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SLEEP AND NEUROLOGICAL CONDITIONS (A AVIDAN, SECTION EDITOR)

Pediatric Sleep Medicine Cases

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Abstract

Purpose of the Review In this review, we will discuss commonly encountered pediatric sleep disorders, their clinical presentations, evaluation, and management.

Recent Findings Sleep problems are common complaints in the pediatric population with an estimated prevalence of at least 25%. This review examines frequently seen pediatric sleep disorders including insomnia, obstructive sleep apnea, hypersomnolence, circadian rhythm sleep–wake disorders, parasomnias, and movement disorders. Their clinical manifestations vary, but left untreated, these sleep disorders result in significant impairment. A detailed sleep history is key component in the evaluation process. Other useful tools include sleep diaries, questionnaires, and actigraphy. Polysomnography is often required for diagnosis. Treatment varies depending on the underlying sleep disorder. Pharmacologic treatment is often limited due to the lack of studies of safety and efficacy in the pediatric population.

Summary Sleep disorders are commonly encountered in the pediatric population. Their clinical manifestations vary, though without treatment, many result in significant impairment. Detailed sleep history is an essential part of the evaluation process, though polysomnography is often required. Treatment depends on the underlying diagnosis.

Keywords Pediatrics · Childhood insomnia · Pediatric obstructive sleep apnea · Narcolepsy · Klein-Levin syndrome · Delayed sleep–wake phase disorder · Restless legs syndrome · Abnormal movements in sleep

Introduction

Sleep is an important process that critically impacts health and functioning. Children require good quality sleep that is adequate in duration and timing and free of sleep disturbances (see Table 1) [1•]. Sleep problems have an estimated prevalence of at least 25% in the pediatric population [2]. Left untreated, these sleep problems result in significant impairment impacting children both physically and psychologically. In this review, we provide an overview of commonly encountered pediatric sleep disorders including clinical cases, specific characteristics seen in the disorders, and management.

This article is part of the Topical Collection on *Sleep and Neurological Conditions*

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Clinical Vignette

Lily is an 8-month-old previously healthy female referred for difficulty falling asleep. Her parents report that since birth, Lily requires rocking and a pacifier to go to sleep. She wakes up frequently during the night when her pacifier falls out of her mouth. During these nighttime awakenings, she needs one parent to rock her back to sleep with the pacifier in place.

Insomnia

Insomnia is defined as persistent difficulty with initiating sleep, maintaining sleep, or waking up with an inability to return to sleep despite adequate sleep opportunity and resulting in daytime impairment [3••]. The estimated prevalence of insomnia is between 25 and 40% in children 4–10 years of age and 11% in adolescents [4, 5]. Childhood insomnia is typically characterized by bedtime resistance, inability to sleep independently, and/or frequent nighttime awakenings [3••]. In children, this may result in behavioral issues such as hyperactivity and inability to concentrate [6].

Table 1 Recommended duration of sleep in pediatric patients [1•]

Age	Recommended hours of sleep in a 24-h period
4 months to 12 months of age	12 to 16 h of sleep including naps
1 to 2 years of age	11 to 14 h of sleep including naps
3 to 5 years of age	10 to 13 h of sleep including naps
6 to 12 years of age	9 to 12 h of sleep
13 to 18 years of age	8 to 10 h of sleep

In the current third edition of the *International Classification of Sleep Disorders* (ICSD-3), insomnia is divided into three diagnostic categories: chronic insomnia, short-term insomnia, and other insomnia disorder [3••]. The second edition of the ICSD delineated multiple insomnia subtypes, including a separate pediatric diagnosis. While no longer used as formal diagnoses, the insomnia subtypes remain clinically useful. The most commonly encountered type of childhood insomnia is behavioral insomnia of childhood (BIC). This is typically associated with difficulty with sleep initiation and sleep maintenance. Within this type, there are three subtypes — sleep-onset association type, limit-setting type, and mixed type (see Table 2).

Evaluation for childhood insomnia involves a detailed sleep history, including information about the child's sleep patterns and sleep environment. Questionnaires and surveys may be utilized. Sleep diaries and actigraphy may be useful in capturing the child's sleep patterns and sleep duration. Polysomnography is not typically employed, though it may be useful to asses for other contributing sleep disorders such as periodic limb movements of sleep (PLMS) and sleep apnea [3••, 7].

Childhood insomnia is generally treated with behavioral interventions [9, 10]. Initial management may focus on improving sleep hygiene [7]. This includes developing a consistent bedtime routine, sleep schedule, and response to nighttime awakenings. A bedtime routine is a set of predictable activities that occur in the hour before a child falls asleep. The bedtime routine often includes activities relating to nutrition (such as feeding), hygiene (such as bathing), communication (such as reading a bedtime story), and physical contact (such as rocking) [11]. A consistent bedtime routine is associated with earlier bedtime, shorter sleep onset latency, fewer nighttime awakenings, and longer sleep duration [11].

Other behavioral interventions for the treatment of insomnia include unmodified extinction, graduated extinction, and scheduled awakenings [8, 9, 12]. In unmodified extinction (commonly described as "crying it out"), the child is put to bed at a set time at night and "ignored" until a designated time the next morning. The caregivers continue to monitor the child for injury and illness but ignore behaviors such as crying and tantrums [13]. Because unmodified extinction can be stressful for caregivers, a variation of unmodified extinction is often employed. In extinction with parental presence, the caregiver remains in the child's bedroom but "ignores" the child [13]. In graduated extinction, the child is "ignored" for specific periods of time on a fixed schedule or with increasing intervals before a caregiver checks in. These check-ins typically involve minimal interactions to avoid reinforcement of crying and tantrums [13]. With scheduled awakenings, the number and time of nighttime awakenings that the child experiences must be known. The caregiver then preemptively awakens the child before a typical spontaneous awakening. These awakenings become scheduled and are faded out with increasing time between awakenings [13].

Pharmacologic treatment for insomnia in children is not commonly used due to a lack of safety and efficacy. There have been limited studies involving melatonin and gabapentin [14–16].

Table 2 Behavioral insomnia of childhood [7, 8]

Subtypes of behavioral insomnia of childhood	Definition	Example
Sleep-onset association type	Children typically require specific objects or circumstances in order to initiate sleep or return to sleep following nighttime awakenings	An infant who requires rocking in order to fall asleep and return to sleep after nighttime awakenings
Limit-setting type	Children typically present with bedtime delaying or refusal. This is usually a consequence of inadequate limit setting. These children have prolonged sleep latency rather than frequent nighttime awakenings	A child who requests additional bedtime stories during bed
Mixed type	Children typically present with bedtime resistance and characteristics of sleep-onset association type	A child who refuses to go to bed but falls asleep with a parent present, and later requires the parent present to return to sleep following nighttime awakening

Table 3 Grading of tonsillar hypertrophy [17]

Tonsil size	Description of tonsils
0	Tonsils are absent or within the tonsillar pillar
1 +	Tonsils occupy less than 25% of the oropharynx
2 +	Tonsils occupy 26 to 50% of the oropharynx
3 +	Tonsils occupy 51 to 75% of the oropharynx
4 +	Tonsils occupy more than 75% of the oropharynx

Table 4 Common physical examination findings in pediatric patients with obstructive sleep apnea [18•]

Findings	
Tonsillar hypertrophy	Retrognathia
Adenoidal facies	High-arched palate
Micrognathia	Obesity

Clinical Vignette

John is a 6-year-old male recently diagnosed with attention-deficit hyperactivity disorder (ADHD) referred for snoring. His parents report that for the past year, he has snored nightly. His mother recently noticed pauses in his breathing when he sleeps. He has 3 + tonsillar hypertrophy on physical examination (see Table 3). He was recently seen by a pediatric otolaryngologist who told the mother that John has enlarged adenoids.

Pediatric Obstructive Sleep Apnea

Pediatric obstructive sleep apnea (OSA) is a common sleep disorder characterized by "intermittent complete or partial obstruction; prolonged partial upper airway obstructions; or both prolonged and intermittent obstructions that disrupt normal ventilation during sleep, normal sleep patterns, or both [3••]." The prevalence ranges from 1.2 to 5.7% [18•, 19–21]. OSA is typically characterized by snoring and difficulty breathing during sleep [3••]. Other common signs and symptoms include sleep enuresis, morning headaches, unusual sleeping positions such as sleeping in a seated position or with a hyperextended neck, and cyanosis [18•]. Daytime sequelae include daytime sleepiness, poor academic performance, inattention, and restlessness [18•, 22]. Predisposing factors include craniofacial anomalies (such as those seen in Pierre Robin sequence, Trisomy 21, Apert syndrome, and Crouzon syndrome), obesity, adenotonsillar hypertrophy, and neuromuscular diseases [3••].

Evaluation for OSA begins with a thorough history and physical examination. Common physical examination findings are listed in Table 4 [18•]. The American Academy of Pediatrics recommends that if a pediatric patient snores regularly and has other signs and symptoms of OSA, the patient should undergo polysomnography or be referred to an otolaryngologist or sleep specialist for further evaluation [18•]. Overnight attended in-laboratory polysomnography remains the gold standard test for the diagnosis of OSA in children [18•]. Positive polysomnogram exhibits "one of more obstructive apneas, mixed apneas, or hypopneas, per hour of sleep" and/or obstructive hypoventilation associated with snoring, flattening of the inspiratory nasal pressure waveform, and/or paradoxical thoracoabdominal movement [3••]. Severity is established based on the apnea–hypopnea index obtained from the polysomnogram. At this time, the American Academy of Sleep Medicine (AASM) does not recommend home sleep apnea tests to diagnosis pediatric OSA (Fig. 1) [23].

First line treatment is adenotonsillectomy in pediatric patients with adenotonsillar hypertrophy and without contraindication to surgical treatment [18•]. Contraindications

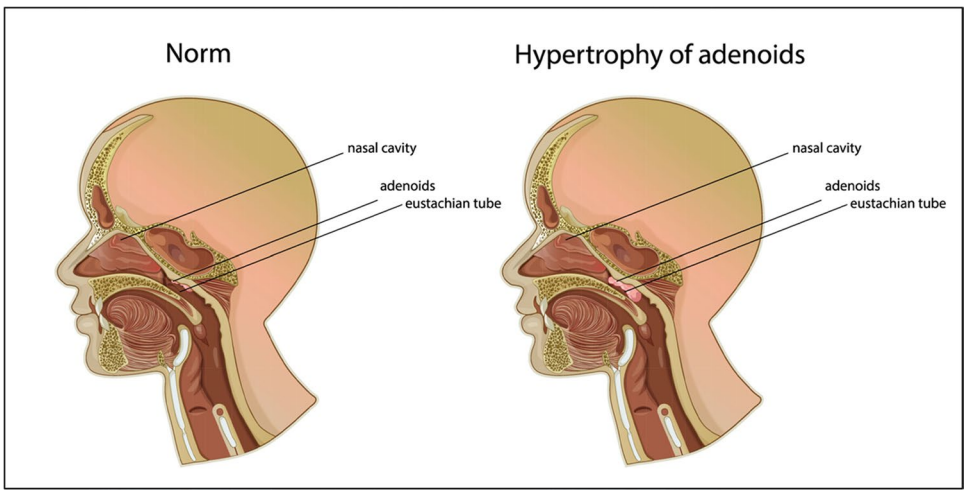

Fig. 1 Adenoidal hypertrophy. Citation: Shutterstock/Shutterstock.com [70]

to adenotonsillectomy include active infection, hematologic disorders, and uncontrolled systemic disease [24]. Improvements in respiratory parameters on polysomnography and quality of life were reported following adenotonsillectomy in children with OSA [25]. If surgical treatment is not an option or if OSA persists despite adenotonsillectomy, continuous positive airway pressure (CPAP) therapy should be considered [18•]. PAP therapy has been demonstrated to be effective treatment in children [26]. Weight loss has also been shown to be successful in obese teenagers, so it may be considered in overweight and obese pediatric patients in conjunction with other therapies [18•, 27]. Montelukast and intranasal steroids may also be beneficial in the management of mild pediatric OSA [28, 29].

Clinical Vignette

Andrew is a 10-year-old previously healthy male referred to pediatric sleep medicine for worsening daytime sleepiness despite adequate sleep opportunity and no evidence of sleep–wake cycle irregularities. His mother reports that for the past 6 months, he has been falling asleep in school unexpectedly and his academic performance has suffered. He has also had significant unexpected weight gain and several unexplained falls.

Central Disorders of Hypersomnolence

Narcolepsy

Narcolepsy is a chronic sleep disorder that is characterized by excessive daytime sleepiness [3••]. The pooled incidence rate of narcolepsy from six European countries is estimated to be 0.83 per 100,000 person-years in children 5 to 19 years of age [30].

Narcolepsy is divided into two types — narcolepsy type 1 (NT1) and narcolepsy type 2 (NT2). NT1 manifests with excessive daytime sleepiness and cataplexy and is associated with hypocretin (orexin) deficiency [3••]. Cataplexy is a brief episode of sudden loss of muscle tone, precipitated by strong emotions, and without loss of consciousness [3••]. It can vary from mild weakness to complete collapse, and is typically less than 1 min in duration [3••]. Cataplectic facies is a facial feature described in children with narcolepsy that usually manifests at disease onset [31]. It is characterized by repetitive mouth opening, tongue protrusion, and ptosis [31, 32]. Other common symptoms of narcolepsy include hypnagogic hallucinations, hypnopompic hallucinations, and sleep paralysis [33]. The hallucinations may be auditory, visual, or tactile [33]. Sleep paralysis presents with transient inability to perform voluntary movement involving skeletal muscle groups with the exception of eye movements and breathing [3••]. Other clinical features include unexplained excessive weight gain and precious puberty [34]. NT1 is associated with low hypocretin-1 in cerebral spinal fluid (CSF) and the presence of human leukocyte antigen (HLA) subtype DQB1*0602 [3••]. However, this HLA subtype is also present in up to 30% of individuals without narcolepsy, and primarily used to exclude the possibility of cataplexy as virtually all patients with NT1 are positive for this allele. NT2 presents with excessive daytime sleepiness, but cataplexy is absent [3••]. Hypnagogic hallucinations and sleep paralysis may also be seen in NT2, but to a much lesser degree than NT1 [3••].

Rapid eye movement (REM) behavior disorder (RBD) is associated with pediatric narcolepsy [3••]. It is characterized by abnormal behaviors (including both violent and nonviolent actions) during REM sleep [3••]. This sleep disorder may manifest prior to symptoms of narcolepsy [3••, 35]. Thus, in the pediatric population, children with RBD are thought to have narcolepsy until proven otherwise.

The diagnostic workup for narcolepsy includes polysomnography and multiple sleep latency test (MSLT) [3••, 36]. Actigraphy and a sleep log should be completed for at least 2 weeks prior to these studies to ensure that the test is interpreted in the absence of insufficient sleep or circadian rhythm irregularities [3••, 37]. Polysomnography should be conducted the night prior to the MSLT to measure sleep and rapid eye movement (REM) sleep latencies and to assess for another sleep disorder that could be contributing to the excessive daytime sleepiness. Patients on antidepressants or other medications that alter REM sleep must be tapered off of these medications for at least 2 weeks or five times the half-life of the medication with the longest acting metabolite before the polysomnogram. The MSLT is considered positive if the average sleep latency is less than 8 min and there are at least two sleep onset rapid eye movement periods (SOREMPs) present during the MSLT or there is one SOREMP present on the MSLT and one SOREMP present on the preceding polysomnography [3••]. HLA typing for HLA DQB1*0602 may be performed, but it is not diagnostic for narcolepsy [3••, 37]. Neuroimaging may be valuable in the case of trauma or infections and CSF levels of hypocretin-1 may be measured, particularly in the setting of a central nervous system process [3••, 37]. There is often a delay in the diagnosis of narcolepsy with a mean delay of 15 years [38]. This diagnostic delay is often due to lack of symptom recognition. Pediatric patients are often misdiagnosed with ADHD, epilepsy, depression, and insomnia [38].

Treatment for narcolepsy includes both behavioral and pharmacologic therapies. Behavioral treatment includes adherence to good sleep hygiene with a consistent sleep schedule and scheduled daytime naps [39, 40]. Pharmacologic therapies include wake-promoting agents, stimulants,

and antidepressants, though most of these medications are used off-label in the pediatric population [41, 42]. Only methylphenidate and amphetamines are FDA-approved for the treatment of EDS in the pediatric population [43]. Sodium oxybate is currently the only FDA-approved medication for the treatment of EDS and cataplexy and only in pediatric patients age 7 years of age and older (Fig. 2) [43].

Klein-Levin Syndrome (KLS)

Klein-Levin Syndrome (KLS) is a rare disorder with an estimated prevalence of 1 to 2 cases per million with disease onset is in early adolescence [3••, 44]. It is characterized by recurrent episodes of hypersomnia that are associated with behavioral, cognitive, and psychiatric disturbances [3••, 44]. During these episodes of hypersomnia, patients often sleep for 16 to 20 h a day waking only to eat and void [3••]. The associated disturbances include compulsive eating, hypersexuality, anxiety, depression, and hallucinations [3••]. Other common characteristics of the episodes include anterograde amnesia and derealization (altered perception of the environment) [3••]. The first episode is frequently preceded by infection, alcohol consumption, or head trauma [44]. The subsequent episodes recur every 1–12 months and

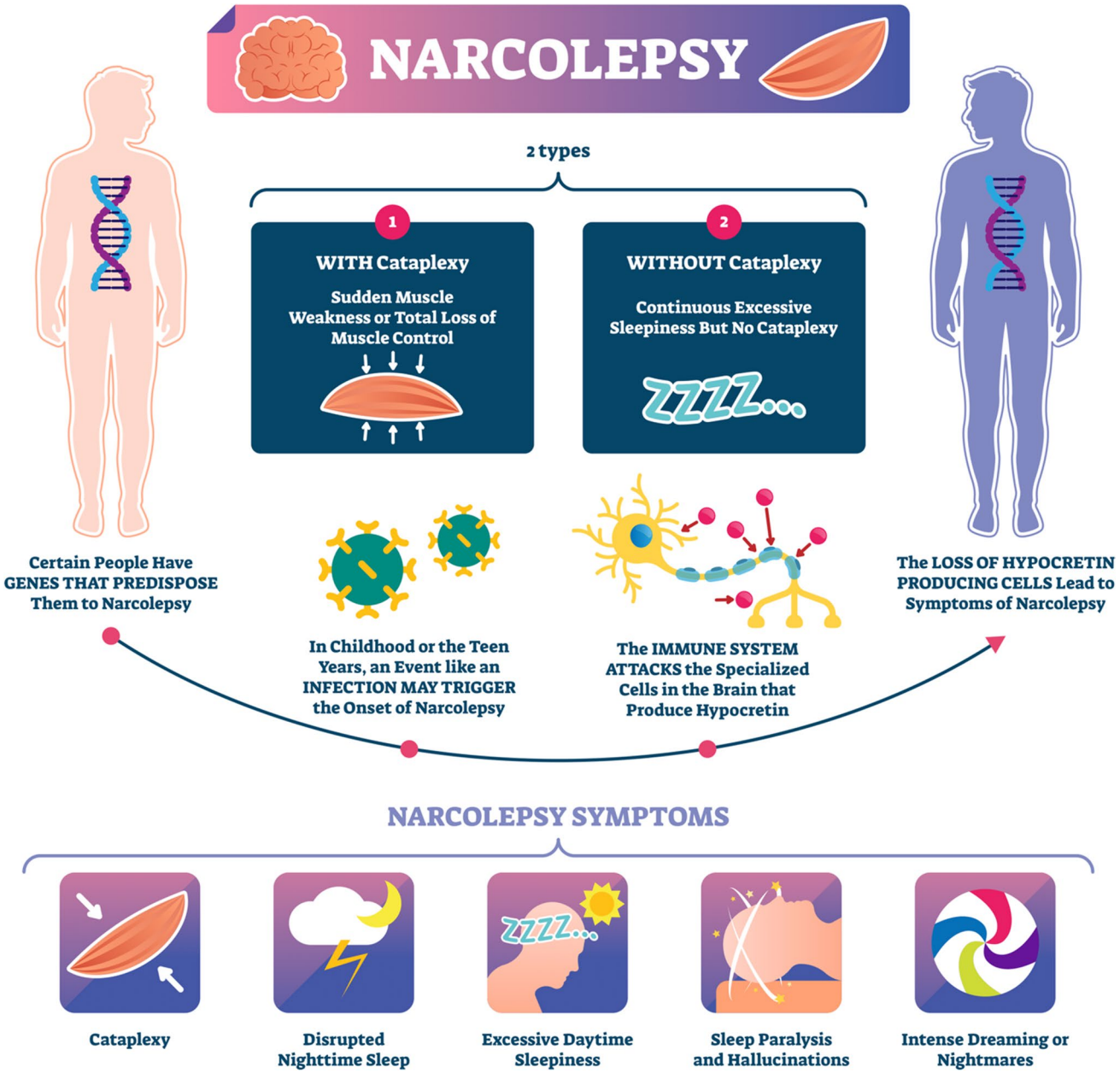

Fig. 2 Narcolepsy. Citation: Shutterstock/Shutterstock.com [71]

can last for several weeks to months [3••]. The disease generally resolves after a median of 8–14 years [3••]. Medications have been used to address specific symptoms of KLS; however, there is a lack of evidence available to support their use [45]. To date, there have been no randomized, placebo-controlled trials of pharmacologic therapies for KLS [45].

Clinical Vignette

Greg is a 16-year-old male with no significant medical history referred for insomnia. He reports that for the past year, he has had difficulty falling asleep. He tries to sleep at 11:00PM, but it takes several hours to fall asleep. He has difficulty waking up at 7:00AM. On weekends, he goes to bed at 2:00AM and wakes up at 10:00AM. He does not notice any other problems with his sleep.

Delayed Sleep–Wake Phase Disorder (DSWPD)

There are seven circadian rhythm sleep–wake disorders (CRSWD) delineated in the ICSD-3 [3••]. These disorders are the result of misalignment of the innate circadian rhythm and the desired sleep–wake schedule [3••]. This misalignment may manifest with EDS and symptoms of insomnia. Academic, occupational, and social performance are often affected.

Delayed sleep–wake phase disorder (DSWPD) is the most commonly diagnosed CRSWD with a reported prevalence among adolescents and young adults ranging from 7–16% [3••]. This disorder is characterized by a delay in sleep onset time relative to the conventional time and prolonged sleep latency when attempting to sleep at the conventional time [46]. The delay in sleep onset time is often 2 h or more [3••, 47]. Common complaints in DSWPD include non-restorative sleep and symptoms of insomnia such as difficulty initiating sleep at the conventional time, difficulty waking up at the desired or conventional time, and difficulty maintaining sleep [3••, 48]. Patients with DSWPD who are unencumbered by daytime obligations and able to follow their preferred sleep schedule have normal sleep quality and duration though at delayed times [46]. Predisposing and precipitating factors for DSWPD include genetic and environmental factors, such as the presence of a polymorphism in the circadian clock gene *hPer3* and exposure to bright light during certain times of the day [3••]. In children, ADHD and autism spectrum disorder (ASD) are associated with circadian rhythm abnormalities [3••]. The consequences of DSWPD vary, but can include depression, poor academic performance, and social difficulties [48]. Truancy, school failure, and loss of employment have been reported as well [46].

DSWPD predominantly affects adolescents, but it is important to recognize that there is a normal biological delay in the sleep pattern of adolescents. The start of adolescence and puberty is marked by significantly delayed sleep timing and tendency towards eveningness; this reverses in early adulthood [46, 49]. Social and behavioral factors such as personal and social activities during the evening may also exacerbate delayed sleep phase in adolescents [3••]. The coronavirus-19 (COVID-19) pandemic has been associated with changes in sleep as well. In Chinese preschoolers, there have been reported changes in sleep patterns with delayed bedtimes and wake times [50]. In Italian adults, the COVID-19 pandemic has been associated with sleep phase delay, decreased sleep quality, and increase in nighttime and early morning awakenings [51].

Evaluation for DSWPD involves a detailed sleep history. Sleep logs and actigraphy over a minimum of 7 days (though 14 days is preferable) are also recommended [3••, 47]. Standardized chronotype questionnaires may also be useful to determine the chronotype of eveningness and morningness [3••]. Polysomnography is not typically indicated. Laboratory measurements indicating delay of circadian rhythms may be helpful to confirm diagnosis [3••].

Treatment for DSWPD centers on circadian phase advancement and sleep timing. Treatment strategies include use of light therapy, exogenous melatonin, and cognitive behavioral therapy (CBT) [52]. There is currently no optimal strategy for light therapy, but light exposure at an intensity of 2500 to 10,000 lx for 1 to 2 h in the morning (between 6:00AM and 8:00AM) is most commonly used [53]. Limiting blue light exposure in the evenings is also key in treating DSWPD as blue light exposure suppresses melatonin secretion and increases alertness [54]. Likewise, there is no optimal dose of melatonin, but melatonin 3 to 5 mg/day 2 h prior to desired bedtime has been shown to advance sleep onset and increase sleep duration [55]. Melatonin and bright light therapy have demonstrated improvements in subjective daytime sleepiness, fatigue, and cognitive function [56]. CBT often includes cognitive therapy, sleep hygiene, and stimulus control therapy [57]. Chronotherapy is another treatment option that has been used. This treatment options involves delaying bedtime and wake time by 3 h each day until the desired sleep schedule is achieved [53]. Other treatment strategies recommended for DSWPD include adherence to sleep schedule, avoidance of naps, and avoidance of bright light exposure in the evening [53].

Clinical Vignette

Nicholas is a 4-year-old previously healthy male referred for sleep terrors. His parents report that at midnight every night, he will scream and cry, but he does not respond to his name

or remember the episodes in the morning. Attempts to wake him up during these spells result in his becoming even more inconsolable and aggressive.

Parasomnia

Parasomnias are defined as abnormal physiological or behavioral events that occur during sleep or during sleep transitions and are divided into non-REM (NREM) and REM parasomnias [3••]. Some commonly encountered NREM parasomnias in the pediatric population include confusional arousals, sleepwalking, and sleep terrors. Commonly encounters REM parasomnias include RREM nightmares and isolated sleep paralysis (seen in adolescents in association with sleep deprivation).

In the ICSD-3, parasomnias are divided into three categories — NREM-related parasomnias, REM-related parasomnias, and other parasomnias (see Table 5, Table 6, and Table 7). Confusional arousals, sleepwalking, and sleep terrors are considered NREM-related parasomnias because they occur during NREM sleep, which is typically during the first third of the night. NREM-related parasomnias are the result of incomplete awakening from sleep [3••]. Other characteristics include absent or inappropriate response to interference or redirection during the parasomnia, partial or complete amnesia during the parasomnia, limited or no associated dream or cognition, and possible confusion and disorientation following the episode [3••]. Confusional arousals are characterized by confusion occurring while in bed [3••]. Sleepwalking manifests with ambulation and other complex behaviors out of bed [3••]. Sleep terrors typically present with a cry or scream accompanied by heightened sympathetic autonomic nervous system activity such as tachycardia, diaphoresis, mydriasis, and tachypnea [3••].

The prevalence of confusional arousals is 17.3% in children aged 3 to 13 years [3••]. In early childhood, the prevalence of sleep terrors is 39.8% and the prevalence of sleep walking is 14.5% [58]. NREM-related parasomnias typically resolve by puberty [3••].

Table 5 Characteristics of NREM-related parasomnias [61]

	Confusional arousal	Sleepwalking	Sleep terrors
Timing	Anytime during sleep	First third of sleep	First third of sleep
Vocalizations	Yes	Yes	Yes (screaming or crying)
Movement out of bed	Rare	Yes	Occasionally
Responsiveness on awakening	Decreased	Decreased	Decreased
Autonomic activity	Normal	Normal	Increased
Confusion following the event	Yes	Yes	Yes
Amnesia of the event	Yes	Yes	Yes
Injury risk	Low if uninterrupted	Low if uninterrupted	More common

Table 6 Characteristics of REM-related parasomnias [61]

	REM sleep behavior disorder	Sleep paralysis	Nightmare disorder
Timing	Last third of sleep	Anytime during REM sleep	Last third of sleep
Vocalizations	Yes	Yes (moaning or groaning)	Occasionally
Movement out of bed	Yes	No	No
Responsiveness on awakening	Responsive	Responsive	Responsive
Autonomic activity	No	No	Yes
Confusion following the event	No	No	No
Amnesia of the event	No	No	No
Injury risk	Possible during dream enactment	None	None

Table 7 Characteristics of NREM-related parasomnias, REM-related parasomnias, and nocturnal seizures [59 60]

	NREM-related parasomnias	REM-related parasomnias	Nocturnal seizures
Timing	First third of sleep	Last third of sleep	Anytime
Frequency of the event	Not nightly	Not nightly	Nightly
Stereotypical behavior	No	No	Yes
Duration of the event	Minutes	Minutes	Seconds to minutes
EEG findings	Normal	Normal	Abnormal

REM-related parasomnias seen in pediatrics include REM sleep behavior disorder, recurrent isolated sleep paralysis, and nightmare disorder [3••]. Recurrent isolated sleep paralysis defined by transient inability to engage in voluntary movement may be seen in adolescents [3••]. The episodes are often associated with chest pressure, feeling of dread and are directly related to sleep deprivation. Nightmares are common in the pediatric population and typically start between 3 and 6 years of age with a peak between 6 and 10 years of age [3••]. They occur in the last third of the night and result in complete awakening [3••]. In the ICSD-3, sleep talking is considered a normal variant [3••]. Previous studies have demonstrated a prevalence of 84.4% in early childhood [58].

On the differential diagnoses for paroxysmal nocturnal events in the pediatric population is nocturnal seizures. Children with nocturnal seizures have stereotyped behavior and can have multiple episodes throughout the night [59, 60] (see Table 7).

Evaluation for parasomnia begins with detailed clinical history and physical examination. Polysomnography may be useful to assess for other sleep disorders, such as in the setting of a child with a high index of suspicion for sleep apnea, as it can be difficult to record the parasomnia event [3••].

Management of NREM-related parasomnias includes parental assurance, ensuring a safe environment, and proper sleep hygiene to minimize the possibility of sleep deprivation and decreased sleep efficiency [60]. Parents should avoid intervening during the parasomnia episodes as this may prolong or worsen the parasomnia [60]. Scheduled awakenings prior to the parasomnia episodes may be helpful if they occur frequently and at predictable times [60]. In this treatment strategy, the time that the parasomnia occurs is identified and the child is woken up 15 to 30 min prior to the identified time [62]. This is done nightly for 2 to 4 weeks until the parasomnia episodes resolve [62]. Medications are rarely used, except in rare situations such as parasomnias with no associated sleep disorder, parasomnias that occur frequently, and parasomnias that may be injurious to the patient or others. These medications include benzodiazepines and tricyclic antidepressants [60]. Clonazepam may be used for parasomnias that occur frequently, are refractory, and injurious. It should be used on a short-term basis and slowly tapered once improvement or resolution occurs [63]. Melatonin has been used to treat parasomnias, though this has been reported only in case reports [64].

Clinical Vignette

Oliver is a 2-year-old previously healthy male referred for restless sleep for the past 3 months. His mother reports that he often moves his legs at night. She is worried because he seems tired and sleepy the next day.

Restless Legs Syndrome

Restless legs syndrome (RLS) is a sensorimotor disorder characterized by an urge to move the legs that is typically accompanied by uncomfortable and unpleasant sensations [3••]. These symptoms become severe at night, improve with movement, and worsen with inactivity [3••]. RLS has a prevalence of 1.9% in children 8 to 11 years of age and 2.0% in children 12 to 17 years of age [65]. Children typically present with poor and restless sleep [66].

RLS is a clinical diagnosis. Diagnostic criteria includes all of the following features: "an urge to move the legs usually but not always accompanied by or felt to be caused by uncomfortable and unpleasant sensations in the legs; the urge to move the legs and any accompanying unpleasant sensations begin or worsen during periods of rest or inactivity such as lying down or sitting; the urge to move the legs and any accompanying unpleasant sensations are partially or totally relieved by movement, such as walking or stretching, at least as long as the activity continues; the urge to move the legs and any accompanying unpleasant sensations during rest or inactivity only occur or are worse in the evening or nigh than during the day; the occurrence of the above features are not solely accounted for as symptoms primary to another medical or a behavioral condition [67]." These symptoms cause impairment in functioning. Polysomnography is not typically required to diagnosis RLS, though it may be helpful in collaborating the clinical history provided by pediatric patients, as well as demonstrating periodic limb movements of sleep (PLMS), sleep abnormalities, and elevated arousal index [3••].

Pharmacologic treatment for RLS in pediatric patients remains limited. Once RLS has been suspected, evaluation of iron status should be considered given the association between RLS and iron deficiency. Serum ferritin levels in pediatric RLS patients have been reported to be below 50 mcg/L in 83–89% of cases [68]. Iron therapy has been shown to improve RLS symptoms [69••]. Other medications used in adults to treat RLS include dopaminergic medications, gabapentin, pregabalin, benzodiazepines, and clonidine [69••]. However, these medications have not been adequately studied in the pediatric population.

Abnormal Movement in Sleep

(1) Periodic limb movements of sleep (PLMS) typically presents with periodic, stereotyped limb movements that occur during sleep and result in sleep disturbance or other functional impairment [3••]. While these stereotyped limb movements may occur in any extremity, the lower extremities are most frequently involved [3••].

(2) Periodic limb movement disorder (PLMD) consists of PLMS and difficulty with sleep initiation, sleep maintenance, and unrefreshing sleep [3••, 67]. Diagnosis requires a polysomnogram that demonstrates an elevated PLMS index [3••]. In pediatric patients, this is greater than 5/h [3••]. The management of PLMD is similar to the management of RLS. Initial treatment includes evaluation of iron status (such as evaluation of ferritin), and consideration of iron therapy [69••]. As with RLS, there has been limited studies of other pharmacologic therapies in children.
(3) Sleep related bruxism results from rhythmic masticatory muscle activity (RMMA) [3••]. It typically presents as clenching and grinding of the teeth and/or thrusting or bracing of the mandible [3••]. Common complaints include morning jaw muscle pain, temporal headache, and tooth pain [3••]. Sleep related bruxism may result in abnormal tooth wear as well [3••].
(4) Sleep related rhythmic movement disorder (RMD) is typically seen in infants and children and is characterized by repetitive, stereotypical, and rhythmic motor behaviors [3••]. These behaviors include body rocking, head banging, body rolling, leg banging, and leg rolling [3••]. They typically occur near sleep onset, though may occur when drowsy or asleep [3••]. They may result in injury, interference with sleep, or impairment in daytime functioning [3••].
(5) Benign sleep myoclonus of infancy (BSMI) manifests as repetitive myoclonic jerks seen in neonates and infants [3••]. These movements typically involve the limbs, trunk, or whole body and occur only during sleep [3••]. They resolve upon arousal [3••].

Conclusion

Sleep problems are common complaints in the pediatric population. They may be the result of commonly encountered sleep disorders, such as insomnia, sleep disordered breathing, hypersomnolence, circadian rhythm abnormalities, parasomnias, and movement disorders. Detailed sleep history is critical for diagnosis. Polysomnography is frequently required in the pediatric population. Treatment strategies vary depending on the etiology of the sleep problem. Left untreated, these sleep disorders often result in impairment in functioning.

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Declarations

Conflict of Interest Yolanda Yu declares that she has no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by the author.

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Papers of particular interest, published recently, have been highlighted as:
• Of importance
•• Of major importance

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