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REVIEW ARTICLE

PEDIATRIC AND ADOLESCENT SLEEP DISORDERS

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Sleep disorders are frequently the sources of parental concerns. In addition, disrupted or insufficient sleep can have negative impact on the mental and physical health of children. Adequate sleep improves child's control of behavior, attention and emotions.[1] Circumferential evidence suggests a basic neurodevelopmental process linking sleep and the regulation of attention, arousal, affect, and social behavior.[2]

I. Sleep Architecture Pattern in Childhood:

At about 28 weeks of gestation, trace discontinue EEG pattern emerges. It is characterized by rhythmic waves lasting for 1-2 seconds at 4-6 Hz frequency. Delta brushes appear and can continue until shortly before term. They are fast waves at 1-20 Hz that are superimposed on slow waves at 0.5- 1 Hz. By 32-34 weeks of gestation, sleep state proportions are composed of 50% active sleep (rapid eye movement-REM) and 50% quiet sleep (non-REM). Newborns usually enter sleep through REM. REM gradually decreases by age 3-4 years to near adult value (about 25%). Trace alternant pattern characterizes quiet sleep and bursts of high-amplitude slow waves lasting for 4-5 seconds alternating with periods of low-amplitude activity at the same duration. This pattern disappears by 4 weeks of term. By 2-3 months post-term, sleep spindles are generally seen. K complexes and slow-wave activities are seen by 4-6 months postterm.[3] The onset of these EEG patterns allows scoring N1, N2, N3, and REM as in older children and adults.[4]

Total sleep time (TST) gradually decreases and sleep cycles gradually lengthen throughout childhood. The normal newborn sleeps 16 to 20 h/d. Sleep generally occurs in 1-4 hour periods, followed by 1-2 hour wake periods. Throughout the first 12 months of life, TST decreases to approximately 14 hours per day. TST continues to decrease to around 12 hours per day during the toddler years (age 1 to 3 years). In preschool years (age 3 to 6 years), TST decreases slightly to around 11 to 12 hours per day. Most children stop taking naps by age 5 years. In the middle childhood years (age 6 to 12 years), children sleep 9-10 hours per day. Daytime sleepiness should be considered rare. The sleep requirement for adolescents is 8 to 9 hours. During teen age years, sleep schedules become more erratic with later bedtimes and earlier wake times for adolescents (see Table 1).[5]

Table 1. Sleep Requirements for Children and Adolescence.[6]

AGE	Nighttime Sleep (hr)	Daytime Sleep (hr)	Total Sleep (hr)
1 wk	8.25	8.25	16.50
1 mo	8.25	7.25	15.50
3 mo	9.50	5.50	15.00
9 mo	10.50	3.50	14.00
1-3 yr	10.75	3.00-1.75	13.75-12.00
4 yr	11.50	0	11.50
10 yr	9.75	0	9.75
15 yr	8.75	0	8.75
18 yr	8.25	0	8.25

II. Medical History and Physical Examination:

In order to properly assess and treat children with sleep problems, a comprehensive evaluation of sleep and waking behavior must be done. Initial medical history should include the following:

1. Detailed sleep history (pauses in breathing, snoring, overall sleep duration and quality, timing of bedtime/waketime, duration of sleep, night awakenings, arousal parasomnias, enuresis, head banging, head rocking, bruxism, and headaches).
2. Sleep diaries and sleep questionnaires.
3. General medical/surgical conditions (sinus problems, asthma, chronic cough, GERD and surgeries involving upper airway).
4. Comprehensive social history.
5. Psychologic/developmental screening (behavior issues, such as hyperactivity or inattentiveness).
6. Mother's pregnancy and perinatal history.
7. Family history of additional family members with suspected or confirmed sleep disordered breathing (SDB), apparent life threatening events, or SIDS, as well as congenital syndromes.

Physical examination should include:

1. Careful inspection of the upper airway.
2. The oropharynx should be inspected (tongue size, uvula size, and tonsil size).
3. Evaluation for adenoid facies, micrognathia, midface hypoplasia, and a high arched palate.
4. The cardiac examination is usually normal, but a loud second heart sound may suggest pulmonary hypertension. Children with obstructive sleep apnea (OSA) may have higher diastolic BP.
5. Assessment of body fat distribution, nasal speech, Down syndrome, and evaluation for the presence of a pectus excavatum.[7,8]

Once assessed, age-dependent response to diagnosis and treatment should be taken into consideration. It is crucial to recognize that sleep is an active process involving physiologically diverse stages.

III. First three years of life:

The most frequent sleep related problems are difficulty going to sleep or staying asleep during the night.[2]

1. ***Sleep-onset association disorder:*** Nighttime arousals are very common in all ages. The complaint usually comes from parents not the child. Parents may incorrectly get involved in the child's sleep transition process. Later on, the child can't make the transition back to sleep alone and rely on the parents to complete the sleep transition. Careful history will establish the diagnosis and the disorder rapidly corrects to simple gradual behavioral interventions.[9]
2. ***Difficulty sleeping alone:*** All children wake up 5-10 times per night around the end of each sleep cycle. Some children can go back to sleep without parental awareness. It is important to condition the child to sleep throughout the night and address parental anxiety effectively.[2]
3. ***Limit setting:*** Parents are unable to enforce nighttime rules with enough consistency to keep the child in bed. This is easily picked up from history. Parents have to learn to be firm and enforce regular bedtime ritual. The child should be kept in the bedroom; if necessary, positive behavior modification for staying in bed may produce positive response (sticker, star, or prizes).[6,9]
4. ***Excessive nighttime feedings:*** Infants who are fed large quantities (8-32 oz) at night have shown to have frequent and continued wakings. This also can be picked up from history especially if the child only goes back to sleep with feeding. Sleep consolidation usually occurs as feedings are decreased and associated habits are eliminated.[1]

IV. Children ages three to eight years of life:

1. ***Confusional arousals:*** are thought to occur in almost all young children in one form or the other especially before the age of 5. It usually begins with movement and moaning, progressing to crying and maybe calling out. A look of confusion, agitation, or distress with eyes open or closed is described. Cuddling doesn't provide comfort and may aggravate the child's agitation. Attempts to wake the child are usually unsuccessful.[10,11]
2. ***Sleep terrors:*** is a variant of confusional arousals.[47] It begins with the child bolting upright with a loud scream. The child is seen awake, aroused, extremely agitated with dilated pupils and racing heart rate. They look afraid and might run out of bed blindly as if they are running from an unseen danger. These events are typically shorter than confusional arousals.[1,11]
3. ***Somnambulism (sleep walking):*** Up to 40% of children sleep walk at least once. About 5% have repeated episodes. The peak prevalence is about 10 years of age and usually disappears by age.[15.10]
4. ***Somniloquy (Sleep talking):*** is verbal output during sleep. One of the most famous literature examples of sleep talking is the episode of Lady Macbeth by Shakespeare. Talking could be brief, incoherent or long and associated with anger.[13]

5. ***Nightmare:*** represents frightening dream that can result in prolonged period of wakefulness. There is sudden arousal to full wakefulness, orientation to environment, clear sensorium and recall of dream. The child is usually easily comforted.[11]
6. ***Sleep paralysis:*** is characterized by inability to voluntary move either in the beginning of sleep (hypnagogic) or on awakening from sleep (hypnopompic). The episodes are usually brief and can be seen with narcolepsy or normal individuals.[11]
7. ***Bruxism:*** is common in children and occurs in stage N1 and N2 sleep. It is easily treated with mouth guard to minimize teeth damage.
8. ***Sleep related breathing disorder:*** Several studies have estimated the prevalence of primary habitual snoring among all children (age 1 to 18 years) to be between 8% and 12%.[12] OSA represents the extreme form of the disorder including sleep disruption along with gas-exchange abnormalities. OSA has a prevalence of 1 to 3% and as high as 5% in older, more obese children. The prevalence of central sleep apnea in children is less known. Clinical significance for periodic breathing (central apnea in newborns), although common, is unknown and normative data is limited. It occurs equally in both sexes,[14] although a male predominance may be seen as the children approach puberty.[14] A study[15] 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. Several factors are influential in the development of SDB in children. There is a higher prevalence among African-American and Asian children than among white children. Obesity increases the risk of SDB, although many children with SDB are not obese. Many children with SDB have a parents or siblings with SDB. Disorders associated with upper airway obstruction (anatomic abnormality) or neuromuscular weakness predisposes children to SDB. There is no significant difference in the prevalence of pediatric SDB based on gender.

Pathophysiology

OSA syndrome (OSAs) involves obstruction at one or more sites along the upper airway. OSA is usually some combination of decreased upper airway patency (upper airway obstruction), reduced capacity to maintain airway patency (decreased upper airway diameter and muscle tone), and decreased drive to breathe in the face of reduced upper airway patency (reduced central ventilatory drive).[16] In normal circumstances, the genioglossus and geniohyoid muscles work together to maintain pharyngeal airway patency during normal respiration while the individual is awake and asleep. The tone of these muscles increases during respiration to counteract the subatmospheric pressures generated by the muscles of respiration. Inability of the dilator muscles to generate the counteracting pressure can result in pharyngeal airway collapse.

The most common obstructive component of OSAs in children is adenotonsillar hypertrophy. There is no direct correlation between the severity of adenotonsillar hypertrophy and OSAs[17] and not all OSAs resolve post adenotonsillectomy. In addition, children with OSAs have a more collapsible pharynx than do normal children, and they lack upper airway responses to hypercapnia and negative pressure during sleep.[18]

Nasal, oropharynx, and other craniofacial may cause upper airway obstruction.[16] Other obstruction may be due to upper airway disease (allergies associated with chronic rhinitis, nasal obstruction, gastroesophageal reflux when accompanied by pharyngeal edema, velopharyngeal flap cleft palate repair), and lower airway disease (asthma). Upper airway size and muscle tone (a small or "floppy" airway) also play an important role in the underlying pathophysiology of OSA).

Clinical Manifestations

Nocturnal Symptoms: The most common presenting symptom is snoring; however, children who are weak, particularly infants, may not snore. Snoring is usually loud and associated with breathing pauses, gaps in breathing, snorting, gasping, or choking. Children with obstructive hypoventilation often have continuous snoring without pauses or arousals. Sweating during sleep, restless sleep, nocturnal enuresis, and sleeping with a hyperextended neck are common with SDB, but can occur in the absence of SDB. Witnessed apnea requiring parental intervention (such as shaking to initiate breathing) is probably the most specific symptom. Sweating during sleep is a very nonspecific marker of SDB.

Daytime Symptoms: Daytime symptoms are frequently encountered in children. Numerous studies[19,20] have identified a correlation between sleepiness, behavioral and cognitive dysfunction, and SDB symptoms. The typical presentation daytime symptoms manifested by sleepiness are hyperactivity, inattentiveness, or other cognitive/behavioral abnormalities. Children may fall asleep in school, display behavior problems, or have difficulty paying attention in class and may be mislabeled with attention-deficit hyperactivity disorder. "Microsleeps" are common and often misinterpreted as daydreaming or absence seizures. These daytime symptoms may result in poor academic performance. Standard intelligence quotient test results are reported to be lower in children with OSAS. These findings may be due to intermittent hypoxia, sleep fragmentation, or both.[21] Other daytime symptoms may include falling

asleep while riding in the car, difficulty swallowing, mouth breathing, morning headaches, and poor appetite.

Risk Factors

A number of risk factors for pediatric SDB have been identified: adenotonsillar hypertrophy, anatomic obstruction, upper airway size, and muscle tone. Adenotonsillar hypertrophy results from multiple etiologies, the most common of which are recurrent upper respiratory infection, allergic irritants, chronic nasal obstruction from allergies, and pharyngeal edema from gastroesophageal reflux disease (GERD). Adenotonsillar hypertrophy is the most prominent in children up to age 6 to 8 years, when natural regression of this tissue begins to occur. Decreased airway size that occurs in obesity also occurs in many syndromes Table 2. Such as Prader-Willi syndrome, achondroplasia, mucopolysaccharidoses, Pierre Robin syndrome, Treacher Collins syndrome, Apert syndrome, and many others. Decreased upper airway tone results from brainstem lesions such as tumors and malformations and diseases affecting overall neuromuscular tone. Down syndrome is perhaps the most prevalent of the "pediatric" conditions leading to risk for SDB. Children with Down syndrome may have any or all of the predisposing factors for SDB, including macroglossia, midface hypoplasia, micrognathia, and muscular hypotonia. Other genetic syndromes are relatively uncommon but may be encountered by the sleep specialist.[22]

Table 2. Genetic Syndromes Associated with OSA[23,24]

- Micrognanthia
  - Robin sequence
  - Treacher-Collins Syndrome
- Midface hypoplasia
  - Achondroplasia
  - Crouzon Syndrome
  - Apert Syndrome
  - Pfeiffer Syndrome
- Disorders of Respiratory Control
  - Arnold-Chiari malformation
  - Prader-Willi Syndrome
- Multifactorial Disorders
  - Mucopolysaccharidoses
  - Down Syndrome
  - Sickle Cell Anemia

Complications

The most severe complications Table 3. Of pediatric sleep problems include death, growth failure, and cardiac, GI, or pulmonary system disorders. More common complications are the cognitive and behavioral disorders.

The incidence of death as a result of SDB is an associated in children with a family history of sudden infant death syndrome (SIDS).[22] Although some characteristics of SIDS may lead to death in these infants, it is unlikely SIDS represents a pure form of OSA. Death caused by SDB is limited to the extremes of OSA and is likely associated with other medical or surgical conditions.

Cardiovascular complications associated with extremes of OSA in association with other disorders include chronic respiratory acidosis, cor pulmonale, hypertension, polycythemia, and pulmonary hypertension. OSA has been established as a clear cause of secondary hypertension in adults; however, there is limited evidence pediatric SDB and hypertension. Amin et al25 studied 60 children and demonstrated that children with OSA had significantly higher night-to-day systolic BP, greater mean BP variability during wakefulness and sleep, and smaller nocturnal dipping of mean BP when compared to children with primary snoring. Leung et al26 found that children with elevated apnea-hyponea index (AHI) had higher awake and asleep BP, and obese children with a high AHI had a higher prevalence of hypertension than obese children with a low AHI. A smaller study27 of 23 patients also demonstrated that BP positively correlated with the degree of SDB.

GI complications associated to OSA include GERD and feeding difficulties. GERD supports movement of gastric contents into the esophagus due to increased intraabdominal pressures and more negative intrathoracic pressures during apneas and hypopneas. Although clearly associated with OSA in adults, GERD in children needs further study.[24] Feeding difficulties can be broadly credited to poor sleep.

Pulmonary complications often includes chronic aspiration (follows gastroesophageal reflux), pulmonary edema (follows severe upper airway obstructive events), and development of a pectus excavatum (follows the effects of nightly severe upper airway resistance and the pressure required to overcome this resistance).

Behavioral and cognitive consequences of OSA include attention deficit, hyperactivity. Beebe[19] recently reviewed 61 studies of neurobehavioral morbidity in children with SDB. The association between SDB, behavior regulation and attention is strongly evident with limited evidence for sleepiness associated with mood and emotional control. There is no evidence of effect on visual perception or construction, little evidence of effect on expressive lan-

guage, and increasing evidence of effect on intelligence testing (especially in preschool years).

Nocturnal enuresis has been closely associated with SDB. Treatment of even mild forms of SDB has demonstrated improvement.

Table 3. Complications of OSA in Children:

- Death
  - SIDS
  - Growth failure
  - Obesity
- Cardiovascular
  - Cor pulmonale
  - Pulmonary HTN
  - Polycythemia
  - Chronic repiratory acidosis
  - HTN
- GI complaints
  - Feeding difficulties
  - GERD
- Pulmonary Complications
  - Chronic aspiration
  - Pulmonary Edema
  - Development of pectus excavatum
- Behavioral and Cognitive complications
  - Poor behavior regulation
  - Poor attention
  - Nocturnal enuresis

Diagnostic Criteria and Diagnostic Methods:

Clinical scores that include factors such as difficulty breathing during sleep, observed apneas, and snoring have been suggested to differentiate children with OSA,[28] but are not helpful when compared to polysomnographically proven OSA in children.[28]

There was a significant relationship between the adenoidal/nasopharyngeal ratio and the duration of obstructive apneas, as well as the extent of oxyhemoglobin desaturation, suggesting that adenoid size correlated with the severity, but not the number, of respiratory events. A prospective study[17] in the United States showed that neither the adenoidal/nasopharyngeal ratio nor the observed size of the tonsils predicted the number of respiratory events during sleep. A retrospective review[29] of 50 children from a Hong Kong sleep clinic combined a lateral neck radiograph with history and physical findings and showed good sensitivity (>90%) in detecting OSA, but poor specificity (50%).

Home Studies

Studies in the home have the advantage of a more natural sleeping environment, but fewer channels yield less precision of measurement. Also without a technologist available to solve technical problems, it would be necessary to repeat some percentage of home studies. Pulse oximetry detects events that result in oxyhemoglobin desaturation but misses events that result in arousal before a desaturation occurs. However, pulse oximetry is subject to motion and other artifact.[30] Pulse transit time (PTT) is the interval between the R wave of the ECG and the arrival of the photoplethysmographic pulse at the finger. The travel time of the pulse wave is inversely proportional to arterial wall stiffness, determined by BP. Arousal at the termination of an obstructive event causes a transient increase in BP with a resulting decrease in the PTT.[31] This technique is most useful in children with moderate-to-severe OSA but was "barely adequate" in mild OSA. PTT is unable to detect central respiratory events.[31]

Polysomnography

Polysomnography remains the "gold standard" for diagnosing OSA in children. Full polysomnography is necessary to differentiate OSA from primary snoring and define the severity of OSA, so proper treatment and monitoring can be planned, and evaluate a differential diagnosis for other sleep disorders, including narcolepsy and nocturnal seizures. Polysomnography includes monitoring of frontal, central, and occipital EEG and submental electromyographic activity to determine sleep architecture.

Performing and interpreting polysomnography in children as compared with a cooperative adult is challenging. To make the study more children friendly, décor of the laboratory should be child focused but not too juvenile to discourage adolescents. Provide an extra bed, cot, or lounge chair in the room so a parent can sleep with the child but not cause movements or other activity that can either disturb the child or be misinterpreted as originating with the child.[33] Ensure that technologists are comfortable in dealing with children and their families. The study should be performed overnight. Individual nap study parameters significantly underestimate the severity of sleep-disordered breathing and are not very sensitive in predicting overnight polysomnography findings.[30] One hour nap studies performed in the laboratory showed significantly less children had obstructive apnea and arterial oxygen saturation (SAO2 < 90%) than during overnight. In addition, peak partial pressure of end-tidal

CO_2 and the SAO_2 nadir were significantly worse during overnight polysomnography.[25] Objective confirmation of SDB requires some form of testing. Debate continues as to the efficacy of home monitoring. Portable, multichannel unattended monitoring has been used successfully in research studies.[35] However, other studies[36] comparing unattended portable monitoring and standard polysomnography have demonstrated that home monitoring in pediatric SDB is presently not a reliable substitute for standard polysomnography. Furthermore, portable monitoring based only on oximetry does not appear to be adequate for identification of OSA in otherwise healthy children.[37] Polysomnography normative values have not been definitively established for children. Two recent studies[38,39] support the following normal values in children: central apneas lasting < 20 seconds without a SPO2 drop to < 89%, or a SPO_2 drop > 4% from baseline; a central apnea index : 1.0; an obstructive apnea index : 1.0; a SPO2 nadir > 92%; and PCO_2 at end-expiration > 50 mm Hg for < 10% of total sleep time. Although there are no standard criteria for defining the severity of SDB, most clinicians grade the disease based on ***clinical judgment:*** (1) Severe disease is a respiratory disturbance index (RDI) of 10, or any RDI with a desaturation nadir < 92%, (2) moderate disease is an RDI of 5 to 10 without desaturation, and (3) mild disease is an RDI of 1 to 5. Treatment should be considered even for mild SDB when these features are present.

Treatment

Management can encompass surgery, pharmacotherapy and positive airway pressure. Surgery (adenotonsillectomy) is indicated in those cases if adenotonsillar hypertrophy is present.[31] Because adenotonsillar hypertrophy is the most common condition associated with pediatric OSA, adenotonsillectomy provides definitive therapy in the majority of patients. 70% to 90% of patients with uncomplicated cases achieve complete reversal of clinical symptoms.[40] Children at high risk for postoperative complications include those < 3 years old; children with severe OSAs; children with comorbid medical conditions such as morbid obesity, hypotonia, and craniofacial syndromes; and children in whom significant clinical sequelae (failure to thrive, cor pulmonale) have already developed.[40]

Nasal decongestants or nasal steroids[29,31] can be helpful in children whose symptoms seem to be due to allergic rhinitis. Leukotriene-modifying agent, montelukast, seems to be effective in mild SDB.[30] Supplemental oxygen is not recommended because oxygen therapy can worsen hypoventilation and CO_2 retention. The use of external nasal dilators in children has not been studied. Positive airway pressure (continuous positive airway pressure/bilevel) is useful in children in whom surgery is contraindicated or has been ineffective, or as a bridge while definitive craniofacial surgery or weight loss is awaited.[42,43] All children who undergo adenoidectomy and/or tonsillectomy should undergo at least a repeat clinical evaluation to ensure resolution of symptoms at least 6 to 8 weeks after the procedure to allow for adequate healing time. Nasal CPAP, the standard of care in adults with OSA, is effective in children. Because CPAP requires both a motivated patient and family, it is usually reserved for children in whom adenotonsillectomy has failed or is not possible. Repeat polysomnography to ensure adequate treatment should be considered every 6 months in children < 3 years old, annually in children age 3 to 8 years, and every 18 to 24 months in older children. Regular follow-up is needed to ensure compliance. General recommendations include follow-up every 6 months until attainment of Tanner stage 5 puberty Table 4.

The routine use of autotitrating CPAP would seem an ideal therapeutic option for children, particularly those with rapid growth or increasing weight; however, CPAP without in-laboratory assessment of tolerance is probably not appropriate for the pediatric population. In obese children, weight management should include comprehensive nutritional, exercise, and behavioral counseling for both the patient and the family. Oral appliances may be utilized in adolescents when growth is completed.

Table 4. American Academy of Pediatrics Clinical Practice Guidelines for OSAs.

1. All children should be screened for snoring.
2. High-risk patients should be referred to a specialist.
3. Children with cardiorespiratory failure should be screened immediately.
4. Diagnostic testing should be done to detect primary snoring verses OSA syndrome, the "gold standard" being polysomnography.
5. Adenotonsillectomy is the first line of treatment for most children, with continuous positive airway pressure (CPAP) as an option for those poor surgical candidates or those children who do not respond to surgery.
6. High-risk patients should be monitored as inpatients postoperatively.
7. Children who have adenotonsillectomy should be reevaluated postoperatively with polysomnography to determine whether additional treatment is required.

V. Adolescence:

1. ***Circadian rhythm disorders:*** They include advanced sleep phase and delayed sleep phase. Delayed sleep phase is common in adolescents. They prefer late bedtimes and have difficulty waking up in the

morning. This usually is exacerbated by early school start time. The treatment involves negotiating a reasonable bedtime and wake time during school days and weekend. Bright light in the morning and Melatonin 1hour before desired bedtime are helpful. Children with advanced sleep phase prefer to go to bed early evening and have early morning awakenings. Treatment includes gradual delaying bedtime and may be helped with bright-light therapy.[44-46]

2. ***Narcolepsy:*** The symptoms of narcolepsy generally start before age 15, and sometimes as young as 5 years. Cataplexy is the most specific feature of narcolepsy and is present in 50-70% of children.[47]

In summary, this review article provided current information on sleep, sleep disorders, and the impact of sleep on health and behavior in infants, children, and adolescents to physicians interested in pediatric sleep. It is imperative to be familiar with respiratory disorders and non-respiratory disorders to accurately diagnose and manage children with sleep complaints. The frequent sleep disorders that are affecting children have significant influence on their attention, behavior, school achievement and emotions. Treating the children at younger ages will provide them and their parents with a better quality of life.

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