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Review of Narcolepsy and Other Common Sleep Disorders in Children

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Keywords

• Narcolepsy • Sleep-related breathing disorders • Circadian rhythms disorders • Parasomnias • Restless leg syndrome

Key points

- Common sleep disorders in children include narcolepsy, sleep disordered breathing, circadian rhythm disorders, parasomnias, restless leg syndrome, periodic limb movements in sleep, and periodic limb movement disorder.
- These disorders can have a wide range of effects in children, including severe daytime sleepiness, abnormal behaviors, hyperactivity, lack of concentration, and difficulties in school.
- Ongoing research is helping to clarify the pathophysiology and clinical features of these disorders.
- Early diagnosis can significantly improve quality of life, and there are increasing options for management.

INTRODUCTION

The identification of sleep problems in children and adolescents is increasingly important given the growing evidence indicating that disrupted sleep in children can impair cognitive, emotional, neurobehavioral, and social development

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[1,2]. Sleep is essential in children's learning, memory processes, school performance, and general well-being [3]. The prevalence of sleep problems in children is at least 25% [2,3]. A misdiagnosis of narcolepsy or other sleep disorders has resulted in delayed treatment, causing increased symptom burden. We provide an overview of the common sleep disorders that are seen in children and adolescents.

NARCOLEPSY

Narcolepsy is a complex sleep disorder that can manifest in childhood or adolescence. It is the most common cause of excessive daytime sleepiness and can occur in isolation or in combination with other symptoms such as cataplexy, hypnogogic/hypnopompic hallucinations, disturbed nocturnal sleep, and sleep paralysis [3,4]. Cataplexy distinguishes type 1 (narcolepsy with cataplexy) from type 2 narcolepsy (narcolepsy without cataplexy) [4]. Cataplexy is reported by 60% to 75% of patients with narcolepsy and is defined as a transient, sudden-onset loss of skeletal muscle tone with retained consciousness. It is often in seen in response to a strong emotion (eg, laughter, startle, or anger) [4,5]. Narcolepsy is rare, occurring in 0.025% to 0.050% of the population, with a peak incidence between the ages of 10 and 19 years. The exact prevalence of narcolepsy in children is unknown, but studies indicate that symptoms began during childhood in more than one-half of adult narcolepsy patients [6]. Delays in diagnosis have been reported with a mean delay of up to 15 years [6,7]. Diagnosis in children can be difficult owing to a lack of general awareness of this disorder, atypical presentations of cataplexy, and the associated medical, sleep, and behavioral comorbidities that often lead to misdiagnosis [8]. Narcolepsy can impair social and academic performance in otherwise intellectually normal children [5,7,9], and its implications are often misunderstood by patients, parents, teachers, and health care professionals.

Genetic and environmental factors reportedly play a role in the etiology of narcolepsy [7,10]. Evidence suggests that the loss of hypocretin is highly associated with its development [10]. Hypocretin, a neuropeptide found in the lateral and posterior hypothalamus, targets monoaminergic and cholinergic areas. The loss of hypocretin neurons results in the characteristic symptoms of narcolepsy, including the inability to sustain long periods of wakefulness and frequent lapses into sleep [7,10]. Evidence suggests a link between HLA DQB1*06:02 and narcolepsy, indicating an autoimmune basis for narcolepsy that is mediated by hypocretin neurons [10].

Sleepiness is a normal, cyclic experience that invariably occurs after prolonged wakefulness. In healthy persons, mild sleepiness during the daytime is apparent only during boring, sedentary situations (eg, watching television). Children often lapse into sleep when not stimulated (eg, being driven in a car or during school). The majority of children stop requiring daytime naps by the age of 5 years [11]. Hypnagogic or hypnopompic hallucinations are vivid, dreamlike sensations that an individual feel, hears, or sees and that occur near the onset of sleep (hypnogogic) or upon waking up from sleep

(hypnopompic). Sleep paralysis is defined as the temporary inability to move or speak while being conscious and that occurs when a person is falling asleep or waking up from sleep. These hallucinations and sleep paralysis are less common in children than adults, but can be confused with seizures in children. A lack of stereotypical features distinguishes these events from seizures, and the relationship of sleep with these hallucinations distinguishes them from hallucinations of psychosis. Cataplexy in children with narcolepsy may present as a wide range of motor disturbances that do not meet the classic definition of cataplexy, and can include more complex active movements (eg, perioral movements, dyskinetic–dystonic movements, or stereotypic movements) [8]. These motor disturbances may resolve later in the course of the disorder. Other predominating features of narcolepsy in children are restlessness and motor overactivity. Academic deterioration, inattentiveness, and emotional lability are common in affected children. Patients with narcolepsy tend to take short and refreshing naps (ie, REM-type naps) during the day. Their daytime naps may be accompanied by dreams [5–7].

Many patients with narcolepsy also have trouble sleeping at night, and children with narcolepsy have difficulty with morning arousal, which can be associated with aggressive behavior. Over the past several years, various common comorbidities have been described in children with narcolepsy [9,12], including obesity, accelerated pubertal development, and attention deficit hyperactivity disorder symptoms [3,9,12,13]. These children often were misdiagnosed as having attention deficit hyperactivity disorder, learning disability, depression, or another neurologic disorder.

Diagnosis

The diagnosis of narcolepsy requires a detailed clinical history and diagnostic studies such as an overnight polysomnography (PSG) followed by a multiple sleep latency test (MSLT) [5,7,14,15]. An MSLT includes 4 or 5 nap periods of 20 minutes, each with a 2-hour break between naps. An MSLT with sleep latency of 8 minutes of less and 2 or more sleep-onset REM periods and the exclusion of other sleep disorders on PSG provide a definitive diagnosis of narcolepsy [14,16]. The PSG performed the night before MSLT is important for excluding other primary sleep disorders such as sleep apnea and periodic limb movement disorder. Abnormal MSLT findings are not specific for narcolepsy and may be produced by other sleep disorders, such as sleep apnea, circadian misalignment, other mental or medical conditions, medications or substance use, or sleep deprivation [16]. An alternative criterion for diagnosis is a cerebrospinal fluid hypocretin level of 110 pg/mL or less [4,17].

Treatment

Narcolepsy is treatable; however, a multimodal approach with both nonpharmacologic and pharmacologic components is required for the most favorable outcome. Currently, no US Food and Drug Administration (FDA)-approved pharmacotherapy is available for children; however, the medications used to treat adults have been used off-label in children with positive results.

The primary treatment is stimulant therapy, which aims to improve alertness and functioning. Wake-promoting medications such as modafinil and armodafinil are considered first line therapy for excessive daytime sleepiness and are FDA approved for patients older than17 years. Both methylphenidate and modafinil have been used off label for patients 6- to 15-year-old children and are effective [18]. Stimulants such as methylphenidate and amphetamines such as dextroamphetamine and methamphetamine are commonly prescribed in younger children. The undesirable side effects of stimulant medications include headache, irritability, nervousness, and gastrointestinal complaints. Nocturnal sleep may be impaired, resulting in decreased total sleep time. Sleep hygiene is important. Most patients improve if they maintain a regular sleep schedule, usually 7.5 to 8.0 hours of sleep per night.

Sodium oxybate, a precursor for GABA, is the only treatment for cataplexy that has been approved by the FDA. Although sodium oxybate was approved by the FDA for narcolepsy in patients 18 years old or older, off-label use in younger children reportedly controls the cataplexy and severe hypersomnia [19]. Tricyclic antidepressants (eg, clomipramine), selective serotonin reuptake inhibitors (eg, fluoxetine), serotonin, and norepinephrine reuptake inhibitors (eg, venlafaxine) have all been used to treat cataplexy [20].

Although pharmacologic management is at the forefront, behavioral and lifestyle modifications can play a crucial role in decreasing the impact of the disease. Nonpharmacologic treatments include scheduled brief naps, practicing good sleep hygiene, and regular exercise during the day to improve alertness. People with narcolepsy should also be cautious around moving equipment/machinery and heights. Children should be encouraged to participate in afterschool activities and sports. School personnel should have narcoleptic children refrain from unsupervised activities if they seem to be drowsy. Avoiding foods high in refined sugars may improve daytime sleepiness. Providing emotional and educational support to children with narcolepsy, counseling regarding high-risk behaviors such as drug and alcohol use, and inquiring about depression, family conflict, and other psychosocial problems are important.

Regular follow-up with a sleep physician is recommended for children with narcolepsy to monitor response to treatment, to assess for the presence of comorbid sleep disorders such as obstructive sleep apnea or periodic limb movement disorder, and to assess for other psychosocial issues that often arise. Research into future treatments is focusing on preventing the loss of hypocretin (orexin)-producing neurons by targeting the proposed autoimmune-driven mechanism of narcolepsy [20].

SLEEP DISORDERED BREATHING IN CHILDREN

Sleep disordered breathing (SDB) refers to several different disorders that affect the quality of oxygenation and ventilation during sleep. About 1% to 5% of children have SDB, and some may have more than 1 disorder simultaneously [1,21]. SDB prevalence is much higher in children with certain risk factors,

especially those with underlying genetic syndromes (eg, Down syndrome), craniofacial abnormalities, and neuromuscular disease.

Obstructive sleep apnea syndrome

Obstructive sleep apnea syndrome (OSAS) is characterized by recurrent partial or complete upper airway obstruction that causes fluctuations in oxygenation and sleep fragmentation. OSAS is accompanied by frequent snoring (>3 nights/wk), labored breathing during sleep, gasping for air, observed apneas, restless sleep, enuresis, morning headaches, daytime sleepiness, hyperactivity, and/or concentration/learning problems [4,22]. The children at greatest risk for OSAS are those with adenotonsillar hypertrophy, obesity, craniofacial anomalies (eg, Pierre Robin sequence, Crouzon and Treacher Collins syndrome), disorders resulting in hypotonia (eg, Down syndrome, Prader-Willi syndrome, cerebral palsy, neuromuscular disorders), metabolic diseases, and laryngomalacia [21].

Polysomnography with oximetry and end-tidal carbon dioxide monitoring are used to diagnose OSAS and other SDB disorders in children. The severity of upper airway obstruction is determined by the apnea-hypopnea index, which is the mean number of apneas and hypopneas that occurred per hour of total sleep time. In children, OSAS is present when the apnea-hypopnea index is 1 or greater, and AHIs of 1 to 4, 5 to 9, and 10 or greater are considered mild, moderate, and severe, respectively.

Untreated childhood OSAS can contribute to neurocognitive, behavioral, cardiovascular, and metabolic comorbidities. Children may suffer from attention problems, hyperactivity, aggression, as well as poor social, communication, and adaptive skills [22]. Metabolic abnormalities have also been reported, including increased low-density lipoprotein cholesterol, reduced high-density lipoprotein cholesterol, reduced insulin sensitivity, and elevated liver enzymes, especially in obese children [23]. Children with underlying congenital heart disease, chronic lung disease, or sickle cell anemia may be more vulnerable to the effects of intermittent nocturnal hypoxia.

Fortunately, the treatment of OSAS improves behavior, learning, OSA symptoms, quality of life, and health indices. The first-line treatment of OSAS in children is adenotonsillectomy. Risk factors for residual OSAS after surgery include obesity, severe OSAS before surgery with an apnea-hypopnea index of greater than 20 per hour, age greater than 7 years, high Mallampati score, African American ethnicity, and underlying asthma, craniofacial abnormalities, and neuromuscular disease [24,25].

For children who are not surgical candidates or whose surgery did not fully resolve their OSA, there are additional treatment options. Nasal corticosteroids and/or oral montelukast can be used in children with mild to moderate OSA [26]. Continuous positive airway pressure or bilevel positive airway pressure can treat OSA of any severity. Positive airway pressure therapy should be titrated to the correct pressure for the child by a repeat in-laboratory PSG [24]. Mandibular distraction osteogenesis and mandibular advancement

surgeries have also been shown to improve OSA in children with mandibular insufficiency [27]. Tracheostomy is an option in severe cases when all other treatment options have failed.

Central sleep apnea

Central sleep apnea (CSA) is the lack of airflow owing to the absence of any respiratory effort. There are 2 main physiologic mechanisms by which CSA occurs [28]. There is either instability in the autonomic nervous system that senses and responds to O_2 and CO_2 levels (ie, the input pathway to the central respiratory control center in the medulla) or there is dysfunction in the phrenic nerve output and/or respiratory apparatus that does not allow the respiratory system to correctly respond to systemic O_2 and CO_2 levels (ie, the output pathway from the central respiratory control center to the lungs). The latter usually results in hypoventilation.

CSA can be seen in newborns, especially those born preterm. In fact, most preterm infants have central apneas within the first week of life owing to the immaturity of their central respiratory drive and chemoreceptor responses to O_2 and CO_2, as well as abnormal upper airway reflexes [29]. The incidence and severity of CSA in preterm infants is inversely related to gestational age and is diagnosed by recurrent, prolonged apneas lasting more than 20 seconds and periodic breathing with cyanosis, oxygen desaturations, and bradycardia [29]. Apnea of prematurity does not require a PSG for diagnosis [29].

The initial intervention is physical stimulation of the infant to induce spontaneous breathing. Oxygen and caffeine therapies are supportive while the infant matures to term gestational age [29]. Significant prematurity may require early mechanical ventilation or continuous positive airway pressure support. The persistence of CSA after 43 weeks gestational age indicates the presence of another disorder and CSA should be classified accordingly (ie, CSA owing to a medical or neurologic condition) [4]. Additional comorbidities need to be considered, including abnormal neurodevelopment or intracranial pathology (eg, Chiari malformation), anemia, infection/sepsis, metabolic derangements, gastric reflux, toxic substance exposure, and respiratory depressant medications/anesthesia. Treatment is aimed at correcting the underlying condition.

CIRCADIAN RHYTHM DISORDERS

Circadian rhythm, a biological process that has an endogenous and entrainable oscillation of about 24 hours, is controlled by the suprachiasmatic nucleus located in the anterior hypothalamus. Circadian rhythm disorders (CRSDs) occur when there is a desynchrony between the biological and environmental clocks and can occur when the person's natural circadian timing differs from the sleep–wake times required by work or school [4,30]. *Zeitgebers*, German for "time givers," are cues or entraining forces that keep most people synchronized to the 24-hour clock. Zeitgebers include light, social interactions, timing of food and drink, activity, and temperature. CRSDs can lead to significant daytime sleepiness, which can cause difficulty for schoolchildren. Inconsistent

or late bedtimes contribute to insufficient sleep and 25% of children get less than the recommended amount of sleep [11]. Daytime naps can worsen sleep hygiene by decreasing sleep drive and worsening the child's ability to fall asleep, contributing to insomnia.

Children's nighttime sleep needs vary, from infants who require 6 to 13 hours to teenagers who require 6 to 10 hours [11]. Beginning in infancy, a child may have a tendency for a delayed or advanced circadian rhythm and begin to show a preference of rising early or sleeping late [11].

Delayed sleep phase disorder (DSPD) is the most common CRSD with an estimated prevalence of 0.17% in the general population and 7% to 16% among adolescents and young adults [30]. It is estimated that 5% to 10% of patients with chronic insomnia in sleep clinics have DSPD. Several studies indicate a genetic predisposition for DSPD, with about 40% of patients having a positive family history [30]. DSPD is characterized by difficulty falling asleep before midnight and difficulty waking in the morning. People with DSPD often prefer to sleep until the late morning, and it is very common among high school and college/university students, who have later bedtimes and who wake later, especially on weekends. For many adolescents, school start times are too early for their circadian timing, which can lead to sleep deprivation. The deprivation can accumulate and lead to fatigue, poor cognitive functioning, and, if severe, growth delay. Insufficient sleep time decreases the amount of human growth hormone released and may alter the cortisol curve, which can further exacerbate sleep issues [30,31]. This sleep deprivation has public health implications, because teens are showing increasing signs of significant sleep deprivation owing to school starting much earlier than it did 20 years ago.

Irregular sleep–wake rhythm disorder (ISWRD) occurs when sleep timing is disorganized and must include a sleep wake pattern with multiple sleep and wake periods throughout the 24-hour cycle. The pattern is more common in children with developmental disabilities or the elderly with dementia, as well as patients with traumatic brain injury or other psychiatric problems. ISWRD may be caused by a weakened central circadian oscillation and temporal disorganization of circadian rhythms secondary to a decreased exposure to zeitgebers or degeneration of the suprachiasmatic nucleus neurons [30,32]. Treatment options include timed light and melatonin to improve daytime alertness, decrease napping, and consolidate nighttime sleep. Melatonin and light therapy are effective treatments for ISWRD. Combining melatonin, bright light, and behavioral interventions that engage the patient in a cognitively enriched environment with scheduled social and physical activities throughout the day, consistent bedtime routines, and good sleep hygiene are crucial in treating ISWRD [31,33–35].

Patients with advanced sleep phase disorder have difficulty staying awake from 6 PM to 9 PM, and they wake up between 2 AM and 5 AM. Advanced sleep phase disorder is less common in children than adults, and only occurs in 1% of the population. Advanced sleep phase disorder has a genetic component [32].

The treatment of CRSD includes lifestyle changes including improved sleep hygiene, bright light therapy, and melatonin supplementation given at precise times. Hypnotics can help to promote sleep and stimulants can promote wakefulness [30].

PARASOMNIAS IN CHILDREN

Parasomnias are classified by the *International Classification of Sleep Disorders* as non-REM (NREM) sleep arousal disorders and REM sleep behavior disorders. Parasomnias occur in 10% to 28% of children and differ in pathophysiology and management from sleep–wake disorders in adults. The NREM parasomnias are most common in children and adolescents but may persist into adulthood [36] (Table 1).

NREM-related parasomnias can be triggered by significant sleep deprivation, OSA, fever, psychological stress, or substances [4,36]. Parasomnias can be difficult to distinguish from nocturnal seizures and may require video-electroencephalogram PSG [4,37,38].

Treatment of NREM parasomnias includes ensuring a safe environment, especially for patients who sleep walk. NREM parasomnias often resolve by adulthood. Behavioral interventions include parental education, scheduled awakenings, resolution of sleep deprivation, hypnosis, treatment for medical causes, and minimizing the use of medications that contribute to the disorder [38,39]. Medications used to treat NREM parasomnias include clonazepam, temazepam, tricyclic antidepressants, and selective serotonin reuptake inhibitors.

Nightmares, which happen during REM sleep, occur in up to 50% of children between 3 and 6 years old, and in up to 80% of children with generalized and other anxiety disorders [38]. Most patients outgrow nightmares by adulthood [4,38,39]. Nightmare disorder includes recurrent episodes of awakenings from sleep, with the patient recalling intensely disturbing dreams that usually involve fear or anxiety; other emotions such as anger, sadness, disgust, and other dysphoric emotions can occur. The patient wakes to full alertness, has

Table 1
Parasomnias

NREM-related parasomnias	REM-related parasomnias
Disorders of arousal (from NREM sleep)	REM sleep behavior disorder
Confusional arousals	Recurrent isolated sleep paralysis
Sleepwalking	Nightmare disorder
Sleep terrors	
Sleep-related eating disorder	
Other parasomnias	Isolated symptoms and normal variants
Exploding head syndrome	Sleep talking
Sleep-related hallucinations	
Sleep enuresis	
Parasomnia owing to a medical disorder	
Parasomnia owing to a medication or substance	
Parasomnia, unspecified	

little confusion or disorientation, recalls being asleep and recalls the dream content. The presence of nightmare disorder can impair quality of life, resulting in sleep avoidance and sleep deprivation, with a consequent increase in the intensity of the nightmares. Other consequences such as insomnia and sleep avoidance, daytime sleepiness, and fatigue are also common. It can also cause or exacerbate underlying psychiatric distress and illness. Nightmare disorder is more prevalent in patients with comorbid psychiatric disorders or with trauma/posttraumatic stress disorder and requires trauma-focused intervention [38,39].

Effective behavioral treatment options include cognitive–behavioral therapy, image rehearsal therapy, eye movement desensitization reprocessing, exposure, relaxation, rescripting therapy, and desensitization. Clonazepam and venlafaxine are not recommended for the treatment of nightmare disorder. In some studies, prazosin was helpful for reducing nightmare frequency and intensity, increasing total sleep time and improving daytime symptoms of posttraumatic stress disorder [31,38,40].

RESTLESS LEGS SYNDROME, PERIODIC LIMB MOVEMENTS IN SLEEP, AND PERIODIC LIMB MOVEMENT DISORDER

Restless legs syndrome (RLS), also known as Willis-Ekbom disease, is a common neurologic sensorimotor disorder that manifests as an irresistible urge to move the body to relieve the uncomfortable sensations. These sensations always occur during resting, sitting or sleeping. Pediatric RLS, which occurs in about 2% of children [41], commonly worsens at night, causing difficulties with initiating sleep [42]. In adults, it is more prevalent in females, but there is no gender difference in pediatric RLS. A family history of RLS was detected in 90.9% of the patients with RLS, indicating high hereditability. RLS also has a significant association with circadian rhythm, with symptoms worsening in the evening and having a short remission in the morning after waking up [43]. Attention, sleep, mood, cognition, and quality of life are significantly affected in pediatric RLS patients [44], and attention deficit hyperactivity disorder, depressive symptoms, and anxiety are common comorbidities [45]. Pain is a common presentation in pediatric RLS, with 45% of patients using the terms "pain" and "hurting" to describe their symptoms; it is often misdiagnosed as growing pains [46]. A definitive diagnosis of RLS generally can be made in typically developing children by the age of 5 to 6 years, because they are able to accurately describe their symptoms [46,47]. The diagnosis of RLS in children is made using the criteria recommended by the International Restless Legs Syndrome Study Group. A definite diagnosis of RLS in children requires that the child meets all 4 essential adult criteria for RLS and is able to describe the leg discomfort in his or her own words, for example, "lot of energy in my legs," "my legs tickle," "feels like spiders crawling on my legs," and so on. Age-appropriate words to describe their symptoms are encouraged. If unable to describe these feelings, the child must meet all 4 essential adult criteria for RLS and 2 out of 3 supportive criteria. These criteria include the presence of

a sleep disturbance for age, having a biologic parent or sibling with definite RLS, or a PSG-documented periodic limb movement index of 5 or more per hour of sleep [47,48].

RLS diagnostic criteria in adults include 4 core essential symptoms:

1. An urge to move the legs
2. The urge to move begins or worsens when sitting or lying down
3. The urge to move is partially or totally relieved by movement
4. The urge to move is worse in the evening or night than during the day or only occurs in the evening or night.

The diagnosis of RLS is more difficult in children who are developmentally delayed, and in preschool-aged children who have restless and fragmented sleep; PSG is recommended for these children. Periodic limb movements (PLMS) are polysomnographic findings and are characterized by stereotypical jerks lasting between 0.5 to 10.0 seconds and occurring at 15- to 40-second intervals. An index of 5 or more PLMS per hour is abnormal in pediatric patients [47,49]. PLMS are present in approximately 80% of patients with RLS, and a periodic limb movement index of 5 or greater, when associated with a sleep complaint not accountable by any other sleep disorder, may suggest a periodic limb movement disorder (PLMD).

Various systemic changes including increased blood pressure and heart rate occur during PLMS [50,51]. Most patients with PLMS are not aware of these changes and the associated arousals that disturb their sleep. There seems to be a close relationship between RLS and PLMD in children based on family history and long-term follow-up studies [44,51–53].

The exacerbating factors for RLS include sleep deprivation, irregular sleep schedules, low body iron stores, caffeine, nicotine, alcohol, and certain medications (eg, antihistamines, serotonergic antidepressants, and neuroleptics). The management of RLS and PLMD in children involves both nonpharmacologic and pharmacologic approaches; currently there are no FDA-approved medications for children. Although emerging literature supports medical therapy in children with RLS and PLMD, experiences with these medications are limited. Most children and adolescents with RLS and PLMD have low iron storage; therefore, iron therapy should be considered as the first line of treatment. A serum ferritin of greater than 50 ng/mL is considered an adequate therapeutic target in children [44]. Correcting iron deficiency in children with RLS/PLMD can be difficult because it not well-absorbed in the gastrointestinal tract. Systemic illness in children can further limit iron absorption. Compliance with oral iron therapy in children is difficult owing to its gastrointestinal side effects, such as constipation. Iron absorption can be enhanced by oral vitamin C and by not taking it within 2 hours of consuming calcium/dairy products. An alternative intravenous iron sucrose treatment has shown to be beneficial for children with RLS/PLMD who cannot tolerate oral iron preparations or when there is a need for a rapid replenishment of iron stores [44].

The literature supports the use of off-label medication in pediatric RLS. Clonidine is the most commonly used medication for children with sleep problems and it is particularly useful when there are severe sleep onset problems in children with RLS [54,55]. It is typically prescribed at a dose of 0.2 to 0.4 mg at bedtime. Gabapentin, an anticonvulsant, can improve sleep quality and decrease the sensory symptoms of RLS [44,55]. Ropinirole and pramipexole are dopamine agonists that are FDA approved for RLS in adults.

SUMMARY

In recent years, an increased understanding and awareness of sleep disorders in children have been achieved. Further understanding and research into the pathophysiology, epidemiology, clinical evaluation methods, sequelae, and supportive treatment options for children with sleep disorders are needed to effectively manage them in the clinical settings. Clarification of the important role that sleep disturbances play in children with medical, developmental, and mental health disorders is a goal for future research.

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