



A Review of Pediatric Nonrespiratory Sleep Disorders*

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Sleep problems are extremely common during childhood, from infancy to adolescence. Despite the prevalence of sleep problems, childhood sleep disorders are often underrecognized and undiagnosed, despite being either preventable or treatable. Sleep impacts almost all aspects of a child's functioning, and thus the increased recognition and treatment of sleep disorders will positively affect a child's well-being. Children experience the same broad range of sleep disturbances encountered in adults, including sleep apnea, insomnia, parasomnia, delayed sleep phase, narcolepsy, and restless legs, but their clinical presentation, evaluation, and management may differ. Although snoring and sleep apnea may be the most common indication for an overnight sleep study in a child, one quarter of children presenting to a sleep clinic for evaluation will have a second sleep diagnosis, which is often nonrespiratory in nature. Especially in children, ruling out sleep apnea is rarely the end point of the sleep evaluation. Clinicians involved in sleep medicine must be prepared to recognize, evaluate, and manage plans for sleep disorders across the lifespan of the patient. This article will provide an updated review of nonrespiratory pediatric sleep disorders within a developmental framework. (CHEST 2006; 130:1252-1262)

Key words: behavioral sleep disorders; delayed sleep phase; insomnia; narcolepsy; parasomnias; restless leg syndrome

Abbreviations: ADHD = attention deficit hyperactivity disorder; DSPS = delayed sleep phase syndrome; ICD-9 = *International Classification of Diseases*, ninth revision; ICSD-2 = *International Classification of Sleep Disorders*, second edition; MSLT = multiple sleep latency test; NREM = non-rapid eye movement; PLMD = periodic limb movement disorder; PTSD = posttraumatic stress disorder; REM = rapid eye movement; RLS = restless leg syndrome; RMD = rhythmic movement disorder; SDB = sleep-disordered breathing

Increased research and clinical advances in sleep medicine over the past 2 decades have led to improved recognition of pediatric sleep disorders. According to polls from the last few years^{1,2} about

sleep in US children, three fourths of parents would change something about their child's sleep, 10 to 14% of parents have asked their doctor about a sleep concern, and today's children are not getting enough sleep (Table 1). Chronic sleep problems can have a major negative impact on child and family functioning. Sleep impacts children's physical, emotional, cognitive, and social functioning, and sleep problems can exacerbate any existing medical, psychiatric, or developmental disorder.

Although snoring and sleep-disordered breathing (SDB) may be the most common reasons for referring a child to a sleep laboratory, almost 25% of these children may have a second sleep disorder that is associated with equally important clinical morbidity.³ Similar to the case in adults, and especially in children, ruling out sleep apnea is rarely the end point of the sleep evaluation. The prevalence of the most common nonrespiratory sleep disorders are summarized in Table 2. In our local experience, at least one third of pediatric sleep medicine referrals

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Table 1—The Amount of Sleep Children and Adolescents Need Compared to How Much They Are Getting*

Age Group	Recommendation, h	Study Finding, h
Infants (3–11 mo)	14–15	12.7
Toddlers (12–35 mo)	12–14	11.7
Preschoolers (3–6 yr)	11–13	10.3
School age (1st–5th grade)	10–11	9.5
Adolescents (6th–12th grade)	9.25	7

*From the Sleep in America Polls 2004¹ and 2006.²

have other medical or neuropsychiatric comorbidities that complicate diagnostic assessment and challenge management. Today's sleep medicine specialist must be prepared to recognize, evaluate, and create management plans for sleep disorders across the lifespan of the patient. This article is designed to provide current information regarding the most common nonrespiratory sleep disorders in children from a developmental perspective (Fig 1) and to broadly describe the current state of treatment. More comprehensive information about the clinical assessment of pediatric sleep disorders, symptom-based algorithms for evaluation and management, and specific sleep disorders in children are available elsewhere.^{4–6}

INFANTS AND TODDLERS

Behavioral Insomnia of Childhood

Behavioral insomnia of childhood (*International Classification of Diseases*, ninth revision [ICD-9] code 307.42; *International Classification of Sleep Disorders*, second edition⁷ [ICSD-2] code V69.5 can be used with another primary diagnosis), a new category in the ICSD-2, is defined as difficulty falling and/or staying asleep with an identified behavioral etiology. It is a type of "learned insomnia" with some analogies to psychophysiologic insomnia in adults. The sleep-onset-association type is characterized by the child's dependency on stimulation, objects, or environments for initiating or returning to sleep. The limit-setting type is typified by bedtime stalling or refusal resulting from inadequate limit setting. Young children often have features of both types. This disorder is extremely common, with an estimated prevalence of 10 to 30%.⁸ Behavioral insomnia of childhood may be preventable with early intervention and parent education about appropriate sleep habits (*ie*, regular bedtimes and sleep routines, and putting the child to bed awake so she or he learns to fall asleep independently).

Table 2—Prevalence of Nonrespiratory Pediatric Sleep Disorders*

Sleep Disorder or Behavior	Prevalence, %/Study
Sleep talking	55/Owens et al ¹²
Bruxism	28.1/Owens et al ¹²
Night terrors	17.3/Owens et al ¹²
Rhythmic movements	17.2/Owens et al ¹²
Behavioral insomnia of children (including bedtime refusals and night wakings)	10–30/AASM ⁷
Confusional arousals	17/Littner et al ¹⁰³
Sleepwalking	13.8/Owens et al ¹²
Nightmares	10–50/Sheldon et al ⁶
Insomnia	
Children	1–6/AASM ⁷
Adolescents	10.7/Hoban and Chervin ¹⁰¹
Delayed sleep phase	5–16/Anderson ¹⁰⁰
Restless leg syndrome	Unknown
Narcolepsy	0.05/Dauvilliers et al ⁹¹

*AASM = American Academy of Sleep Medicine. From the new ICSD-2 nomenclature.⁶

Polysomnography is rarely indicated since the problem is waking behaviors, not sleep *per se*. The management of behavioral insomnia begins with parent education about establishing appropriate sleep habits and usually includes some form of extinction, wherein the undesired behavior (*ie*, bedtime resistance) is gradually faded out and is replaced by another, more appropriate bedtime behavior. The treatment efficacy of behaviorally based strategies such as extinction is supported by evidence-based reviews.^{9,10} Extinction involves ignoring the child's negative behaviors (*eg*, crying, yelling, or requests to get out of bed) until it is time to wake or a more graduated approach that includes scheduled parental checks.^{11,12} In cases in which parents are willing and able to implement a plan on their own, education about extinguishing negative sleep-related associations, information from the Internet and/or self-help books, and collaborative development of an extinction plan may be sufficient. For certain families, such as those with long-standing or complex complaints, referral to a mental health professional may be required.

Sleep-Related Rhythmic Movement Disorder

Sleep-related rhythmic movement disorder (RMD) [ICD-9 code 307.3] consists of rhythmic movements that occur on falling asleep or during any stage of sleep and can involve any part of the body with a frequency ranging from 0.5 to 2 Hz.^{13–16} Seen most frequently in childhood, the motor activity is characterized by repetitive and stereotyped body motions, and includes a spectrum of behaviors (*eg*,

Infant/ Toddler (1-2 yrs) Behavioral Insomnia of Childhood Rhythmic Movements	Preschool (3-5 years) Behavioral Insomnia of Childhood Sleep Terrors Rhythmic Movements	School-Age (6-12 years) Insufficient Sleep Bedtime Resistance Sleep-Walking	Adolescence (13-18 years) Insufficient Sleep Delayed Sleep Phase Narcolepsy
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FIGURE 1. Common nonrespiratory sleep problems by age group.

head banging, head rolling, and body rocking). Rhythmic movements occur in a high proportion of otherwise healthy children as a self-limiting phenomenon starting and remitting within early childhood. However, some forms of RMD occur against a background of developmental, psychiatric, or neurologic disorders (eg, mental retardation, autistic spectrum disorders, attention deficit hyperactivity disorder [ADHD], obsessive-compulsive disorders, and after head trauma or encephalitis), persist beyond childhood, or have their onset in adulthood.¹⁷ Thus, RMD can be both a physiologic and a pathologic phenomenon. When the clinical presentation alone is not sufficient to provide diagnostic certainty, videopolysomnography or video-EEG monitoring can be useful.¹⁸

Most typically developing children with RMD do not require specific treatment, and the family can be reassured that most children outgrow the condition uneventfully. Management includes education about the benign, transient nature of the condition and simple safety measures. Further treatment is rarely indicated unless RMD interferes with sleep or is associated with injury, daytime dysfunction, or developmental delay. For children with developmental disabilities or particularly violent movements, judicious use of a protective helmet or padding in the crib or bed should be considered.¹³ Other proposed treatments include hypnosis¹⁹ and other behavioral modification techniques.^{13,20,21} Drug treatment of RMD has not been systematically studied in children, but there are anecdotal reports of successful treatment with benzodiazepines (specifically clonazepam), tricyclic agents, and atypical antipsychotics.^{13,22–27} Serious injury to the eyes, soft tissues, or bone has been reported in extreme cases.^{13,28–30} When head banging occurs in the setting of developmental disabilities and/or causes tissue damage, referral to a mental health professional with expertise in the comprehensive behavioral and pharmacologic management of self-injurious behaviors is recommended.

PRE-SCHOOL-AGED CHILDREN (3 TO 5 YEARS OF AGE)

Bedtime resistance, difficulty falling asleep, and night awakenings are common in preschoolers. As a

result of expanded language and cognitive skills, children become more articulate in their protests and engage in more limit-testing behavior. The limit-setting type of behavioral insomnia of childhood becomes more prominent. Common sleep issues are naps (92% of 3-year-olds and 57% of 4-year-olds nap, but only 27% of 5-year-olds nap) and consistent sleep schedules.⁵

Nighttime Fears

Dark rooms and imaginary creatures are two common fears in preschoolers. Such fears are usually short-lived and benign, typically disappearing by age 5 to 6 years. Almost all children experience nighttime fears, with a peak between ages 3 and 6 years, and a second peak in school-aged girls, especially those who are highly anxious. Parental anxiety and family conflict also play a role perpetuating the nighttime fears by raising the child's level of emotional arousal. The differential diagnosis includes bedtime resistance (when the expression of fears is used as a stalling technique), nightmares, anxiety disorders, and child abuse. An important aspect of treatment is counseling parents about responding to nighttime fears, including maintaining a balance between reassurance and reinforcement. Children and adolescents with persistent or severe bedtime fears who are unresponsive to parental intervention should be referred to a mental-health professional.

Nightmare Disorder

Nightmares (ICD-9 code 307.47) are common in childhood, with prevalence estimates of 5 to 30%, and 75% of children report experiencing at least one nightmare,⁵ which is a frightening dream occurring in rapid eye movement (REM) sleep, usually resulting in awakening. The content of nightmares differs across age groups. While toddlers worry about separation from parents, preschoolers may incorporate frightening imaginary creatures. Nightmares have the following features: occurrence in the last half of the night when REM predominates; recollection of dream content; morning recall of the event; no confusion or disorientation; and delayed return to sleep.

Risk factors include stressors, traumatic events, anxiety, sleep deprivation, medications that alter

REM sleep, and withdrawal of therapy with medications that suppress REM sleep. Specifically, children with other medical or neuropsychiatric conditions may be treated with a variety of drugs that can trigger nightmares and bizarre dreams including the following: sedative/hypnotics; β -blockers; amphetamines; dopamine agonists; nonsteroidal antiinflammatory drugs; leukotriene receptor antagonists like montelukast; certain antibiotics (*eg*, erythromycin); and antimigraine medication (verapamil). Withdrawal from therapy with medications that are REM suppressants (*eg*, clonidine, the most popular prescription sedative/hypnotic in children,³¹ and many antidepressant or anxiolytic agents) can result in REM sleep rebound, vivid dreaming, and nightmares.

The differential diagnosis for nightmares includes sleep terrors and posttraumatic stress disorder (PTSD) in cases in which recurrent nightmares are common. Events such as car accidents, which may not be traumatic for parents, may be traumatic for children, so a detailed inquiry into stressful events is crucial. The management of nightmares for younger children includes active parental reassurance, while older children may benefit from positive reinforcement for developing independent coping skills such as relaxation. Problem nightmares that are medication-related should be self-limited once the offending medication is recognized and discontinued or the REM rebound from drug withdrawal has resolved. Children with nightmares that are persistent, severe, or suggest PTSD or other psychiatric disturbances should be referred to a mental-health professional.

Parasomnias, Disorders of Arousal From Non-REM Sleep

Disorders of arousals from non-REM (NREM) sleep are more common in children than adults because children spend more time in deep NREM sleep. Such disorders usually occur within 1 to 2 h after sleep onset and coincide with the transition from the first period of slow-wave sleep. They are often present only once a night, and if they do recur, their periodicity matches ultradian sleep cycles. Other common features include difficult arousal, amnesia for the event, and a range of automatic behavior and autonomic arousals. Any conditions that lead to sleep fragmentation or deprivation or that increase slow-wave sleep (*eg*, increased body temperature) can predispose a child to parasomnias, and parasomnias may be comorbid with other sleep disorders. Studies have also linked parasomnias with SDB.^{32,33} Genetics are important, with 60% of patients reporting a first-degree relative who has para-

somnias.³⁴ In children, insufficient sleep is an important trigger; correcting sleep debt alone may be a sufficient intervention.

Sleepwalking (ICD-9 code 307.46) is common between the ages of 3 and 13 years, and most episodes resolve after age 10 years.^{14,35} Usually, episodes last from 5 to 15 min. The morbidity associated with sleepwalking is typically a result of injury in this unresponsive state. Confusional arousals (ICD-9 code 327.41) include movements usually with vocalization, including uncontrollable crying in infants. Sleep terrors are less common, but more extreme and distressing for parents. A loud scream may initiate the event. The child will appear tremulous with diaphoresis and an expression of extreme terror on their face. Again, these are usually brief, lasting 5 to 15 min. If any recollection of the event is present, it is usually fragmented.

The differential diagnosis for NREM parasomnias includes nightmares, seizures, nocturnal panic attacks, and PTSD. Evaluation requires special emphasis on a detailed description of the nocturnal episodes including the following: timing; presence of stereotypic movements; odd postures or rhythmic behaviors; response to intervention; recall of the event; daytime occurrences; and enuresis. Overnight polysomnography is usually not a routine part of the evaluation unless there is concern about an underlying sleep disorder like SDB or atypical features (*ie*, repetitive, stereotypic behaviors, odd posturing, and timing associated with transition to sleep) that raise the possibility of a seizure disorder. When nocturnal seizures are suspected, the evaluation should include a baseline clinical EEG and consultation with a neurologist.

The management of NREM parasomnias involves parental education as to the benign nature of these phenomena and the importance of adequate sleep. Modifiable triggers such as hot baths and late night exercise should be avoided, and comorbid sleep disorders that fragment sleep such as obstructive sleep apnea should be treated in children who are prone to problem parasomnias. Pharmacotherapy is available; however, caution is advised using medications for what is typically a self-limited problem. Clonazepam is the most widely used medication in the treatment of these disorders^{36,37}; however, well-controlled studies are lacking.

SCHOOL-AGED CHILDREN (6 TO 12 YEARS)

Middle childhood is a critical time for the development of positive sleep habits. Developmental issues that affect sleep in school-aged children are increased understanding of real-life dangers (*ie*, vio-

lent crime), less parental supervision, and more responsibility for health behaviors.⁵ Involvement in academic, social, and athletic activities or parent work schedules may conflict with sufficient time for sleep. Media and electronics (*eg*, television, the Internet, video games, or cellular phones) all compete against sleep time. In fact, the presence of a television in the child's bedroom has been associated with difficulties falling and staying asleep.³⁸ Although sleep problems were traditionally thought to be rare in children in this age group, a new report¹ indicates an overall prevalence of 37% for parent-reported sleep problems, including bedtime resistance (15 to 25%), sleep-onset delay (10%), and daytime sleepiness (10%).¹ Additionally, children may begin to demonstrate a circadian preference for a delayed (later) sleep phase that may cause bedtime struggles.

Inadequate Sleep Hygiene and Insufficient Sleep

Sleep hygiene denotes the variety of behaviors and conditions that promote the amount and quality of sleep. Children and teens with inadequate sleep (ICSD-2 code V69.4) may present with excessive daytime sleepiness, fatigue, difficulties with concentration, and complaints of negative mood. An assessment of the features of healthy sleep habits (Table 3) and a daily sleep diary may help to confirm inadequate sleep hygiene or insufficient sleep. The first step in treating inadequate sleep hygiene is parent and child education at developmentally appropriate levels. The importance of adequate sleep time and consistent sleep routines should be emphasized, potential outcomes of sleep deprivation should be discussed, and a specific sleep plan should be developed.

Table 3—Target Areas for Improving Sleep Habits

Temperature, lighting, and noise level of bedroom (it should be cool, dark, and quiet)
Bedroom should be conducive to sleeping (Does the child share a room? Share a bed? Is anyone else in the room waking the child up at night? Is the room safe?)
Avoid caffeine in late afternoon or evening
Exercise (good for daytime, but avoid late evening exercise before bed)
Academic, social, or vocational demands (minimize interference with sleep schedule)
Restrict bedroom electronics (<i>eg</i> , television, computer, cellular phone, or text messaging) at bedtime
What is the child thinking about when trying to fall asleep? Specific worries?
Is there a consistent bedtime routine? Is the child going to bed and waking up at about the same time each day?
Put infants/children in their crib/bed sleepy but not asleep, so they can learn to fall asleep independently

Sleep-Related Bruxism

Sleep-related bruxism (ICD-9 code 780.58) is the repetitive, audible grinding and clenching of teeth during sleep, a behavior that is a problematic in approximately 5% of the population.^{5,39} Approximately 50% of infants grind their teeth with the eruption of deciduous incisors, which is a behavior that is thought to be clinically insignificant. Adult-type bruxism usually begins in late childhood or early adolescence. The risk factors or conditions that exacerbate bruxism include anxiety, stress, malocclusion, allergies, cerebral palsy/mental retardation, alcohol and stimulant medications use, treatment with serotonin reuptake inhibitors, and primary sleep disturbances.⁴⁰ Associated problems include muscle pain, limited jaw opening, temporomandibular joint problems, headache, neck/shoulder pain, sensitive teeth, daytime bruxism, and dental damage. Although bruxism is usually self-limiting, treatment for more chronic symptoms may include dental appliances for children with dental damage, pain relief, and stress management.

Restless Leg Syndrome and Periodic Limb Movement Disorder

Symptoms of restless leg syndrome (RLS) [ICD-9 code 333.99], as well as the related problem periodic limb movement disorder (PLMD) [ICD-9 code 327.51] have been increasingly recognized in childhood with a prevalence of 2% for RLS.⁴¹ RLS is a common condition in adults that is likely underdiagnosed in children, given that 38% of adults have reported the onset of symptoms prior to age 20 years, and 10% prior to age 10 years.⁴² The sleep disturbance associated with this disorder can manifest as difficulty falling asleep, bedtime resistance, or night wakings, and symptoms similar to those of ADHD.⁴³⁻⁵¹ Some children in whom "growing pains" are diagnosed actually meet the diagnostic criteria for RLS, and a family history of RLS is common in these children.^{50,52} Symptoms of RLS and PLMD can range from mild to severe and can negatively impact on a child's quality of life.

The RLS diagnostic criteria for adolescents (*ie*, ≥ 13 years of age) are the same as those used in adults,⁵³ as follows: (1) an urge to move the legs; (2) the urge to move begins or worsens with sitting or lying down; (3) the urge to move is partially or totally relieved by movement; and (4) the urge to move is worse in the evening or night than during the day, or it occurs exclusively in the evening or nighttime hours. Definite RLS in a child < 13 years of age meets all of the four basic adult criteria and includes the child's description of the leg discomfort in his or her own words. If the child meets the first four criteria but cannot articulate the leg discomfort, then

two of the following criteria will support that diagnosis: clinical sleep disturbance; biological parent or sibling with definite RLS; or a sleep study that shows a periodic limb movement index of greater than five movements per hour. The diagnosis of RLS can be challenging because children have difficulty articulating the symptoms of paresthesia or dysesthesia. “Bugs,” “spiders crawling,” and “energy in my legs” are typical descriptions from a child. Furthermore, the sleep disturbance may seem ordinary for age, overshadowing and preceding for years the complaint of leg discomfort.⁵¹ A family history in a first-degree relative is also important because an autosomal-dominant pattern of inheritance is common when RLS begins at a young age.^{54–56}

Although a periodic limb movement index of more than five movements per hour exceeds the normative data for age and can support a diagnosis of RLS, periodic limb movements are not specific to RLS. They occur in patients with other sleep disorders and can be induced or aggravated by certain medications, particularly the selective serotonin reuptake inhibitor-type antidepressants.⁵⁷ A diagnosis of PLMD is based on the following three criteria: (1) periodic limb movements exceed norms for age (more than five movements per hour for children); (2) clinical sleep disturbance; and (3) the absence of another primary sleep disorder or underlying cause (including RLS).⁷ In children, the diagnosis of PLMD can evolve over time to a diagnosis of “RLS with periodic limb movements” as the typical sensations of RLS develop.

Current research suggests that periodic limb movements are possibly due to an underactive dopaminergic function in specific brain pathways, and are a marker of the instability of the sleep system.⁵⁸ While the cause of primary RLS has not been clearly determined, the roles of genetics, dopamine, and iron appear to be particularly important in the pathophysiology.^{51,59,60} Although the association between low iron stores and RLS/PLMD in children is more recent,^{51,59,61} the relationship between iron status and neural functioning and cognitive development via altered dopamine metabolism is well-known.^{62–64} The assessment of serum ferritin levels is appropriate as these children can benefit from iron therapy when values are $< 35 \mu\text{g/L}$ since iron deficiency is common in the age range of 10 to 35 years.^{65,66} Nonpharmacologic interventions (*eg*, good sleep habits, and the avoidance of caffeine, tobacco and alcohol) should always be part of any treatment plan.

Medication should be considered when nonpharmacologic interventions are inadequate and RLS-related sleep disturbance impacts on daytime functioning. Large, well-controlled studies of pharmacotherapy

for RLS in children are lacking, so the potential risks of medication must be balanced against the long-term consequences of the sleep disturbances. Pharmacologic options include off-label uses of clonidine, clonazepam, clonidine, and gabapentin, and dopamine receptor agonists. Clonidine, the most common prescription drug to facilitate sleep onset in children with a variety of disorders,^{31,67,68} may be beneficial when RLS causes severe sleep-onset problems but can also induce or aggravate sleep terrors. Clonazepam, which has been used to manage severe sleepwalking in children,^{36,37} may improve sleep quality and decrease RLS discomfort, but some children can experience daytime “hang-over” because of the long half-life (18 to 50 h) or even paradoxical activation. Another therapeutic option is gabapentin, which reduces the sensory and motor symptoms of RLS, and appears to improve sleep quality.^{69,70} Other hypnotic agents that are commonly used in adults to facilitate sleep onset may be considered, but pediatric experience is lacking. Medications that increase dopamine, suppress RLS sensations, and reduce periodic limb movements are the drugs of choice for most adults with significant RLS,⁷¹ but evidence on safety and efficacy in children is lacking. Anecdotal reports^{72,73} have suggested that dopaminergic agents may be of benefit in children with RLS and comorbid ADHD. While these drugs could be considered in children and teenagers with severe RLS-related discomfort, caution is required.

ADOLESCENTS (12 TO 18 YEARS)

Data have suggested that chronic partial sleep deprivation is a serious problem in teenagers, with a prevalence of at least 20%.⁷⁴ Only 15% of teenagers report that they get the recommended amount of sleep regularly (Table 1). Parents report that $> 75\%$ of teenagers between the ages of 13 and 18 years go to bed at 11:00 PM or later on school nights.⁷⁵ Chronic sleep deprivation has significant daytime consequences, including a negative impact on mood, reaction time, attention, memory, behavioral control, and motivation. This may result in declines in school/work performance, the use of alertness-promoting agents such as caffeine and stimulant medications, and increased risk-taking behaviors. School, homework, jobs, and social activities interfere with sleep in adolescents, frequently resulting in delayed sleep onset, variable sleep-wake patterns (especially from weekday to weekend), and early waking.

Delayed Sleep Phase Syndrome

Delayed sleep phase syndrome (DSPS) [ICD-9 code 327.31], the most common sleep disorder in adolescence (estimated prevalence, 10%), is a circa-

dian rhythm disorder that is characterized by habitual sleep-wake times that are delayed, usually more than 2 h, relative to conventional clock times.^{7,76} A typical teenager has difficulty initialing sleep and prefers late wake-up times, but once sleep begins, sleep is reported to be normal. Although a preference for staying up late may be a normal consequence of adolescence, staying up until 12:00 AM or later has been shown to be a strong risk factor for the development of DSPS.⁵ Poor sleep hygiene may also be related to later bedtimes, and thus to DSPS. Patients often present with excessive daytime sleepiness, academic problems, truancy, and/or mood disruptions. While the teenager's schedule is usually the focus of the diagnostic interview, a chaotic family sleep-wake schedule can contribute to the problem. A regular sleep diary and comprehensive sleep history may also help to clarify the differential diagnoses such as depression, anxiety, and substance abuse.

Treatment focuses on aligning the teenager's natural sleep rhythm with a sleep-wake schedule that promotes school attendance and family functioning. The management of DSPS begins with regularizing the teenager's sleep-wake schedule, limiting daytime napping, minimizing night-to-night and weekday-to-weekend variability, eliminating caffeinated beverages, minimizing the use of bedroom electronics (*eg*, television, computers with Internet access, and text messaging), and increasing morning sunlight. If these nonpharmacologic measures are insufficient, the currently accepted treatment strategies include chronotherapy, early-morning bright light, and evening oral melatonin therapy. Chronotherapy involves delaying sleep onset for approximately 3 h every day until sleep occurs at the desired sleep time.⁷⁷ While effective, the need for parent supervision, a controlled environment, and the length of treatment decrease the practicality of chronotherapy. Light affects circadian timing in a phase-dependent manner; that is, delays in phase occur when light is administered before the body temperature nadir, and advances in phase occur when light is given after the temperature nadir.⁷⁸ This nadir typically occurs 2 h before waking, so bright light administered for 1 to 2 h in the early morning together with light restriction in the evening, has also been shown to be effective.⁷⁹ However, the feasibility of this somewhat lengthy daily treatment may be limited in a population that is already having trouble getting to school on time.

Because of the practical limitations of both light and chronotherapy, melatonin taken orally in the evening has been increasingly investigated as a potential treatment for DSPS. Melatonin also affects circadian timing in a phase-dependent manner; that is, advances in phase occur when melatonin is ad-

ministered in the evening, and delays in phase occur when melatonin is given in the morning.⁸⁰ In patients with DSPS, melatonin given in the evening has been shown to advance sleep, temperature, and melatonin rhythms; to decrease sleep latency; and to improve quality of life.^{81–87} Melatonin is a weak hypnotic. When used as a phase shifter, a recent study⁸⁸ of melatonin administration in young adults with DSPS suggested that maximal phase shifts occurred when melatonin administration began 6.5 h (compared with 1.5 h) before measured dim light melatonin onset, which is time-linked to a fall in body temperature.

Narcolepsy

Narcolepsy (without cataplexy, ICD-9 code 347.00; with cataplexy, ICD-9 code 347.01) is a chronic neurologic disorder that is characterized by excessive daytime sleepiness, cataplexy, hypnagogic hallucinations, sleep paralysis, and sleep fragmentation,⁷ affecting 1 in 2,000 individuals.⁸⁹ The pathophysiology of idiopathic narcolepsy-cataplexy is linked to hypocretin ligand deficiency in the brain and cerebrospinal fluid and positivity of the human leukocyte antigen DR2/DQ6 (DQB1*0602).^{90,91} Over half of patients recall onset of their symptoms before age 20 years, including one third of adult patients who recall onset before age 15 years, and 16% before age 10 years.^{92,93} However, narcolepsy is diagnosed in only 4% of patients before age 15 years,⁹² indicating a knowledge gap among physicians. An estimated 10% of patients with narcolepsy have a first-degree relative with the disorder.⁹⁴ Although the presenting symptoms are nearly the same as those in adults, especially for children and adolescents, the symptoms are frequently misdiagnosed as neurologic, psychiatric, or behavioral.^{95–97} Narcolepsy may be overlooked when children are misdiagnosed with ADHD and treated with a stimulant medication. The differential diagnosis includes chronic sleep deprivation, idiopathic hypersomnia, circadian rhythm disturbance, prolonged sleep need, unrecognized sleep disrupters, Kleine-Levin syndrome, psychiatric disorders (*eg*, depression, oppositional disorder, ADHD, and psychosis), and substance abuse. In children, the causes of narcolepsy secondary to a medical condition include Prader-Willi syndrome, CNS tumors, and CNS trauma.^{90,91,98–100} Evaluation includes polysomnography and multiple sleep latency tests (MSLTs). The interpretation of MSLT findings in children can be challenging.¹⁰¹ First, the characteristic finding of two sleep-onset REM periods may not be present early in the disorder, so repeated sleep studies may be required. Second, sleep latency in a child with

suspected narcolepsy cannot be assessed using normative adult data. For example, the mean (\pm SD) normal values for mean sleep latency on a four-nap MSLT are much longer in the prepubertal child (19 ± 1.6 min)¹⁰² than in the adult (10.4 ± 4.3 min).¹⁰³

Narcolepsy appears to be a lifelong condition but is not progressive. However, the natural history of narcolepsy is poorly defined, and rigorous longitudinal outcome studies are lacking. The first line of therapy for children with narcolepsy is wake-promoting stimulant medication including methylphenidate, mixed amphetamine salts, and modafinil.¹⁰⁴ Long-acting preparations are particularly useful for children attending school. Schedule II stimulant medications are controlled substances requiring monthly prescriptions, and schedule IV medications require new prescriptions every 6 months in most states. It is important to inform the child's school and teachers about the diagnosis in order to reduce the potential misperception that the child is lazy, bored, or underachieving. Individualized educational plans may be needed. Educating the child's peers about narcolepsy may also reduce the stigma associated with such symptoms as cataplexy. Other nonpharmacologic strategies such as exercise and scheduled daytime naps may help to reduce daytime sleepiness, and should be included in individualized education plans. Any pharmacologic treatment must be closely monitored for safety and efficacy, and patients should be periodically assessed for changes in symptoms.

Insomnia

The features associated with adolescent insomnia (ICSD-2 code 307.42; due to a medical condition, ICSD-2 code 327.01) include changes in mood, daytime fatigue, poor school performance, cognitive impairment, excessive caffeine use, and hypnotic use. In a large, European population-based study¹⁰⁵ of teenagers 15 to 18 years of age, 25% reported insomnia symptoms and 4% met the criteria for a *Diagnostic and Statistical Manual of Mental Disorders-IV* insomnia disorder. A newer US study¹⁰⁶ of teenagers 13 to 16 years old puts the prevalence at 10.7%, with more than one half having a comorbid psychiatric diagnosis. Insomnia likely results from a combination of predisposing factors (*eg*, genetic vulnerability, underlying medical or psychiatric conditions, or sleep disorders), precipitating factors (*eg*, acute stress), and perpetuating factors (*eg*, poor sleep habits, caffeine use, and unhelpful cognitions). Insomnia can also be part of learned sleep-preventing associations and heightened physiologic arousal.

The differential diagnosis includes transient in-

somnia, RLS/PLMD, obstructive sleep apnea, DSPS, inadequate sleep hygiene, psychiatric disorders, and medical factors. Sleep diaries may be used to identify potential maladaptive bedtime activities, behaviors, or schedules. Polysomnography is indicated when another sleep disorder is suspected. Management includes a thorough evaluation of the causes and contributing factors, maintenance of appropriate sleep hygiene, and screening for psychiatric disorders. Treatment options include behavioral interventions (*ie*, relaxation) and the consideration of hypnotic medication to interrupt the cycle of insomnia; however, controlled trials and pediatric labeling for hypnotic agents are lacking.

CONCLUSIONS

Pediatric nonrespiratory sleep problems are common, underdiagnosed, and treatable. Because of the impact of sleep on children's physical, psychological, academic, and overall functioning, the impact of an increased recognition, evaluation, and management of pediatric sleep disorders will likely have a significant impact on the general health and well-being of children. Since children referred for sleep medicine evaluation frequently have other comorbid medical, psychiatric, or behavioral comorbidities, a multidisciplinary approach to clinical assessment and coordination of care at a specialty level (*eg*, ear-nose-throat, behavioral psychology, neurology, and psychiatry) and close collaboration with pediatric specialists, when available, will optimize the quality of sleep medicine services for children.

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