Review Article

Diagnosis and Evaluation of Obstructive Sleep Apnoea in Children

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Abstract

Introduction: The aim of this article is to review the medical literature and describe clinical and laboratory findings in children with obstructive sleep apnoea (OSA) to differentiate children with OSA from those with primary snoring or other disorders, with a particular focus on Asian children. Methods: Medline search via Pub Med, search terms sleep apnoea and children; and sleep apnoea and children and Asian. Results and Conclusions: Children with OSA usually present with snoring, daytime sleepiness, and/or difficulties in school or behaviour. The prevalence of OSA in Asian children is less than that of other groups, but the severity of the disorder on presentation may be greater. Overnight polysomnography remains the diagnostic "gold standard"; limited studies, or studies in the home, are not sufficient to exclude OSA in a child with suggestive symptoms, nor can they reliably assess the severity of the disorder which is important in planning treatment. Limited studies may, however, be useful in large-scale research studies.

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Children with obstructive sleep apnoea (OSA) may present with nocturnal and/or diurnal symptoms. The history is best obtained from parents, or siblings who share a bedroom, since the child is often unaware of what happens when he or she is asleep.

Nocturnal Symptoms

Snoring is the most common presenting complaint of children with OSA. At night, the children usually snore loudly, with snorting, gasping, or choking. Twelve per cent of all children snore "on most nights",[1] and, although prevalence estimates range from 0.1% to 13%, most studies suggest that 1% to 4% of school-age children have frank OSA.[2] It occurs equally in both sexes,[3] although a male predominance may be seen as the children approach puberty.[2] A study of 62 children who presented to a sleep clinic in Hong Kong showed that "snoring every night" was the single most significant risk factor for OSA.[4]

The parents may notice frank apnoeas. They may describe retractions and increased respiratory effort with paradoxical inward movement of the chest and abdomen. They may describe restless sleep, but sometimes the only manifestation of restlessness is finding the bedclothes all askew in the morning. Children with OSA are at higher risk for enuresis,[5] which may resolve when the OSA is adequately treated.

Daytime Symptoms

Children with OSA may describe daytime sleepiness. They may fall asleep in school, or while riding in the car. However, they may simply display behaviour problems or school difficulties. They may have difficulty paying attention in class, and may be mislabelled with the attention deficit disorder. They may have "micro sleeps" which are misinterpreted as daydreaming or absence seizures. All of these may result in poor academic performance. Apnoeas and nocturnal CO_2 retention may result in morning headaches. The children are frequently noted to be mouth breathers (Table 1).

Physical Examination

A normal physical examination does not exclude OSA in children. The children may fail to thrive, perhaps due to increased work of breathing,[6] or may be obese.[7] The tonsils may be large and the child may have adenoid facies and hyponasal speech. They may have a small jaw, large tongue, and/or a high arched palate. Several genetic

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Table 1. Symptoms of OSA in Children

Nocturnal	Diurnal
Snoring	Daytime sleepiness
Observed apnoeas	Behavioural/school problems
Restless sleep	Morning headaches
Diaphoresis	Mouth breathing
Enuresis	

OSA: obstructive sleep apnoea

syndromes may affect breathing during sleep, particularly those that cause micrognathia (Robin sequence, Treacher Collins syndrome), midface hypoplasia (achondroplasia, Crouzon, Apert, and Pfeiffer syndromes), disorders of respiratory control (Arnold-Chiari malformation, Prader-Willi syndrome), as well as miscellaneous and multifactorial disorders (Mucopolysaccharidoses, Down syndrome, sickle cell anaemia).[8] The cardiac examination is usually normal, but a loud second heart sound may suggest pulmonary hypertension. Any neurologic defect that may affect upper airway motor tone may also predispose the child to OSA.[9]

Diagnostic Methods

There have been many attempts to diagnose OSA in children by clinical criteria. A recent review of the biomedical literature concluded that "clinical history and physical examination are not reliable for diagnosing OSAHS".[10] Less than half of the children referred to sleep centres because of a clinical suspicion of OSA meet polysomnographic criteria for the disorder.[11-14] Questionnaires alone have been unable to differentiate OSA from primary snoring.[15] Li et al[16] developed a questionnaire instrument and tested it on 229 children in Hong Kong. The questionnaire had a positive predictive value of 81%, but a negative predictive value of only 57%. The Sleep Related Breathing Disorder Scale of the validated Pediatric Sleep Questionnaire was shown to predict polysomnography (PSG) results "to an extent useful for research but not reliable enough for most individual patients".[17] Clinical scores that include factors such as difficulty breathing during sleep, observed apnoeas, and snoring have been suggested to differentiate children with OSA[18] but these have not been helpful in diagnosing polysomnographically proven OSA in children who have been referred to a paediatric sleep centre.[13] A retrospective review of 50 children from a Hong Kong sleep clinic combined a lateral neck roentgenogram with history and physical findings and showed good sensitivity (>90%) in detecting OSA, but the specificity was only about 50%.[19] A prospective study in the USA showed that neither the adenoidal/nasopharyngeal (AN) ratio nor the observed size of the tonsils predicted the number of respiratory events during sleep. There was a significant relationship between the AN ratio and the duration of obstructive apnoeas, as well as the extent of oxyhaemoglobin desaturation, suggesting that adenoid size correlated with the severity, but not the number of respiratory events.[7] Analysis of a 15 minute audiotape improved the sensitivity of a clinical score from 0.46 to 0.71, but the authors concluded that audiotapes are not sufficiently specific to reliably distinguish primary snoring from OSA.[20] Adding a "sleep tape" to reinforce clinical criteria resulted in a sensitivity of 0.92, but the specificity was only 0.29 with a positive predictive value of 0.5.[21] A home video recording of the child's "worst breathing" was a bit better, with a sensitivity of 0.94, specificity of 0.68, and positive predictive value of 0.83.[22]

Home Studies

Because of the expense and inconvenience of laboratory-based PSG, there have been several attempts to utilise simpler, more limited studies in the diagnosis of OSA in children. Studies in the home have the advantage of a more natural sleeping environment, but fewer channels make for less precision of measurement. In addition, there is no technologist available to solve technical problems, so some percentage of home studies will need to be repeated. Pulse oximetry would be a simple way to estimate the extent of sleep-disordered breathing, but it can only detect events that result in oxyhaemoglobin desaturation, and misses events that result in arousal before a desaturation occurs. It is also subject to motion and other artifact. In one report almost half of 349 oximetry studies were "inconclusive"; counting inconclusive oximetry as a negative test, the sensitivity of oximetry was only 0.43.[23]

The pulse transit time (PTT) is a novel method to assess respiratory effort and arousal that has been used in the diagnosis of sleep-related breathing disorders in adults. The PTT is the interval between the R-wave of the electrocardiogram and the arrival of the photoplethysmographic pulse at the finger. The travel time of the pulse wave is inversely proportional to arterial wall stiffness, which is determined by blood pressure. Arousal at the termination of an obstructive event leads to a transient increase in blood pressure, with a resulting decrease in the PTT.[24] Brietzke et al[24] found a high correlation (R-squared = 0.73) between PTT arousal index and AHI in 59 children. The technique was most useful in children with moderate to severe OSA, but was "barely adequate" in mild OSA. Foo et al[25] suggested that PTT in combination with heart rate variability and pulse oximetry might be better than any of the parameters alone, but over 10% of events could not be detected because of artifact, and PTT is unable to detect central respiratory events.

A home testing device that included inductance plethysmography, electrocardiogram (ECG), and pulse oximetry to assess respiratory events, with a camcorder and microphone to estimate sleep time demonstrated sensitivity and specificity of 1.0 in discriminating children with an apnoea-hypopnoea index (AHI) >5. Its accuracy was less using other AHI cut-offs, and 13% of studies were unsuccessful even in this selected group of uncomplicated children.[26] Most important, however, was the lack of direct correlation between the AHI on the home study and that determined by laboratory PSG since it is important to know the severity of OSA in deciding treatment options in children. In a study of 69 children, preoperative AHI was the major predictor of a response to surgery. A baseline AHI >19.2 suggested that adenotonsillectomy alone was less likely to result in a "cure" of OSA.[12] In addition, severe OSA on PSG predicts a greater risk for postoperative complications.[27,28] Thus, full PSG is necessary to i) differentiate OSA from primary snoring; ii) define the severity of OSA so proper treatment and monitoring can be planned; and iii) evaluate a differential diagnosis for other sleep disorders, including narcolepsy and nocturnal seizures. However, there may be a role for home studies in the research setting.[29]

Polysomnography

PSG remains the "gold standard" for diagnosing OSA in children. PSG includes monitoring of frontal, central, and occipital electroencephalograph (EEG) and submental electromyograph (EMG) activity to determine sleep architecture. Pneumotachography is the most accurate way to measure airflow, but the facemask required can be bulky and disturbing to the child. Surrogate measurements such as thermisters are inexpensive, easy to use and sensitive, but the signal is non-linear and only qualitative. Nasal pressure sensors can be helpful in discerning hypopnoeas since flow limitation may result in a flattening of the waveform,[30,31] but it can be challenging to maintain a good quality signal in children. End-tidal CO_2 measurements can also help to assess gas exchange. This is particularly important in children with obesity, neuromuscular weakness, or other factors that may place them at higher risk for hypoventilation. Inductance plethysmography provides a semiquantitative estimate of chest wall and abdominal movement. Strain gauges, usually with piezoelectric crystals, are less accurate.[31-35] A single modified ECG lead II is used to assess cardiac rhythm and rate. Serious dysrhythmias are less common in children than adults, but sinus bradycardia is often seen in association with respiratory events. Tibial EMG is used to assess leg movements and is essential to quantitate periodic limb movements during sleep.[36] The child is under the constant video observation of a trained technologist who is available to replace sensors if necessary, insures a quality recording, and assists the patient should he or she develop difficulties that require intervention.

There are unique challenges to performing and interpreting PSG in children compared to a cooperative adult. The décor of the laboratory should be friendly and comfortable to the child, while not too juvenile to discourage adolescents. Technologists need to be comfortable in dealing with children and their families. It is helpful to have an extra bed or cot in the room so a parent can sleep with the child. It is usually better if the parent does not share the same bed, since movements and other activity by the parent can either disturb or be misinterpreted as originating from the child.[37]

The study should be performed overnight. Individual nap study parameters are not very sensitive in predicting overnight polysomnographic findings,[38] and nap studies significantly underestimate the severity of sleep-disordered breathing. Significantly more children had obstructive apnoea and desaturation (SaO_2 less than 90%) during overnight PSG than a 1-hour nap study performed in the laboratory. The peak $PETCO_2$ and the SaO_2 nadir were significantly worse during overnight PSG.[39]

Children tend to have shorter and fewer respiratory events than adults, and a high proportion of hypopnoeas, so the studies must be scored and reviewed with great care. In adults, obstructive apnoeas 10 s or longer are scored, but in children, with a faster respiratory rate, a respiratory event of 2 missed breaths is scored.

Are Asian Children Different than other Groups?

OSA is a very complex disorder determined by several phenotypes such as obesity, craniofacial structure, and differences in neuromuscular and ventilatory control. Genetics may partially explain some of the ethnic clustering of these phenotypes, modulated by cultural and environmental factors. Asian adults are at risk for a more severe degree of illness for lesser degrees of obesity, compared to Caucasians.[40] Ong[41] studied adults with OSA at the Stanford Sleep Clinic in Palo Alto, California. They compared 105 Asian patients with 99 Caucasians matched for age, gender and body mass index (BMI). There was a larger proportion of Asians with severe OSA, defined either by Respiratory Disturbance Index, minimum SaO_2, or oesophageal pressure. Race was associated with severe sleep disordered breathing independent of age, sex and BMI, with an odds ratio of 2.51 compared to Caucasians. There was no difference in the severity of questionnaire-based symptoms or Epworth scores between the Asian and Caucasian patients.

Asian children may also have more severe OSA on presentation than other groups. Eighty-five per cent of Thai

children with clinically suspected OSA met polysomnographic criteria for the disorder, and 36% were classified as severe.[42] A preliminary report suggested that almost half of Thai children with OSA had pulmonary hypertension demonstrated on echocardiography or electrocardiography.[43]

While the severity of OSA may be grater in Asian children, the prevalence may be lower than in western countries. Anuntaseree et al[44] studied 1142 children aged 6 to 13 years from 7 randomly selected schools in southern Thailand. 8.5% of the children snored "on most nights" and the prevalence of OSA by PSG criteria was only 0.69%. The prevalence was the same in boys and girls. Preutthipan[45] speculated that the increased severity of OSA in Thai children maybe due to delayed diagnosis. He attributed this to less awareness of the disorder on the part of patients and parents, and a shortage of paediatric sleep laboratories in Thailand, which may or may not be true in other nations.

Risk factors for OSA in Asian children are similar to other groups. Snoring and OSA are associated with enlarged tonsils,[44,46,47] atopy and allergic rhinitis,[44,48] and obesity.[46,47] Obesity and OSA may predispose children to insulin resistance.[49]

Asian children with OSA generally respond well to adenotonsillectomy.[50] Mandibular osteotomy with distraction was successful in treating 10 Japanese children with OSA caused by micrognathia.[51]

Summary

OSA is common in children, and the resulting sleep fragmentation may result in impaired daytime performance. The children may be misdiagnosed with absence seizures or attention deficit disorder. Snoring is the most common presenting symptom, but the diagnosis should be confirmed with overnight PSG in a laboratory that has expertise in dealing with children. The PSG can define the severity of the disorder so proper treatment and monitoring can be instituted. The prevalence of OSA may be lower in Asian children than other groups, but they may be more severe when diagnosed. Parents and physicians should be aware of OSA in children, and early detection may minimise sequelae such as pulmonary hypertension, insulin resistance and learning problems.

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