Common Sleep Disorders in Children

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Up to 50% of children will experience a sleep problem. Early identification of sleep problems may prevent negative consequences, such as daytime sleepiness, irritability, behavioral problems, learning difficulties, motor vehicle crashes in teenagers, and poor academic performance. Obstructive sleep apnea occurs in 1% to 5% of children. Polysomnography is needed to diagnose the condition because it may not be detected through history and physical examination alone. Adenotonsillectomy is the primary treatment for most children with obstructive sleep apnea. Parasomnias are common in childhood; sleepwalking, sleep talking, confusional arousals, and sleep terrors tend to occur in the first half of the night, whereas nightmares are more common in the second half of the night. Only 4% of parasomnias will persist past adolescence; thus, the best management is parental reassurance and proper safety measures. Behavioral insomnia of childhood is common and is characterized by a learned inability to fall and/or stay asleep. Management begins with consistent implementation of good sleep hygiene practices, and, in some cases, use of extinction techniques may be appropriate. Delayed sleep phase disorder is most common in adolescence, presenting as difficulty falling asleep and awakening at socially acceptable times. Treatment involves good sleep hygiene and a consistent sleep-wake schedule, with nighttime melatonin and/or morning bright light therapy as needed. Diagnosing restless legs syndrome in children can be difficult; management focuses on trigger avoidance and treatment of iron deficiency, if present. (***Am Fam Physician*****. 2014;89(5):368-377. Copyright © 2014 American Academy of Family Physicians.)**

CME This clinical content conforms to AAFP criteria for continuing medical education (CME). See CME Quiz Questions on page 327.

Author disclosure: No relevant financial affiliations.

Sleep is one of the most commonly discussed topics during well-child visits.[1] It is important for primary care physicians to be familiar with normal childhood sleep patterns and common sleep disorders. Epidemiologic studies indicate that up to 50% of children experience a sleep problem,[2-4] and about 4% have a formal sleep disorder diagnosis.[5]

Normal Sleep in Infants and Children

Sleep is an opportunity for the body to conserve energy, restore its normal processes, promote physical growth, and support mental development. The most recognized consequence of inadequate sleep is daytime sleepiness. However, sleepiness in children commonly manifests as irritability, behavioral problems, learning difficulties, motor vehicle crashes in teenagers, and poor academic performance.[6-8] Distinguishing significant sleep disruptions from normal age-related changes can be challenging and can ultimately delay treatment.

Sleep changes considerably during the first few years of life and parallels physical maturation and development. Newborns require the greatest total sleep time and have a fragmented sleep-wake pattern. Starting at five months of age, infants have the ability to sleep for longer periods. At six months of age, children are able to go without nighttime feedings, but significant variation exists. Additionally, breastfeeding infants have more frequent awakenings, shorter sleep periods, and slightly shorter total sleep times.[9] As children age, sleep periods gradually lengthen and total sleep time decreases *(Figure 1)*.

The large variation in sleep behavior among children may be secondary to cultural or genetic differences; however, there are some general trends *(Table 1)*.[10,11] Ultimately, knowing the normal developmental stages of sleep will help differentiate between normal sleep and common sleep disorders, such as obstructive sleep apnea (OSA), parasomnias, behavioral insomnia of childhood, delayed sleep phase disorder, and restless legs syndrome. These disorders are summarized in *Table 2*.[7,12-50]

Obstructive Sleep Apnea

OSA is characterized by upper airway obstruction, despite respiratory effort, that disrupts normal sleep patterns and ventilation.[12] OSA can be associated with obesity,

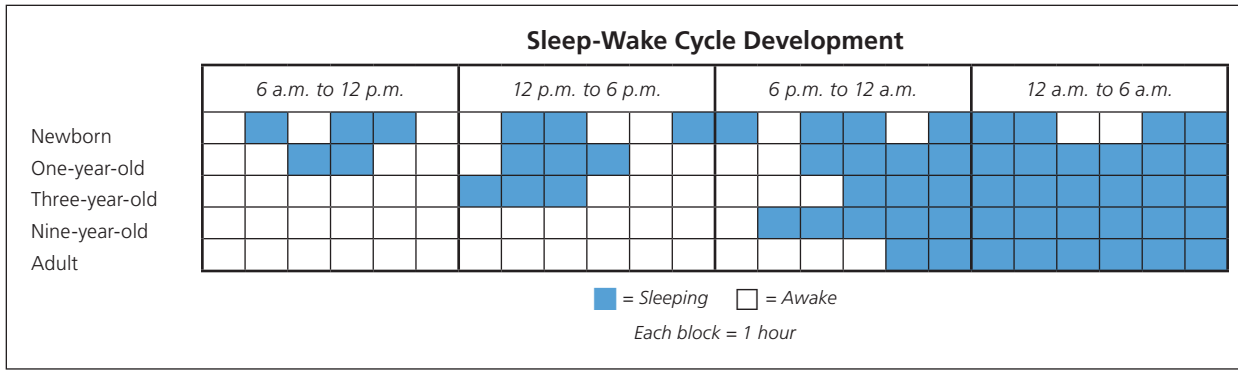

Figure 1. Generalization of sleep-wake cycle patterns at different stages during sleep pattern development. Newborns have a fragmented sleep-wake pattern with no significant sleep consolidation. Over time, daytime napping decreases, and nighttime sleep consolidation occurs.

excessive soft tissue in the upper airway, decreased upper airway lumen size, or failure of pharyngeal dilator muscles. However, in children, the obstruction is primarily due to enlarged tonsils and adenoids.[12,13] Onset usually occurs between two and eight years of age, coinciding with peak tonsil growth, but the condition can manifest at any age.[51] The overall prevalence in children is 1% to 5%.[13] It occurs equally among males and females, but is more common in ethnic minorities.[13]

Snoring and witnessed apneas are the classic symptoms of OSA, but not all snorers have the condition. The prevalence of habitual snoring in children is as high as 27%, which can complicate the recognition of OSA.[13,16,52] Other common symptoms include unusual sleeping positions (e.g., hyperextended neck, seated with open mouth), sleep-related paradoxical breathing, nighttime diaphoresis or enuresis, morning headaches, and excessive daytime sleepiness. However, children are less likely than adults to present with daytime sleepiness. Sleepiness in children is more likely to manifest as depressed mood, poor concentration, decreased attention, or behavioral issues.[6,52,53]

Weight and body mass index are usually normal in children with OSA; however, the incidence of obesity-related sleep apnea is steadily increasing.[5,54] Physical examination findings can include enlarged tonsils, micrognathia, and pectus excavatum. However, subjective grading of tonsil size in children does not always correlate with objective findings.[55]

Results of the history and physical examination alone correlate poorly with objective findings of OSA, and questionnaires have shown a sensitivity of only 78%.[13] Therefore, children with suspected OSA should be referred for polysomnography.[17-19] In addition, referral to a sleep medicine specialist should be considered for those with high-risk features (e.g., attention-deficit/hyperactivity disorder, cardiorespiratory failure, craniofacial abnormalities, congenital defects, Down syndrome).

Untreated OSA is associated with neurobehavioral problems, decreased attention, disturbed emotional regulation, decreased academic performance, nighttime enuresis, impaired growth, and, rarely, systemic hypertension, pulmonary hypertension, and cor pulmonale.[6,17-20,52,53] Adenotonsillectomy is the primary treatment for OSA in children *(Table 3)*.[13,18] Following adenotonsillectomy, postoperative polysomnography demonstrates resolution of OSA in more than 70% of

Table 1. Summary of Normal Sleep Parameters in Children

Age	*Total sleep time*	*Naps (on average)*
0 to 2 months	16 to 18 hours	3.5 per day at 1 month of age
2 to 12 months	12 to 16 hours Most children 6 to 9 months of age sleep through the night	2 per day at 12 months of age
1 to 3 years	10 to 16 hours	1 per day at 18 months of age
3 to 5 years	11 to 15 hours	50% of 3-year-olds do not nap
5 to 14 years	9 to 13 hours	5% of whites and 39% of blacks nap at 8 years of age
14 to 18 years	7 to 10 hours	Napping in this age group suggests insufficient sleep or a possible sleep disorder

Information from references 10 and 11.

Table 2. Summary of Common Sleep Disorders in Children

Sleep disorder	*Epidemiology*	*Clinical features*
Obstructive sleep apnea	Prevalence: 1% to 5% Onset between 2 and 8 years of age Affects males and females equally More common in blacks and in persons with craniofacial abnormalities (e.g., micrognathia, retrognathia, midfacial hypoplasia), Down syndrome, neuromuscular diseases, choanal atresia	Snoring Unusual sleep positions (e.g., hyperextended neck, seated with open mouth) Sleep-related paradoxical breathing Nighttime enuresis or diaphoresis Morning headaches Cognitive/behavioral issues (e.g., depressed mood, poor concentration, decreased attention) Excessive daytime sleepiness (less common) Enlarged tonsils and adenoids Pectus excavatum
Parasomnias		
Sleepwalking (somnambulism)	Prevalence: 17% in children, 4% in adults Peaks between 8 and 12 years of age More common in males Familial history of sleepwalking: 1 parent = 45% risk 2 parents = 60% risk	Ambulation during sleep Difficult to awaken during episode Eyes open Confusion/agitation Unusual or dangerous behaviors Rapid return to sleep Usually occurs during the first half of the sleep period, with no memory of the event Patient may have coexisting confusional arousals and/or sleep terrors
Confusional arousals	Prevalence: 17.3% in 3- to 13-year-olds, 2.9% to 4.2% in those older than 15 years Affects males and females equally Strong familial pattern	"Sleep drunkenness" Inappropriate behavior Slowed responsiveness Slurred speech Confusion after awakening (forced or spontaneous) Usually occurs during the first half of the sleep period, with no memory of the event
Sleep terrors	Prevalence: 1% to 6.5% in children, 2.2% in adults Onset in early childhood Affects males and females equally	Perceived intense fear (e.g., screaming, crying, confusion, walking) Difficult to awaken from episode Potentially dangerous activities Usually occurs during the first half of the sleep period, with no memory of the event Considerable overlap with other parasomnias
Nightmares	Prevalence: 10% to 50% in 3- to 5-year-olds Onset between 3 and 6 years of age, peaks between 6 and 10 years of age Affects males and females equally	Unpleasant dreams Increased sympathetic response (increased heart and respiratory rates, diaphoresis) Usually occurs during the second half of the sleep period, with clear memory of the event Reluctance to sleep increases May be associated with mood disorders or posttraumatic stress disorder

PSG = polysomnography.

normal-weight children, but in less than one-half of obese children.[13,56-58] Postoperative improvements in quality of life and behavior may also occur.[56-59] Children being considered for adenotonsillectomy who are at high risk of postoperative complications (risk factors include age younger than three years, severe OSA, obesity, current respiratory infection, craniofacial abnormalities, failure to thrive, cardiac complications of OSA, and neuromuscular disorders) should undergo the procedure as an inpatient. Once treated, all children should have a clinical assessment six to eight weeks postoperatively, and polysomnography should be repeated to assess for

Diagnostic criteria	*Treatment options*
PSG is required for diagnosis (apnea-hypopnea index greater than 1.5 per hour)	First line: Adenotonsillectomy Other: Continuous positive airway pressure, nasal steroids, rapid maxillary expansion (i.e., orthodontic device widens the upper jaw)
Diagnosed by history PSG not required	Reassurance (usually resolves spontaneously) Increase total sleep time Scheduled awakenings Bedroom/home safety counseling Screening for precipitating factors (e.g., sleep deprivation, obstructive sleep apnea, gastroesophageal reflux disease, acute stress, medication or illicit drug use) Benzodiazepines
Diagnosed by history PSG not required	Reassurance (usually resolves spontaneously) Increase total sleep time Scheduled awakenings Bedroom/home safety counseling
Diagnosed by history PSG not required	Reassurance (usually resolves spontaneously) Increase total sleep time Scheduled awakenings Bedroom/home safety counseling Benzodiazepines
Diagnosed by history PSG not required	Reassurance (usually resolves spontaneously) Increase total sleep time Scheduled awakenings Bedroom/home safety counseling Cognitive behavior therapy Medications that suppress rapid eye movement sleep (selective serotonin reuptake inhibitors; off-label use)

continued

residual OSA in those with obesity, moderate to severe OSA on initial testing, or persistent symptoms.[13,17,18] If ordered, polysomnography should be performed after the pharynx has fully healed, usually no earlier than six weeks postoperatively.

Weight loss interventions have demonstrated benefits of reducing the severity of OSA and should be initiated in all children who are overweight or obese.[13] Continuous positive airway pressure should be offered to those with residual OSA symptoms or if adenotonsillectomy was not performed. There is limited evidence to support the use of intranasal corticosteroids for children with mild

Table 2. Summary of Common Sleep Disorders in Children *(continued)*

Sleep disorder	*Epidemiology*	*Clinical features*
Behavioral insomnia of childhood	Prevalence: 10% to 30% Affects males and females equally	Sleep-onset association type: Difficulty initiating or maintaining sleep when sleep-specific conditions are not present (e.g., a parent rocking the child to sleep) Frequent nighttime awakenings common Falling asleep is a timely process, demanding for parents Symptoms represent a disorder only when they are persistent and highly demanding, or cause significant parental distress Limit-setting type: Difficulty initiating or maintaining sleep Bedtime refusal/stalling Refusal to return to sleep after nighttime awakenings Parents fail to set boundaries and give in to the child (e.g., allow the child to sleep in the parents' bed)
Delayed sleep phase disorder	Prevalence: 7% to 16% in adolescents Onset in adolescence, with the peak age in the 20s It is unknown whether it is more common in one sex 40% of those affected have a family history of the condition	Difficulty falling asleep and waking up at socially acceptable times (at least a two-hour delay) "Night owl"
Restless legs syndrome	Prevalence: 2% based on limited studies More common in women; unknown if it is more common in boys or girls Family history: Early onset associated with primary restless legs syndrome (genetic)	Urge to move the legs with associated discomfort Often begins in the evening, worsens with rest, eases with movement May be associated with iron deficiency Associated with negative behavior and mood, and decreased cognition and attention Higher prevalence in those with attention-deficit/hyperactivity disorder

PSG = polysomnography.

Information from references 7, and 12 through 50.

Table 3. Recommendations for the Diagnosis and Management of OSA in Children

Diagnosis

All children should be screened for snoring at well-child visits.[13]

Clinical and polysomnographic findings should be integrated to diagnose OSA; clinical indicators alone are not consistently reliable for predicting OSA.[13,18]

Adenotonsillectomy is a low-risk procedure, but should be performed only in those with proven OSA.[13]

Preoperative polysomnography is indicated before adenotonsillectomy in children with OSA.[13,18]

Identifying the severity of OSA helps determine the risk of postoperative respiratory complications.[13,18]

Treatment

Adenotonsillectomy is the primary treatment for those with adenotonsillar hypertrophy; it is highly effective and leads to improved quality of life and behavior.[13]

Follow-up

Patients with mild OSA should receive postoperative and periodic clinical assessments for residual symptoms; if symptoms are present, postoperative polysomnography is indicated.[13,18]

Patients with moderate to severe OSA and obesity should receive postoperative polysomnography to assess for residual symptoms, as well as periodic clinical assessments[13,18]

OSA = obstructive sleep apnea.

Information from references 13 and 18.

Diagnostic criteria	*Treatment options*
Diagnosed by history PSG not required	Prevention, parental education, and extinction techniques are effective See Table 4
Diagnosed by history Use of sleep diary and/or actigraphy (i.e., an accelerometer that approximates sleep-wake times) for at least 1 week PSG not required	Sleep hygiene education Regular sleep-wake schedule Avoidance of bright lights before bedtime Melatonin, 0.3 to 5 mg given 1.5 to 6.5 hours before desired bedtime Bright light therapy, 2,000 lux for first 1 to 2 hours after awakening Continued use of sleep logs to monitor progress
Diagnosed by history PSG may be indicated When the child is unable to describe the symptoms, a diagnosis can be made if the history is consistent and at least two of the following are present: a sleep disturbance, a first-degree relative has the condition, or five or more periodic limb movements per hour of sleep during PSG	Avoidance of nicotine and caffeine Discontinue offending medications (antihistamines, selective serotonin reuptake inhibitors, and tricyclic antidepressants) Iron replacement if ferritin level is less than 50 mcg per L; recheck in 3 months Severe cases: levodopa, dopamine agonists, gabapentin (Neurontin), opioids, benzodiazepines (all off-label uses)

OSA or with residual mild OSA following adenotonsillectomy.[60] Although rapid maxillary expansion (i.e., use of an orthodontic device that widens the upper jaw) and montelukast (Singulair) are sometimes recommended, no clinical trial evidence supports the use of these treatments for OSA in children.

Parasomnias

Parasomnias such as sleepwalking (somnambulism), sleep talking (somniloquy), confusional arousals, sleep terrors, and nightmares affect up to 50% of children.[12] They are defined as undesirable events that accompany sleep and typically occur during sleep-wake transitions.[12] They are additionally characterized by complex, awake-like activity by the child that appears purposeful but lacks meaningful interaction with his or her environment. Associated features include confusion, automatic behaviors, difficulty awakening, amnesia, and rapid return to full sleep after the event.

Most parasomnias, such as sleepwalking, sleep talking, confusional arousals, and sleep terrors, occur in the first half of the sleep period during slow wave sleep; children typically have no memory of the event. In contrast, nightmares typically occur in the last half of the sleep period during rapid eye movement sleep, with children able to remember the event. It is important to note that the symptoms and timing of nocturnal seizures can overlap with parasomnias. Physicians should inquire about repetitive stereotypic behaviors and odd posturing that could represent nocturnal seizures.[12]

Genetically predisposed individuals are susceptible to precipitating factors, contributing to the development of parasomnias. Precipitating factors include insufficient sleep and disorders causing partial awakenings from sleep. OSA is a common trigger for parasomnias, and a review of studies showed that more than one-half of children referred for sleep terrors or sleepwalking also had OSA.[21] Other triggers may include periodic limb movement disorder, gastroesophageal reflux disease, forced awakenings, and certain medications.[12,21]

Parasomnias often resolve spontaneously by adolescence; however, 4% of persons will have recurring events.[12,22] Treatment centers on reassurance, reducing precipitating factors, and increasing total sleep times.[21,23,61] When it is appropriate, parents should be counseled about safety measures (e.g., locking doors and windows, using motion alarms, clearing the floor of toys, placing the mattress on the floor). Children who exhibit atypical, harmful, or violent behaviors or who are unresponsive to conservative treatments should be referred for further evaluation.

Table 4. Treatments for Behavioral Insomnia of Childhood

Treatment technique	*Description*
Parental education	Parents are taught about good sleep practices, such as consistent feedings, nap times, bedtime routines, regular sleep-wake times, and placing the child in bed drowsy but awake.
Unmodified extinction	The child is placed in bed at a predetermined bedtime.
	The child's crying, calls for the parents, and tantrums are ignored until the following morning, although significant cries for suspected injuries or illnesses are not ignored.
	Cries are ignored to prevent reinforcing negative learned behavior (e.g., crying is rewarded with parental response/presence).
	This technique can be difficult and distressing for parents.
	Modified version for decreased parental distress: A parent stays in the child's room, but follows the same technique.
Graduated extinction	This is fundamentally the same as unmodified extinction, but with scheduled "check-ins."
	A parent checks on the child on a fixed schedule (e.g., every 10 minutes) or in gradually increased intervals (e.g., first check-in after five minutes, second check-in after 10 minutes).
	Parental interactions with the child are calming and positive, but last no more than one minute at a time.
Positive bedtime routines/faded bedtime with response cost	Positive bedtime routines: Relaxing/calming activities are implemented before bedtime (e.g., bedtime stories).
	Faded bedtime: Bedtime is delayed until the predicted time of sleep onset to decrease the time the child spends in bed awake.
	Response cost: The child is removed from bed for a specific amount of time if sleep onset does not occur within the desired period.
Scheduled awakenings	Parents must document the pattern of nighttime awakenings.
	The child is awakened before the normally predicted nighttime awakening, and the number of scheduled awakenings is slowly decreased over time.

Adapted with permission from Mindell JA, Kuhn B, Lewin DS, Meltzer LJ, Sadeh A; American Academy of Sleep Medicine. Behavioral treatment of bedtime problems and night wakings in infants and young children [published correction appears in Sleep. *2006;29(11):1380].* Sleep. *2006;29(10):1279.*

Behavioral Insomnia of Childhood

Behavioral insomnia of childhood is characterized by a learned inability to fall and/or stay asleep; the estimated prevalence is 10% to 30%.[12,24] The condition is divided into the sleep-onset association type and the limit-setting type. The sleep-onset association type is characterized by the child's inability or unwillingness to fall asleep or return to sleep in the absence of specific conditions, such as a parent rocking the child to sleep.[12] The limit-setting type occurs when parents fail to set appropriate limits, such as when the parents allow the child to sleep in their bed when the child refuses to sleep.[12] Most children with behavioral insomnia of childhood have features of both types.

Prevention is the best treatment for behavioral insomnia of childhood. Physicians should educate parents on normal sleep patterns, good sleep hygiene, realistic expectations, setting boundaries, and sleep plans. These plans should focus on regular and consistent feedings, nap times, bedtime routines, and sleep-wake times. Infants are more likely to become self-soothers (fall asleep on their own) when consistently placed in the crib awake vs. already asleep.[62,63] Creating a regular routine will establish expectations, and the child will eventually learn how to fall asleep on his or her own. Extinction techniques (placing the child in bed and ignoring him or her until the morning, or for a set period) are effective in the treatment of this disorder.[25-28] There are various extinction techniques, and no single method is superior. Techniques for managing behavioral insomnia of childhood are summarized in *Table 4*.[27]

Sleep or sedating medications are ineffective for the treatment of this disorder.[29,30]

SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	*Evidence rating*	*References*
Sleep disorders should be considered in children presenting with irritability, behavioral problems, learning difficulties, and poor academic performance.	C	6-8, 52, 53
Adenotonsillectomy is the primary treatment for children with obstructive sleep apnea.	B	13, 56-58
Sleep or sedating medications have no role in the treatment of behavioral insomnia of childhood.	C	29, 30
If restless legs syndrome is suspected in a child, management should include a workup for iron deficiency and avoidance of triggers.	C	47-50

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to http://www.aafp.org/afpsort.

Delayed Sleep Phase Disorder

The master circadian clock, located within the suprachiasmatic nucleus, controls the timing of sleep and cycles approximately every 24 hours in most individuals.[31] The discrepancy between the internal clock and the external world requires continuous "resetting" by time cues, such as light, melatonin, physical activity, body temperature, and meals. Light is the most powerful of these entrainers. Inappropriate timing of light exposure can alter the circadian rhythm. For example, light exposure before bedtime can suppress melatonin and ultimately delay sleep onset.[31,32]

In children with delayed sleep phase disorder, habitual sleep-wake times are delayed by at least two hours compared with socially acceptable times.[12] The disorder is more common during adolescence when the circadian rhythm is thought to lengthen and the child becomes more social.[31,64] The prevalence in adolescents is 7% to 16%.[12,31,34] The disorder is diagnosed using patient history and documentation of sleep and wake times on a sleep diary or log. Parental concerns usually focus on late bedtimes (2 a.m. or later), sleeping in, difficulty awakening, and school tardiness. However, frequent nighttime awakenings are unusual, and sleep architecture is usually normal *(Figure 2)*.

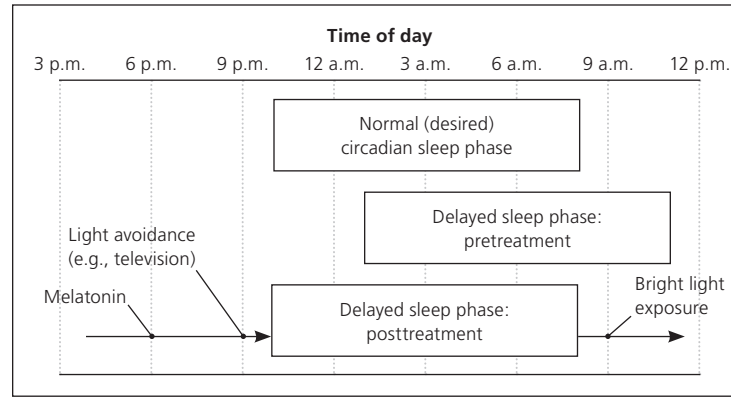

Figure 2. Comparison of the sleep pattern associated with delayed sleep phase disorder (pre- and posttreatment) and that of normal (desired) sleep phase. Treatments such as melatonin, nighttime light avoidance, and early morning bright light therapy will slowly advance the individual's sleep phase to the desired time.

Treatment focuses on aligning the circadian rhythm with desired sleep-wake times. As in all sleep disorders, maintaining a regular sleep-wake cycle and practicing good sleep hygiene are the foundation of treatment. It is important to avoid bright lights before bedtime. Removing all light-emitting devices (e.g., electronics, portable media, tablet computers, cell phones) from the bedroom may be beneficial. Bright light therapy used for the first one to two hours after awakening may also be beneficial and will advance the circadian rhythm.[31] There is strong evidence that melatonin supplementation (0.3 to 5 mg given 1.5 to 6.5 hours before desired bedtime) is an effective treatment for delayed sleep phase disorder, although the exact dose or timing has not been well established.[36-38]

Restless Legs Syndrome

The rate of restless legs syndrome in children is unclear, but limited studies suggest a prevalence of 2%.[12,39] The condition is characterized by an unpleasant sensation in the legs, with the urge to move the legs starting in the evening.[12] Rest worsens symptoms, and movement provides some relief. Other symptoms include difficulty falling asleep, bedtime resistance, "growing pains," and symptoms similar to those of attention-deficit/hyperactivity disorder.[12,40,42,43] The condition in children is associated with negative behavior and mood, and decreased cognition and attention, and it is more common in children with attention-deficit/hyperactivity disorder.[12,39,42]

Dopamine dysfunction, genetics, and iron deficiency are thought to play a role in the pathogenesis of restless legs syndrome.[45-48] Additionally, symptoms may be exacerbated by excessive or inadequate physical activity or the use of caffeine, nicotine, antihistamines, selective serotonin reuptake inhibitors, or tricyclic antidepressants.[47]

Diagnosing restless legs syndrome in children can be challenging because they may be unable to describe the

core symptoms. A diagnosis can be made if the history is consistent with the condition and at least two of the following are present: a sleep disturbance, a first-degree relative has the condition, or five or more periodic limb movements per hour of sleep during polysomnography.[12] Once restless legs syndrome is diagnosed, conservative treatment includes avoiding exacerbating factors.[47] Because iron deficiency is common in children, measuring the ferritin level is reasonable.[49,50] Iron replacement should be initiated if ferritin levels are less than 50 mcg per L, and they should be rechecked in three months.[46,48] There are no medications approved for treating restless legs syndrome in children. Patients with symptoms that do not respond to conservative treatments should be referred for further evaluation.

Data Sources: We searched PubMed using standard search and MESH search terms. The American Academy of Sleep Medicine, American Academy of Pediatrics, American Heart Association, and American Thoracic Society were searched for guidelines and recommendations. In addition, a set of references from Essential Evidence Plus were reviewed and cited as applicable. Search dates: March 2011 through November 2013.

The opinions expressed herein are those of the authors and should not be construed as official or as reflecting the policies of the Department of the Army or the Department of Defense.

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