosis of apnoeas should follow the American Academy of Sleep Medicine (AASM) Manual Scoring of Sleep and Associated Events. The AASM criteria for scoring central apnea in children indicate that central apnea should be scored if there is a drop in the peak signal excursion by $\geq$90% of pre-event baseline using an oronasal thermal sensor, and the event is associated with absent inspiratory effort throughout the entire duration of the event. With regard to the duration of the event, the AASM criteria require at least one of the following conditions: the event lasts $\geq$20 seconds; the event lasts at least the duration of two breaths during baseline breathing and is associated with an arousal or a $\geq$3% arterial oxygen desaturation; or the event lasts at least the duration of two breaths during baseline breathing and is associated with a decrease in heart rate to less than 50 beats per minute for at least 5 seconds or less than 60 beats per minute for 15 seconds (infants under 1 year of age only) [44]. CA occurs frequently in infants and young children, particularly during AS and REM sleep, after a sigh, upon body movement or during the transition from wake to sleep [4,45,46]. Respiratory instability primarily appears during REM sleep in children due to the immaturity of the brain stem centre and phasic inhibitory/excitatory mechanisms inherent to REM sleep [47]. The frequency of CA decreases after the first year of life [9].

Numerous data on normal infants have revealed prolonged CAs up to 25 seconds in association with transient desaturations [48]. Therefore, the clinical significance of CAs is questionable, unless they occur frequently or are associated with prolonged gas exchange abnormalities [49]. By contrast, obstructive apnoeas (OAs) are extremely rare in normal children. In a study of 50 children and adolescents aged 1-18 years old, the mean OA index was 0.1±0.5/h and only 18% of them had one or more OAs, although none of the events lasted more than 10 seconds and no mixed apnoeas occurred in any of the children [50].

Gas Exchange

Hypoxic and hypercapnic ventilatory drives diminish during sleep. Subsequently, normal children experience a small increase in the partial pressure of carbon dioxide (P_aCO_2) and a small decrease in the arterial oxyhemoglobin saturation(SpO_2) compared to wakefulness, which are similar to the changes observed in adults [3]. The magnitude of these changes has not been systematically studied in a large sample of healthy children; however, it is believed that there is an average drop of 2% for SpO_2 and an average increase of 4 to 6 mmHg for P_aCO_2 [51]. These sleep-related changes in ventilation, upper airway stability and gas exchange can be exaggerated in children with an underlying lung disease, upper airway abnormalities and neuromuscular disorders, thus resulting in increased vulnerability to OSA.

COMMON DIFFERENCES BETWEEN PAEDIATRIC AND ADULT OBSTRUCTIVE SLEEP APNOEA (OSA)

OSA is the most common form of sleep disordered breathing (SDB) in children and is characterized by repetitive partial and or complete upper airway obstruction that interrupts normal sleep patterns and ventilation. Therefore, OSA induces intermittent hypoxia and/or frequent arousals [52].

There are many similarities and differences between the clinical manifestations and risk factors for snoring and OSA in paediatric and adult populations, although the treatment options are different.

Prevalence

OSA occurs in children of all ages and affects approximately 1–2% of children [53,54]. The peak prevalence of OSA in children occurs between 2 and 8 years, due to pharyngeal lymphatic tissue hypertrophy (i.e., adenotonsillar hypertrophy) (Figure 1) [53,55]. Compared with adults, in whom OSA is more frequent in males, OSA affects children of both sexes equally [18].

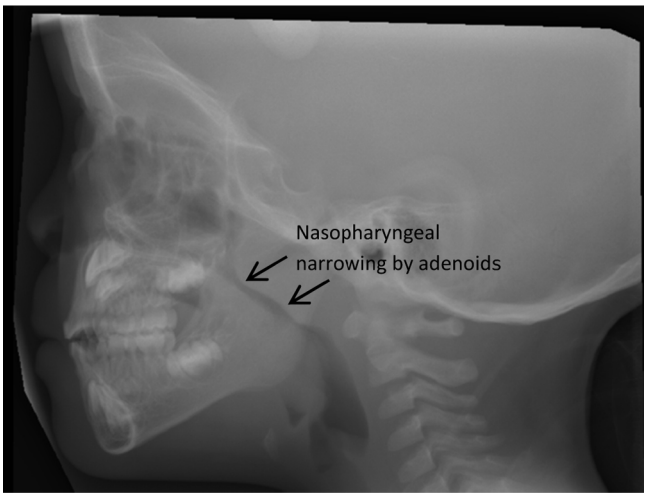

Figure 1. Lateral neck X-ray in a child with obstructive sleep apnoea showing nasopharyngeal narrowing secondary to enlarged adenoids.

Clinical Manifestations of OSA

Evaluation

History. In adults, sleep complaints are raised by the patient or his/her bed partner. However, in children, the concerns are frequently raised by parents. Parents are usually alarmed by the child's snoring and their concerns are strongly affected by whether the child disturbs their sleep. Thus, if the child is developing normally, a detailed sleep history may provide additional important information. While snoring is the most common and severe complaint in both children and adults with OSA, witnessed apnoea in children with OSA was significantly less common than in adults with OSA [56]. Other symptoms, such as daytime sleepiness, morning headache, memory impairment and daytime fatigue, are more prevalent among adult patients with OSA.

Compared with adults, children have unique presentations, such as hyperactivity, emotional difficulties, decreased academic performance and difficulty in concentration [57,58].

Examination. A general examination of a child suspected to have OSA may reveal the appearance of failure to thrive or obesity in young children with severe upper airway obstruction. Proper assessment of the craniofacial structures is necessary to detect abnormalities, such as mid-facial hypoplasia, micrognathia or retrognathia. Moreover, clinical assessment for any syndromal diagnoses such as Down syndrome, Pierre Robin syndrome or Crouzon syndrome is needed. An evaluation of the oral cavity including the tongue, tonsillar size, and the shape of the palate and uvula is also informative.

Although the auscultation of the lung is usually normal, signs of longstanding obstruction such as pectus excavatum and Harrison sulcus or signs of pulmonary hypertension should be monitored. A physical examination must also include a neurological survey for hypotonia, which may be found in patients with neuromuscular disorders (Table 1).

Table 1
Signs that should be evaluated during an assessment of a child with suspected OSA.

Failure to thrive, obesity
Craniofacial abnormalities, micrognathia, retrognathia, adenoidal face
Underlying syndromal diagnoses: e.g., Down syndrome, Pierre Robin
Nasal and oral cavity: Macroglossus, high arch palate, adenotosillar hypertrophy
Signs of longstanding obstruction: Pectusexcavatum,Harrisonsulcus, signs of pulmonary hypertension
Nervous system examination: Hypotonia

Aetiology

The aetiology of paediatric OSA is multifactorial. OSA peaks in the preschool age group, in which the increased adenotonsillar growth is the largest in relation to the upper airway size. Although adenotonsillar hypertrophy is clearly an important risk factor for paediatric OSA, there is no absolute correlation between the size of the adenoids and tonsils and the presence of OSA [59–61]. In adult patients with OSA, adenoid and tonsillar hypertrophy are uncommon, whereas obesity is one of the major risk factors. Nevertheless, obesity is increasingly becoming an important and common risk factor of late childhood and adolescent OSA [56].

Childhood OSA is a dynamic process resulting from a combination of structural and upper airway neuromotor abnormalities. Marcus et al included 52 subjects to measure dynamics upper airway response during sleep across the age span from infancy through adulthood and upper airway response was tested in several ways including both brief and prolonged application of sub-atmospheric pressure administration [62]. In addition, upper airway response was tested in some of children and adults by administrating carbon dioxide during sleep. This study has showed that adults have more collapsible upper airway during sleep than infants and children. Children have showed a vigorous response to both sub-atmospheric pressure administration and hypercapnia during sleep ($P<0.01$), whereas adults had no significant changes. Infants had an airway that is resistant to collapse and showed very rapid response to sub-atmospheric pressure administration during sleep, which indicate the presence of active upper airway reflexes in young children during sleep although these reflexes decrease during adolescence. Upper airway compensatory response to sub-atmospheric loading decreases with age due to the depressant effect of age on the degree of ventilatory drive, which leads to a decrease in upper airway neuromotor tone indepdent of puberty development [22]. In another separate study, Hugn et al demonstrated that obese adolescents without OSA have a strong compensatory neuromuscular response to sub-atmospheric pressure load during sleep, making them less likely to develop upper airway collapse, whereas those with OSA have weak protective airway reflexes during sleep [21].

Other risk factors for childhood OSA include an impaired neural response, an abnormal central arousal mechanism, congenital and craniofacial abnormalities and a combination of structural defects and neuromuscular factors, which predispose the child to obstructed breathing during sleep.

Investigations

Lateral neck radiography or flexible naso-pharyngoscopic examinations to evaluate the adenoidal tissue size and the site of airway obstruction should always be performed. Even if the posterior nasal space is patent during the awake state, its patency may decrease during sleep, particularly in REM sleep when the muscle tone is diminished. Nevertheless, lateral neck radiography does not correlate with the presence or severity of OSA [61].

Polysomnography (PSG)

Differences between paediatric and adult overnight polysomnography(PSG)

A patient's history and physical examination alone are unreliable to diagnose or determine the severity of OSA in adults and children [63,64]. Overnight PSG is the gold standard diagnostic test to diagnose and assess the severity of OSA, allowing the

analysis of sleep stages, respiratory movements, airflow, and gas exchange. This recording should be performed with minimal disruption to the child's usual sleep patterns, which requires a child-friendly environment and a special approach. Although the clinical characteristics and polysomnographic findings of paediatric OSA have been described in several studies, little attention was paid to the differences in the PSG between children and adults with OSA [56].

There are several important differences between PSG studies of children and adults that may affect the interpretation of the sleep study. A sleep study in children requires well-trained sleep technicians who have experience and skills in child care. There should be collaborative efforts by the health care providers to make the environment non-threatening by allowing a parent to stay with the child throughout the night and allowing a period after the child is connected to the equipment for the child to become familiar with his/her surroundings. Even with all of these measures, the young child's tolerance for the study may affect the quality of the data obtained. Therefore, sleep studies must be reported by an expert sleep physician who understands the developmental neuro- and cardiorespiratory physiology of children [65].

There are several differences in the characteristics of OSA in children and adults during overnight polysomnography (Table 2).

Treatment

Surgical Treatment

Unlike adults with OSA, adeno-tonsillectomy (TA) is the first-line treatment in most paediatric OSA cases [63]. TA results in subjective (symptoms) and objective (PSG parameters) improvements in the majority of paediatric OSA patients with a cumulative cure rate of 80% and a significant reduction in health care utilization [75,76]. Both the American Academy of Pediatrics and the American Academy of Otolaryngology-Head and Neck Surgery recommended close postoperative oximetry monitoring overnight for paediatric OSA patients at high risk for respiratory complications [63,77]. Moreover, both societies recommended that TA for children with should be performed in a centre with experts in managing airway problems [63,77]. Post-operative respiratory compromises among children with OSA have been reported to occur in up to 27% of patients, particularly in high-risk patients, such as children younger than 3 years, patients with severe OSA, cardiac complication of OSA(e.g., right ventricular hypertrophy), failure to thrive (FTT), obesity, craniofacial anomalies, neuromuscular disorders or a current respiratory infection [63,78,79] In a study that assessed the post-operative complications of urgent TA, Brown et al. reported that severe OSA (defined as SpO_2 nadir <80%) and associated medical conditions (cardiorespiratory, neuromuscular, or craniofacial comorbidities) were associated with increased respiratory complications in the post-operative period [80]. Nevertheless, a recent study conducted on 221 school-aged healthy children with AHI range of 1.2-27.7/h and obesity prevalence of 31% reported a low risk of post-adenotonsillectomy complications [81]. Moreover, none of the polysomnographic or demographic parameters predicted post-operative complications. A more recent prospective, observational study conducted on 329 children with OSA revealed that PSG predicted perioperative respiratory, but not non-respiratory complications [82]. The following PSG parameters were predictors of respiratory complications: apnea hypopnea index, SpO_2 nadir, sleep time with SpO_2 <90%, peak end-tidal CO2, and sleep time with end-tidal CO2 >50 mm Hg. Associations were also found between respiratory complications and age <3 years or black race. Based on the above, it is obvious that there is no agreement on the proper predictors of post-operative complications of adenotonsillectomy. Therefore, clinical evaluation is still needed to determine which children with OSA need pediatric hospital and to be admitted after adeno-tonsillectomy.

A follow up PSG at 6-8 weeks after the operation is necessary to ensure that no additional treatment is required for high-risk patients [52].

Medical Treatment

Positive Airway Pressure(PAP)

In adult patients, positive airway pressure (PAP) therapy is the mainstay treatment of OSA. However, in children with OSA, PAP therapy has an important role, but only in a small group of paediatric OSA children who do not respond to TA or in whom the major cause of the upper airway obstruction is not adeno-tonsillar hypertrophy (such as patients with obesity or craniofacial abnormalities). The continuous positive airway pressure (CPAP) treatment is well-tolerated in infants and older children with OSA and is highly effective [83,84]. The use of PAP is successful in eliminating obstructive events, even in infants [85]. Practitioners face several challenges when trying to use PAP therapy in children with OSA, which include difficulty in finding the appropriate equipment and interface, particularly for infants and young children, the intolerance of the child to the CPAP machine, and the rejection of the mask. Most of the commercially available PAP machines that have an S/T mode were designed to be used on adults and hence have a respiratory rate that is inadequate for infants and young children, as they have a higher baseline respiratory rate [67,86].

Auto-titrating PAP devices have been used in adult patients with OSA for unattended CPAP titration at home [87]. However, the

Table 2
Differences between pediatric and adult OSA overnight Polysomnography (PSG)

PSG	Pediatric OSA	Adult OSA
Apnoea definition	Cessation of airflow (drop in the peak signal excursion by ≥90%) for at least 2 respiratory cycles [56,66].	Cessation of airflow (drop in peak signal excursion by ≥90%) for ≥ 10 seconds.
Obstruction	Persistent partial upper airway obstruction (hypoventilation) Figure 2 [67].	Frank and cyclic partial or complete upper airway obstruction.
Transcutaneous CO_2	Rise of $PaCO_2$ [52,65,68].	In selected cases
Cortical Arousals	Low frequency of arousals secondary to respiratory events(only20% of obstructive apnoea are followed by cortical arousal) [52,56]	Usually apnoeic episodes are followed by arousal.
Sleep Architecture	Normal sleep architecture	Fragmented sleep and decrease sleep efficiency [69].
State of OSA	Obstructive apnoea and hypopneas are a REM related phenomena [52,56,70].	Usually occur in both REM and NREM sleep [71,72]
Severity of OSA	Mild >1 and <5 events /hour Moderate>5 events/hour Severe >10 events/hr [73]	Mild 5-15events /hour Moderate 15- 30 events/hour Severe > 30 events/hour [74]

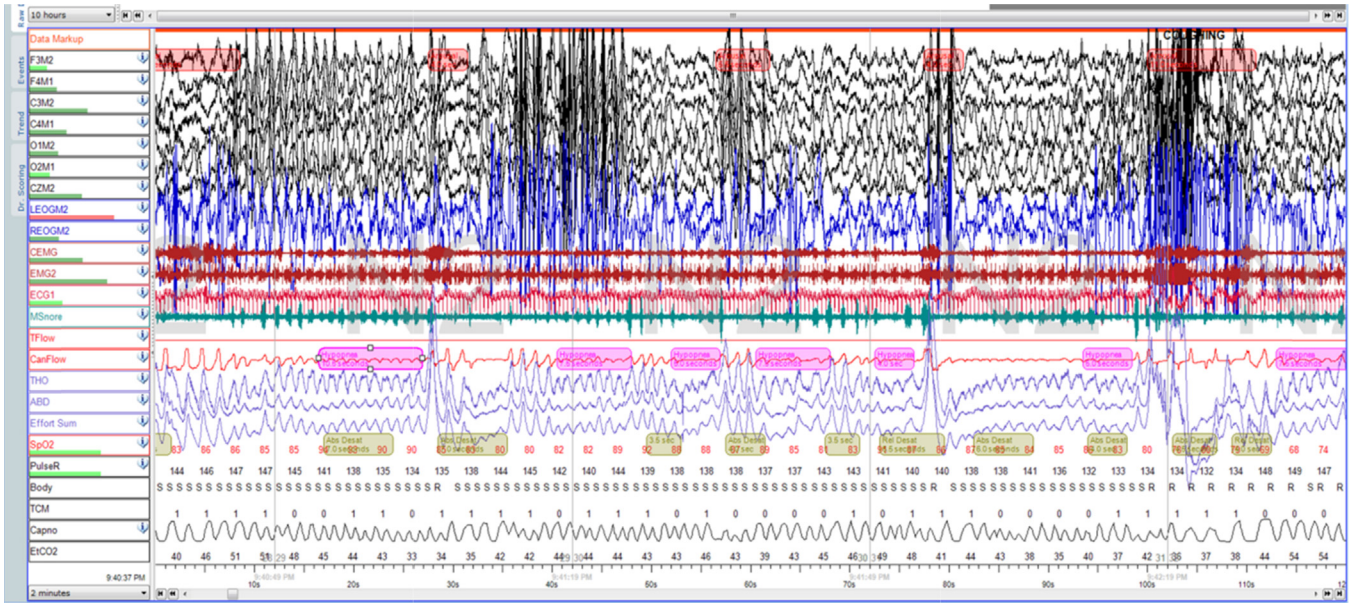

Figure 2. A magnified2-minute Epoch showing examples of obstructive hypopnea. The record demonstrates a reduction in airflow associated with continuing paradoxical respiratory effort and oxygen desaturation, followed by arousal.

evidence supporting the efficacy and safety of using auto-titrating PAP devices on children is lacking.

Unlike adult patients with OSA, who do not exhibit dramatic changes in the severity of OSA over time, in children, OSA can be viewed as a dynamic disorder, where in the AHI may change frequently, particularly during growth periods or when a significant weight gain or loss has occurred. Therefore, periodic PSG re-assessment and re-titration of the PAP therapy is recommended for children with OSA [86].

Although most children use PAP devices effectively without side effects, some children may have complications similar to those observed in adult patients, such as local skin irritation and erythema, nasal bridge ulceration or discomfort secondary to poor mask fitting or too tight headgear, eye irritation or conjunctivitis, and aerohagia.

Complications and Morbidities

Cardiovascular Complications

The intermittent repetitive complete upper airway obstruction and desaturation with subsequent changes in autonomic nervous system result in a negative influence on the cardiovascular system. In adults, OSA is well-known to be associated with serious sequelae including cardiovascular morbidities that may lead to increased mortality [88]. However, in paediatric OSA, there is a paucity of data on this issue [89]. The available data indicate that children with severe OSA are also at a higher risk for cardiovascular sequelae, including right ventricular hypertrophy, increased sympathetic nervous system activation and systemic hypertension [52].

Although the available data are limited, the current evidence suggests that TA in children with OSA is associated with improvements in their systemic and pulmonary hypertension, heart rate, pulse rate variability and cardiac structure and function [89–91]

Impact on Growth

A unique complication of OSA in children, which is not observed in adults, is its impact on growth. The impact of OSA on growth has been documented since 1976 [92]. It has been shown that children with OSA often gain weight poorly and have a catch-up growth period after TA [93].

Cognitive, Learning and Behavioural Functions

OSA has been shown to affect cognitive function in adults [94]. However, the impact of OSA on the cognitive functions of children is more serious, as it affects their learning ability. Even mild OSA and primary snoring have been associated with significant attention impairments and lower memory and intelligence scores [58,95].

A review of 25 published studies evaluating the impact of TA for OSA in children revealed that treatments for OSA in children were associated with improvements in one or more measures, including quality of life, behaviour, or cognitive function [96]. The assessment of neuropsychological development in school-age children with OSA after tonsillectomy was published recently [97].

SUMMARY

Although OSA is a common sleep breathing disorder among children, early recognition and treatment remain a major problem. Delays in the recognition of OSA in children are largely related to the fact that practitioners think that OSA in children mimics OSA in adults. However, OSA may have a completely different presentation in children. Early recognition and treatment are essential to avoid the deleterious consequences of OSA, as current evidence shows that treatments for OSA in children result in a good outcome with a 'cure rate' of 80% after TA. The assessment of OSA in children is complicated by several factors that are unique to children, such as the physiological and maturational changes of breathing in this age group, which requires a good knowledge of OSA in children to interpret their symptoms and the PSG data.

FUTURE DIRECTIONS

Further research should consider that:

- The natural history of OSA in children is not well elucidated. Therefore, long-term studies are needed.

- The long-term impact of OSA on cardiovascular and neurocognitive function is well documented. Longitudinal studies are needed to evaluate the cardiovascular and neurocognitive sequelae of untreated OSA in children.
- Randomized control studies are needed to evaluate the safety and efficacy of auto-titrating PAP devices in children.

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