Sleep Disorders in Children

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ABSTRACT

Purpose of Review: The purpose of this review is to examine how sleep disorders in children are affected by age and comorbid medical influences, and to discuss current understanding of how the clinical manifestations, pathophysiology, and treatment of common childhood sleep disorders differ from those of the adult population.

Recent Findings: Recently established age-specific norms are required for accurate interpretation of polysomnograms and multiple sleep latency tests in children.

Summary: Sleep disorders such as insomnia, obstructive sleep apnea, and excessive daytime somnolence are common in both children and adults, but the clinical manifestations and underlying pathophysiology of these disorders vary substantially with age. For example, the bedtime struggles of a temperamental toddler are associated with different symptoms and causative factors compared to psychophysiologic insomnia affecting a middle-aged person. Similarly, a 6-year-old child with obstructive sleep apnea is more likely to exhibit daytime inattention and hyperactivity as a referable daytime symptom than the clear-cut lethargy or sleepiness that most affected adults experience. This review will examine how insomnia, excessive sleepiness, and obstructive sleep apnea differ in children compared to adults.

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Relationship Disclosure: Dr Hoban has performed medicolegal review of pediatric neurology-related cases.

Unlabeled Use of Products/Investigational Use Disclosure: Dr Hoban discusses the use of treatments for sleep apnea in children, including nasal steroids and maxillary expansion devices, which are unlabeled.

SLEEPLESSNESS IN INFANTS AND YOUNGER CHILDREN

Determining whether *night waking* in an infant or young child represents a clinically significant problem sometimes requires careful comparison of the child's sleep patterns and sleep duration to highly variable and age-dependent normative data (**Figure 10-1**).[1] Most newborns and younger infants initially sleep in brief 2- to 4-hour periods equally distributed across the daytime and nighttime hours and affected by the need for frequent feedings during early infancy. As a result, frequent night waking is considered entirely normal for younger infants.

As circadian regulatory mechanisms grow stronger and become entrained to recurring behavioral and environmental cues, nighttime sleep gradually becomes longer and more continuous and daytime napping diminishes via a process called *settling*. Although most infants settle during the first year of life, about 10% do not achieve consolidated nighttime sleep by the age of 12 months.[2] Brief waking, up to several times nightly, persists as a normal phenomenon for many toddlers without clinical sleep problems, usually at a level not noticed by parents and caregivers.[3] Night waking is considered abnormal when it is disproportionately frequent, prolonged, or disruptive for the person's age. Problematic night waking is estimated to affect 20% of infants and toddlers.[4]

Night waking in infants and younger children is frequently associated with bedtime struggles, which may take the form of crying in infants, tantrums or

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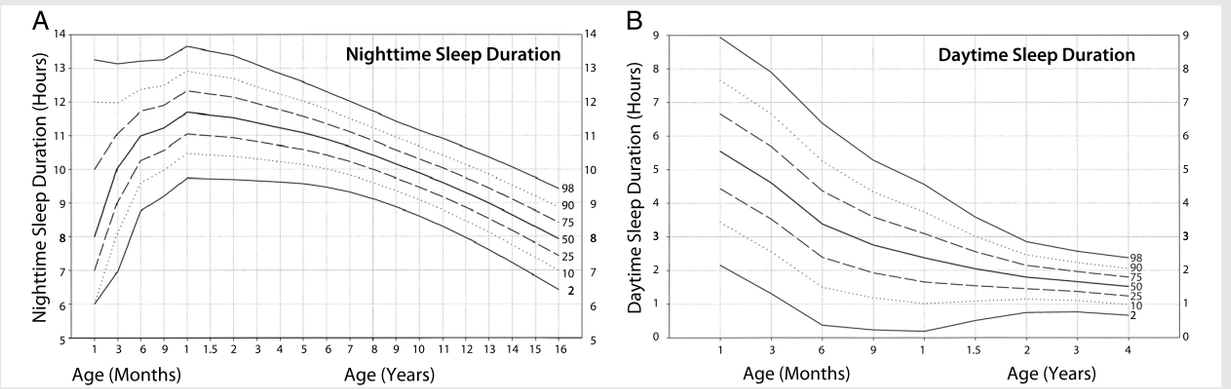

FIGURE 10-1 Percentiles for total nighttime (*A*) and daytime (*B*) sleep duration during childhood.

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KEY POINTS

- The symptoms, pathophysiology, and treatments for some sleep disorders are substantially different for children compared to adults.
- Ten percent of children do not achieve consolidated nighttime sleep by 1 year of age.

"curtain calls" in toddlers, and other forms of bedtime resistance that vary with age (**Case 10-1**). Problematic behaviors at bedtime may reflect a variety of underlying influences, including anxiety, lack of sleepiness, a desire to engage the parent, or deliberate effort to forestall sleep onset. Although bedtime struggles may occur on an occasional basis in healthy

Case 10-1

A 12-month-old infant was referred by his pediatrician for evaluation of long-standing difficulties settling to sleep at bedtime, associated with frequent night waking. The child's mother reported that he had always had difficulty settling to sleep unless she was with him. On most nights, he would fall asleep near 9:00 PM while being held by his mother, who was usually watching television. Although he would initially remain asleep upon being transferred to his crib, on most nights he would awaken 2 hours after sleep onset, crying, fully awake, and difficult to console. He would return to sleep after 30 to 60 minutes only if held by his mother until asleep. Night waking of similar duration and character would recur during the remainder of the night at intervals of about 90 minutes until morning waking at or before 7:00 AM. The mother reported similar difficulties and circumstances settling her son for his 90-minute midday nap. The family's attempts to let the child settle to sleep alone were unsuccessful, as he would cry unabated until picked up and held, typically after 20 to 40 minutes.

No obstructive symptoms or restlessness was reported during nighttime sleep, but daytime irritability was consistently observed when night waking was worse. The child was developmentally normal and otherwise healthy apart from allergic rhinitis. Physical examination results were notable for mild nasal congestion and 2+ tonsillar size (**Figure 2-4**), but otherwise normal.

Comment. This case illustrates a typical example of behavioral insomnia of childhood, a disorder in which a child's difficulty falling asleep or staying asleep is secondary to inappropriate sleep-onset associations—in this case a habitual need to have his mother present while falling asleep—or inconsistent limit setting.[5]

younger children, more substantial bedtime resistance is estimated to affect 10% to 20% of toddlers and preschool-aged children.[6]

In the *sleep-onset association type* of behavioral insomnia, a child habitually settles to sleep via circumstances that cannot be independently sustained, such as being rocked or having a parent present. Affected children become so dependent on these routines for transition to sleep that they are unable to settle to sleep without them. Attempts to do so result in insomnia or distress at bedtime, and inability to return to sleep following the physiologic awakenings normally occurs during the middle and latter portions of the nighttime sleep period.

The *limit-setting type* of behavioral insomnia is characterized by inconsistent limit setting by parents or caregivers in response to a child's bedtime struggles or night waking. Whereas consistent limit-setting reduces the frequency and severity of problematic behaviors at these times, inconsistent or suboptimal responses to a child's inappropriate behavior tend to perpetuate the sleep problem.

Treatment of insomnia and night waking for infants and young children begins with optimization of sleep hygiene, practices that promote optimal nighttime sleep. Structured bedtime routines appropriate for age help many younger children transition from a higher level of daytime activity to a quieter and more relaxed state that facilitates sleep onset. Routines for younger children often include bathing, tooth-brushing, changing clothes, reading stories, and being ''tucked in.'' Potentially stimulating activities such as vigorous play or watching television are best avoided in children who have difficulty with settling at bedtime.

Establishing an optimal sleep environment is also important for younger children with sleep problems. To the extent possible, the child should be provided with an age-appropriate crib or bed in a quiet and comfortable environment. Exposure to ambient light at bedtime and during the night should be minimized apart from judicious use of small night lights for children who are afraid of the dark. Maintaining a regular sleep schedule can promote circadian entrainment, which often facilitates sleep onset close to the habitual bedtime.

Structured behavioral interventions are often additionally necessary for effective treatment of behavioral insomnia of childhood. Well-established methods include *extinction* (systematic ignoring of crying or other behaviors until the child eventually falls asleep) and *graduated extinction* (a variation in which extinction techniques are applied for progressively increasing intervals before parents are allowed to briefly resettle their child).[7,8] *Bedtime fading* involves transiently moving a child's bedtime later, ie, closer to the time of actual sleep onset, with eventual return to an earlier, age-appropriate bedtime after the child is falling asleep more quickly.[9] Consistent and sustained application of these interventions is usually necessary to achieve sustained clinical improvement in children with more severe forms of bedtime struggles and night waking.

Drug treatment of insomnia in infants and younger children has received scant formal study and is not routinely recommended.

INSOMNIA IN OLDER CHILDREN AND ADOLESCENTS

Insomnia remains common as children grow older, affecting at least 10% of adolescents,[10] but underlying causes change substantially with advancing age. Older children and adolescents have increasing autonomy with respect

KEY POINTS

- Sleep-onset association disorder is one of the most common underlying or contributing causes for insomnia and night waking in infants and younger children.
- Many families try extinction-based interventions for only a few nights, encounter an ''extinction burst'' of temporarily worse symptoms, and abandon the technique before it has had time to be effective.

KEY POINTS

- Delayed sleep phase is an extremely common cause or contributing factor to insomnia in adolescent or older preadolescent children. When present, medication treatment alone is unlikely to be effective.
- ''Sleeping in late'' on weekend and non–school days can reduce the effectiveness of other interventions for delayed sleep phase.

to bedtime and sleep schedule as they grow older, often resulting in bedtimes that are too late to permit habitually sufficient nighttime sleep. Evening-time use of electronic devices such as computers, smart phones, televisions, or video games can delay bedtime or be sufficiently stimulating to delay sleep onset even after cessation of the activity.[11] Consumption of caffeinated beverages, particularly late in the day, may also be associated with insomnia and sleep problems in this age group.[12]

One of the most important influences that affect insomnia in older children and adolescents is the well-recognized tendency for many children in this age range to become ''night owls'' who gravitate toward later bedtimes and waking times.[13] *Circadian rhythm disorder, delayed sleep-phase type*, can be diagnosed when a tendency toward delayed sleep phase is associated with habitual inability to fall asleep and/or wake up at desired or socially acceptable times. Some patients with this disorder compensate for insufficient nighttime sleep with daytime naps, which may lead to further difficulties falling asleep at a conventional bedtime. The common adolescent practice of habitually later bedtime and waking time on non–school nights can lead to worsening of the sleep-phase delay and result in additional irregularity of sleep schedule compared to school nights.[14]

Other forms of insomnia, such as psychophysiologic insomnia and insomnia due to drug or substance, can also affect older children and teenagers, with symptoms and contributory influences similar to those of affected adults. Insomnia may occur secondary to underlying medical conditions such as attention deficit hyperactivity disorder or developmental disabilities. Insomnia can also represent an associated feature of other sleep disorders such as restless legs syndrome.

Treatment of insomnia for older children and adolescents is seldom successful unless all pertinent influences are addressed and the child is motivated enough to make the lifestyle and sleep schedule changes that are usually necessary to correct the problem.

Sleep hygiene should be examined and optimized before more specific and labor-intensive treatments are implemented. Any excessive consumption of caffeinated beverages should be normalized on a gradual basis to avoid withdrawal symptoms and any late-day intake should be eliminated. Potentially stimulating activities, including homework, vigorous exercise, or use of electronic devices, should be moved to alternative times and optimally replaced with a structured prebedtime routine incorporating less stimulating activities.

A target weekday sleep schedule should be identified based on the child's age, school schedule, individual sleep needs, and family needs. Efforts should be made to keep bedtime and waking time on non–school days consistent with those on school days to eliminate irregularity of sleep schedule. Any daytime napping should be eliminated. Sleep schedule goals should be negotiated with the family in a pragmatic fashion, which sometimes requires that initial targets be achievable, although perhaps not ideal.

When insomnia related primarily to delayed sleep phase is mild, consistent implementation of the measures described above are sufficient to alleviate insomnia and permit adequate nighttime sleep duration. Gradual but consistent advancement of the child's bedtime and waking time toward the earlier times of the target schedule may be additionally necessary.

Chronotherapy is a useful method for treating severe or resistant forms

of insomnia related to delayed sleep phase. This treatment entails progressive delays of the child's bedtime and waking time by 2 to 3 hours daily until the target sleep schedule is attained, typically within 5 to 7 days.[15,16] The technique is rapid, safe, and effective, but benefits are only sustained when the target sleep schedule is rigorously maintained following the initial treatment. Light therapy using carefully timed exposure to high-intensity full-spectrum sources following morning waking is also sometimes used for treatment of delayed sleep phase in children and adolescents.[16]

Drug treatment of insomnia in older children and adolescents has not been rigorously studied despite seemingly widespread use of homeopathic, nonprescription, and off-label prescription agents in this population.[17,18] Several studies have reported melatonin to be effective in treating insomnia for school-aged children.[19,20]

OBSTRUCTIVE SLEEP APNEA IN CHILDREN

Obstructive sleep apnea (OSA) is estimated to affect about 2% of children, with peak incidence thought to occur between 3 and 8 years of age.[21] Clinical features of OSA in children compared to adults are summarized in **Table 10-1**.[22]

The nighttime symptoms exhibited by affected children are similar to, but often milder than, those exhibited by adults. Most children with OSA have some degree of snoring or noisy respiration during sleep, but this may be mild or intermittent. Other common but variable nighttime symptoms include mouth breathing, restlessness, and diaphoresis. Dramatic pauses in respiration during sleep are observed by parents and caregivers only when OSA is severe, consistent with current understanding that childhood OSA is more commonly characterized by intermittent or sustained partial obstruction during sleep as opposed to recurrent episodes of complete obstruction (**Supplemental Digital Content 10-1**, *links.lww.com/CONT/A28*).

Daytime symptoms of OSA in children may be more subtle or variable compared to adults. Children with OSA are seldom obviously sleepy during the day unless nighttime airway obstruction is severe. Daytime sleepiness in affected children is more commonly intermittent or mild, sometimes evident only during sustained sedentary activities such as riding in an automobile. Disturbances in attention, behavior, and academic performance represent more common and prominent daytime symptoms in children with OSA, but these are frequently misattributed to alternative causes, such as attention deficit hyperactivity disorder (ADHD).

A small proportion of children with OSA may present with isolated systemic or constitutional symptoms such as failure to thrive or unexplained hypertension.[23] A variety of medical conditions may be associated with increased risk for childhood OSA (**Table 10-2**),[24] including Down syndrome, Prader-Willi syndrome, cleft palate repair, and craniofacial disorders characterized by micrognathia, such as Pierre Robin syndrome (**Figure 10-2**).[25,26] Obesity represents an increasingly prevalent risk factor for OSA in children, particularly during adolescence.[27]

Although the physical examination findings of children with OSA are frequently normal, some children exhibit characteristic findings associated with increased risk for upper airway obstruction during sleep, including mouth breathing, tonsillar hypertrophy, narrow upper palate, and maxillary or mandibular hypoplasia. Although tonsillar hypertrophy is common among children

KEY POINTS

- Obstructive sleep apnea is underdiagnosed in children in part because the lack of observed respiratory pauses and obvious daytime sleepiness in most affected children limit parent and medical practitioner recognition that the condition might be present.
- Although obesity represents a risk factor for childhood obstructive sleep apnea, children with low or normal body weight can have substantial obstructive sleep apnea, particularly when underlying adenotonsillar hypertrophy is present.

TABLE 10-1 Comparative Features of Obstructive Sleep Apnea in Children Compared to Adults[a]

Features	Children	Adults
Physical characteristics		
Gender	Younger children: sexes equally affected	Primarily males, postmenopausal females
	Adolescents: males>females	
Peak age	2–8 Years	Middle aged and older
Body weight	Usually normal, occasionally overweight	Most often obese
Upper airway	Adenotonsillar enlargement frequent	Adenotonsillar enlargement occasional
	Redundant soft tissue occasional	Redundant soft tissue frequent
Symptoms during sleep		
Snoring	Frequent, continuous, or intermittent	Frequent, often interrupted by pauses
Witnessed apnea	Occasional	Frequent
Polysomnographic characteristics		
Obstruction	Prolonged partial obstruction>intermittent	Cyclical intermittent obstruction
Sleep architecture	Normal>fragmented	Frequent arousals with sleep fragmentation
Secondary symptoms		
Daytime sleepiness	Most often absent or intermittent	Frequent, usually prominent
Neurobehavioral	Inattention, hyperkinesis, disturbed behavior	Cognitive slowing, increased accident risk
Cardiovascular	Hypertension, cor pulmonale	Hypertension, heart disease, stroke

[a] Reprinted with permission from Hoban T, Ann N Y Acad Sci.[22] *onlinelibrary.wiley.com/doi/10.1111/j.1749-6632.2009.05112.x/abstract*.

with OSA (**Figure 2-4** in ''Approach to and Evaluation of Sleep Disorders''), its presence is neither necessary nor sufficient for the diagnosis of childhood OSA.

Current criteria for the diagnosis of OSA in children were established in 2005 by the *International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual*.[1] Definitive diagnosis of the disorder requires a combination of clinical criteria associated with symptomatic OSA (eg, snoring, diaphoresis, daytime hyperactivity, or behavior problems) and polysomnographic confirmation that airway obstruction associated with disturbed gas exchange or sleep architecture is present. Whereas the diagnostic criteria for adult OSA require that at least five respiratory disturbances per hour be present during polysomnography (PSG), pediatric criteria require a minimum of only one disturbance per hour,

TABLE 10-2 Medical Conditions Associated With Increased Risk for Childhood Obstructive Sleep Apnea[a]

▶ **Craniofacial Syndromes Featuring Prominent Maxillary or Mandibular Hypoplasia**
Apert syndrome
Crouzon syndrome
Pierre Robin syndrome
Saethre-Chotzen syndrome
Treacher Collins syndrome
▶ **Other Skeletal and Craniofacial Disorders**
Achondroplasia
Choanal atresia
Cleft palate, especially following surgical repair
Velocardiofacial syndrome
▶ **Systemic Genetic and Metabolic Disorders**
Down syndrome
Hypothyroidism
Mucopolysaccharide storage disorders (eg, Hunter syndrome, Hurler syndrome)
Prader-Willi syndrome
▶ **Other Medical and Neurologic Conditions**
Brainstem and cranial nerve disorders (eg, syringobulbia, Chiari malformation)
Chronic nasal obstruction (eg, septal deviation, allergic rhinitis, polyp, infections)
Obesity
Sickle cell disease

[a] Reprinted from Hoban TF, Chervin RD, Sleep Med Clin.[24] © 2007, with permission from Elsevier. *www.sciencedirect.com/science/article/pii/S1556407X07000550*.

KEY POINT

- Of children undergoing adenotonsillectomy for treatment of obstructive sleep apnea, 50% to 75% may still have some degree of obstructive sleep apnea postoperatively (usually milder).

reflecting the increased vulnerability of children to the effects of mild nocturnal airway obstruction compared to adults.[28]

Adenotonsillectomy represents the most commonly used treatment for OSA in children. Although this procedure had historically been considered to be highly effective in treating childhood OSA,[29] recent studies using contemporary diagnostic criteria and PSG have suggested that respiratory parameters during sleep fully normalize in only 25% to 50% of children with OSA who undergo adenotonsillectomy.[30,31] Although these studies should be interpreted with due recognition that many of the children whose OSA was not completely cured by adenotonsillectomy nevertheless experienced significant partial improvement of their OSA, these and other reports suggest that

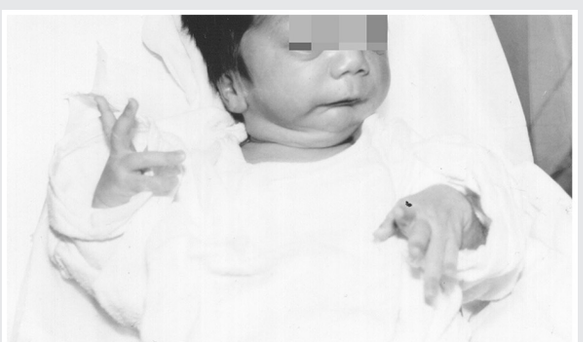

FIGURE 10-2 Child with Pierre Robin syndrome and micrognathia.

Reprinted from Tewfik TL, Der Kaloustian VM, Oxford University Press.[25] By permission of Oxford University Press, USA.

KEY POINT

- Continuous positive airway pressure is considered a first-line treatment for obstructive sleep apnea in children.

clinicians must remain vigilant for residual or recurrent OSA following adenotonsillectomy in children.[32,33]

Most children with OSA who undergo adenotonsillectomy exhibit tangible improvements in obstructive symptoms during sleep and in overall sleep quality. The impact on daytime symptoms may be variable, especially when comorbid non-OSA influences are present, but multiple studies have reported improved academic performance or ADHD symptoms in children whose OSA was treated via adenotonsillectomy.[34,35]

Continuous positive airway pressure (CPAP) is also considered a first-line treatment for childhood OSA. Although used less frequently than adenotonsillectomy for the pediatric population, CPAP is particularly useful in children whose OSA persists despite adenotonsillectomy and in children who are not appropriate candidates for adenotonsillectomy (eg, morbid obesity, craniofacial disorders, and other conditions in which airway obstruction is not primarily related to adenotonsillar obstruction) (**Case 10-2**).

Case 10-2

An 8-year-old boy with a history of autoimmune hepatitis and recent liver transplantation was referred for evaluation of snoring and daytime sleepiness. Snoring had become severe since liver transplantation and was associated with mouth breathing and significant restlessness during sleep. The family also reported substantial daytime tiredness for at least 1 year despite 10 hours of sleep per night on average. The child typically napped after school 2 to 3 days weekly and would fall asleep quickly during automobile rides. His teachers at school reported substantial problems with drowsiness, inattention, oppositional behavior, and poor academic performance. Initial examination was notable for obesity (body mass index of 34 kg/m^2) and noisy mouth breathing. Examination of the oropharynx was significant for a Mallampati class IV airway, large scalloped tongue, mildly high-arched palate, and 2+ tonsillar size.

A baseline polysomnogram confirmed the presence of severe obstructive sleep apnea (OSA), characterized by frequent obstructive apneas and hypopneas associated with marked sleep fragmentation and prominent desaturation of SpO_2. The apnea-hypopnea index was 86.7 events/h and 9.7% of total sleep time spent with SpO_2 levels below 90.0%. End-tidal carbon dioxide monitoring demonstrated no sleep-related hypoventilation.

The patient underwent adenotonsillectomy and was admitted overnight for postoperative respiratory monitoring (as is standard practice for children with severe sleep apnea undergoing this procedure). Following the child's recovery from surgery, his family reported that his snoring and restlessness during sleep had improved substantially and become mild in severity. Daytime somnolence and napping had nearly resolved. The child's teachers reported little improvement with respect to inattention and distractibility at school, however, and told the child's mother that he was at risk of not being promoted to the next grade.

A postoperative polysomnogram demonstrated mild OSA, characterized by hypopneas associated with mild desaturation of SpO_2 and slight fragmentation of sleep architecture. The apnea-hypopnea index was

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1.9 events/h, rising to 6.2 during REM sleep. A subsequent continuous positive airway pressure trial demonstrated that pressure settings of 7 cm and 9 cm of water provided good control of obstructive respiratory disturbances during sleep.

The child was highly compliant with home CPAP use, and the family reported that snoring and restlessness during sleep resolved completely when it was initiated. Inattention and distractibility at school improved moderately after CPAP was started, and the family reported no substantial academic concerns at their last appointment.

Comment. This case illustrates several important clinical aspects of childhood OSA, including the facts that severe OSA frequently persists in a milder form following adenotonsillectomy, that mild OSA without daytime somnolence may still be associated with neurobehavioral sequelae that benefit from treatment, and that CPAP can successfully be implemented as long-term treatment for OSA in a relatively young child with complex medical needs.

CPAP is generally considered to be both safe and effective for the treatment of childhood OSA, although acquired maxillary hypoplasia has been reported as a rare complication of long-term treatment.[36] Limitations of this treatment in children relate primarily to the fact that not all children successfully acclimate to use of the device or achieve long-term compliance with its use. Use of CPAP may be especially challenging in young or developmentally disabled children, although structured desensitization techniques are sometimes effective in helping these patients achieve successful long-term use.

Alternative treatments for childhood OSA are considered when the first-line treatments are either ineffective or not feasible. Several studies have suggested that use of nasal steroids or maxillary expansion devices may help alleviate upper airway obstruction for some children with OSA, but the overall effectiveness and safety of these treatments have not been well established.[37,38] Use of supplemental oxygen or positional therapy during sleep may provide some (often partial) benefit for children with OSA. Children with severe and otherwise refractory OSA sometimes benefit from alternative surgical interventions, such as uvulopalatopharyngoplasty, maxillary or mandibular advancement, or even tracheostomy, although pediatric data regarding these procedures are extremely limited.

EXCESSIVE DAYTIME SOMNOLENCE IN CHILDREN

Healthy children and adolescents who receive sufficient nighttime sleep normally exhibit excellent levels of daytime alertness and seldom nap during the day. The presence of persistently excessive sleepiness in a child generally signifies one of three major possibilities:

1. That the child is not receiving sufficient nighttime sleep
2. That an underlying sleep disorder has severely disrupted nighttime sleep
3. That a sleep or medical disorder that may cause sleepiness even in the absence of disrupted nighttime sleep (eg, narcolepsy, anemia, hypothyroidism) is present

Determining whether a young patient is receiving habitually sufficient nighttime sleep requires careful review

KEY POINTS

- Nasal steroids and rapid maxillary expansion represent promising alternative techniques for treatment of childhood obstructive sleep apnea.
- Some pediatric studies suggest that mild obstructive sleep apnea is more likely to be associated with symptoms suggestive of attention deficit hyperactivity disorder than more severe forms are.
- The presence of significant sleepiness in a child with snoring is concerning for the presence of severe underlying obstructive sleep apnea.

KEY POINT

- Chronically insufficient nighttime sleep represents a very common cause of sleepiness and poor academic performance in children.

TABLE 10-3 Ontogeny of Sleep EEG Background and Sleep Architecture During Infancy and Childhood

▶ **Primary EEG Background Activity During Sleep in Premature Infants**
26–30 Weeks postconceptional age: tracé discontinue
28–30 Weeks postconceptional age: delta brushes (disappear by term)
32–36 Weeks postconceptional age: tracé alternant (disappears by 12 weeks postterm)

▶ **Ontogeny of Key Sleep-Staging Landmarks**
30 Weeks postconceptional age: REMs apparent
2 Months postterm: sleep spindles apparent
4–6 Months: K complexes and well-differentiated non-REM stages

▶ **Development of the Waking Posterior Dominant Rhythm**
2 Months: ≥4 Hz
6 Months: ≥6 Hz
3 Years: ≥8 Hz
9 Years: ≥9 Hz

▶ **Ontogeny of Sleep Architecture**
Term neonates: 50% REM; 50% non-REM (sleep-onset REM common)
6–12 Months: 30%–35% REM; 65%–70% non-REM
3–5 Years: 25% REM; 75% non-REM

of the child's sleep schedule and comparison to age-appropriate norms (**Table 10-1**).[1] If daytime sleepiness resolves after nighttime sleep is lengthened, additional investigation and treatment are often unnecessary.

When excessive sleepiness persists despite adequate or lengthened nighttime sleep, other potential causes must be explored. A detailed medical history often identifies symptoms and risk factors that guide further investigation, such as symptoms of nocturnal sleep disruption (eg, snoring, restlessness), a history of medical disorders that can be associated with fatigue and sleepiness (eg, depression, Epstein-Barr virus infection), or use of potentially sedating

TABLE 10-4 Pediatric Norms for Multiple Sleep Latency Testing[a,b]

Tanner Stage	Mean Sleep Latency (Minutes)[c]	SD
Stage 1	19.0	1.6
Stage 2	18.5	1.9
Stage 3	16.1	3.8
Stage 4	15.8	3.4
Stage 5	16.6	2.1
Older adolescents	15.7	3.4

SD = standard deviations.
[a] Adapted from Carskadon MA, Addison-Wesley.[43]
[b] Reprinted from Hoban TF, Chervin RD, Semin Pediatr Neurol.[44] © 2001, with permission from Elsevier.
[c] Data averaged from tests performed on 3 successive days of recording.

medications. Physical examination sometimes identifies additional clues, such as anatomic features associated with increased risk for OSA or physical findings suggestive of underlying anemia or hypothyroidism.

Although sleepiness in children is most often secondary to insufficient nighttime sleep or another underlying sleep disorder, such symptoms are occasionally the result of hypersomnias of central origin, such as recurrent hypersomnia (Kleine-Levin syndrome) or narcolepsy.

Kleine-Levin syndrome is a rare condition characterized by recurrent episodes of sleepiness, encephalopathy, and altered appetite that last days to weeks.[5,39] Behavioral disturbances during episodes may include aggression or inappropriate sexual behaviors that require additional treatment and safety precautions beyond symptomatic treatment of the hypersomnia. This disorder most often affects adolescent boys and sometimes remits spontaneously with time.

Narcolepsy in children is characterized by the same clinical manifestations

Case 10-3

An 8-year-old girl was referred by another neurologist for further evaluation of pervasive daytime sleepiness and intermittent weakness sometimes associated with loss of posture. The family reported that her pervasive sleepiness had been noted during the prior school year, when the child had occasionally fallen asleep in school. Sleepiness at the time of initial evaluation was described as substantial, with the child sleeping almost constantly on automobile rides and napping for up to 90 minutes daily despite 10 to 11 hours of sleep during most nights.

Episodic weakness was first noted at 2 years of age and became gradually more prominent with advancing age. Events were reported to occur almost exclusively during giggling or laughter, persisting for several seconds to several minutes before subsiding. Milder episodes were characterized only by brief, subtle dipping of the head, whereas more severe events were characterized by diffuse weakness and loss of postural tone. (The accompanying video [**Supplemental Digital Content 10-2**, *links.lww.com/CONT/A29*] depicts a different patient with a similar condition.) Several spells had been associated with falls resulting in broken bones.

The family reported no snoring or significant nighttime sleep problems apart from brief partial awakenings and mild restlessness during sleep. They observed no hallucinatory events or sleep paralysis near sleep onset or offset. Episodes of weakness and loss of posture were not associated with any impairment of consciousness, and an extensive prior workup for seizures had been negative. The initial examination was remarkable only for mouth breathing, a mildly narrow upper palate, and equivocal limitation of upward gaze.

A nocturnal PSG was essentially normal, recording 458.5 minutes of sleep and no significant respiratory disturbances. A multiple sleep latency test performed the next day recorded sleep during all five nap opportunities, with mean sleep-onset latency of 0.7 minutes and four sleep-onset REM periods. Findings were interpreted as supporting the presence of severe sleepiness and compatible with the diagnosis of narcolepsy.

Daytime sleepiness and cataplectic episodes improved considerably following initiation of treatment with modafinil and tricyclic agents. School performance also improved, but declined several months later despite continued good control of sleep-related symptoms. This prompted further diagnostic investigation. A skin biopsy and cholesterol esterification studies demonstrated findings consistent with Niemann-Pick disease type C.

Comment. This case illustrates several important clinical aspects of childhood narcolepsy, including multiple sleep latency test findings that were strongly indicative of childhood narcolepsy, the fact that narcolepsy often responds well to off-label treatment using modafinil, and that children who present with narcolepsy at a younger-than-typical age or experience progressive symptoms despite treatment may require screening for other underlying disorders.

experienced by adults: daytime somnolence, sleep paralysis, hypnagogic and hypnopompic hallucinations, and sometimes cataplexy. Onset of the condition is occasionally abrupt, but symptoms more commonly evolve in an insidious fashion over time. Clinical manifestations most commonly begin during the second decade of life, and onset at younger ages is sometimes associated with the presence of additional underlying conditions such as Niemann-Pick disease type C or Prader-Willi syndrome.[40,41]

Sleep laboratory assessment of excessive sleepiness in children often requires both PSG and multiple sleep latency testing (MSLT). Investigation commences with nocturnal PSG, which documents the duration of nighttime sleep and screens for OSA and other potential disruptors of nighttime sleep. The nocturnal PSG should be interpreted using pediatric norms[42] (**Table 10-3**), which differ substantially for children compared to adults. MSLT is performed the next day to objectively assess the severity of daytime somnolence and screen for sleep-onset REM periods and other findings that help differentiate between narcolepsy and other potential causes of excessive sleepiness. Because mean sleep latency scores for healthy children undergoing MSLT are significantly longer than those for adults, it is essential that the nap study also be interpreted using pediatric norms (**Table 10-4**).[43,44] Most children with narcolepsy demonstrate multiple sleep-onset REM periods and mean sleep latency of less than 5 minutes on MSLT.

Treatment of excessive daytime sleepiness in children is customized based on the severity of somnolence and the nature of the associated clinical circumstances. Some sleepy children require nothing more than lengthening nighttime sleep or treatment of an associated medical or sleep disorder that is causing sleepiness on a secondary basis. Children with hypersomnias of central origin such as narcolepsy, however, usually benefit from treatment with wake-promoting medications, such as modafinil, stimulants, or sodium oxybate, which are used cautiously on an off-label basis in this population. Off-label use of selective serotonin reuptake inhibitors or tricyclic agents is sometimes effective in controlling cataplexy for children with narcolepsy (**Case 10-3**).

VIDEO LEGENDS

Supplemental Digital Content 10-1

Sleep apnea syndrome in a child. Video demonstrates sleep apnea syndrome in a child. This child is overweight and has grade 4+ tonsils, which has led to complete cyclical airway occlusion and resuscitative arousals. Obstructive sleep apnea in children may have different causes and consequences than in adults. Children with untreated obstructive sleep apnea may experience neurocognitive decline, hyperactivity, hypersomnolence, and decline in IQ scores.

links.lww.com/CONT/A28

Supplemental Digital Content 10-2

Child cataplexy. Video demonstrates cataplexy in a child, depicting loss of muscle tone triggered by laughter.

links.lww.com/CONT/A29

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