The Parasomnias

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KEYWORDS

• NREM parasomnias • Confusional arousals • Sleepwalking • Sleep terrors
• REM-related parasomnias • Sleep enuresis

KEY POINTS

- Parasomnias affect a large proportion of children.
- Pediatricians and psychiatrists may not be aware of these sleep disorders and the implications for their patients.
- Parasomnias (especially those that are rapid eye movement related) may be associated with psychiatric comorbidities.
- Understanding of the pathogenesis and diagnostic testing for these are still being developed.

INTRODUCTION

The International Classification of Sleep Disorder (ICSD-3) defines parasomnias as "undesirable physical events or experiences that occur during entry into sleep, within sleep, or during arousal from sleep."[1] The term parasomnia derives from the Greek word para meaning around and the Latin somnus meaning sleep.[1] The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, defines parasomnias as recurrent episodes of incomplete awakening from sleep with usual amnesia of the episode, and little or no dream imagery and distress or social impairment.

Parasomnias include several disorders that share clinical and physiologic characteristics: (1) clear and dramatic symptoms associated with skeletal muscle activity; (2) correlation with age; (3) unassociated medical problems; (4) absence of specific polysomnographic anomalies; (5) spontaneous resolution; and (6) unknown cause.

They are classified based on the sleep stage during which they occur: non–rapid eye movement (NREM)–related parasomnias, which include disorders of arousal

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(confusional arousals, sleepwalking, sleep terrors, and sleep-related eating disorder); rapid eye movement (REM)–related parasomnias (REM sleep behavior disorder [RBD], recurrent isolated sleep paralysis, and nightmare disorder); and other parasomnias (exploding head syndrome, sleep-related hallucinations, and sleep enuresis) (**Table 1**).

The clinical diagnosis is mainly based on description of the event by parents, but often these descriptions are inaccurate and a home video recording of the event (ie, with a smartphone) can be helpful for the diagnosis. Parents should be guided to have a better picture of the event with a set of specific questions: (1) timing of appearance of the symptom; (2) specific manifested movements and symptoms; (3) reaction to external interventions; (4) presence of stereotypies (rhythmic, repetitive movements); and (5) recall of the episode in the morning.[2]

Polysomnography(PSG), or better video-PSG (vPSG), is not always recommended for the diagnosis or evaluation of typical parasomnias, but it is indicated for injurious parasomnias and when nocturnal seizures or other comorbid sleep disorders are suspected.

Most parasomnias are associated with specific sleep stages and have a benign evolution, with spontaneous resolution during puberty.[3]

NON–RAPID EYE MOVEMENT–RELATED PARASOMNIAS

NREM-related parasomnias, also named disorders of arousal (DoAs), are defined in the ICSD-3 as recurrent episodes of incomplete awakening from sleep, characterized by inappropriate or absent responsiveness to efforts of others to intervene or redirect the person during the episode and with limited or no associated cognition or dream imagery. Patients have partial or complete amnesia for the episode.[1]

DoAs include confusional arousals, sleep terrors, sleepwalking (also called somnambulism), and sleep-related eating disorder (SRED). There is usually only 1 event per night, which typically occurs during the first third of the major (usually nocturnal) sleep episode. The individual may continue to appear confused and disoriented for several minutes, or even longer, following the episode. DoAs are benign events, typically occurring in childhood and ceasing by adolescence. Some individuals may experience more than 1 type of arousal parasomnia.[3]

In some cases, differential diagnosis is not simple because DoAs may mimic nocturnal seizures or RBD and parents' descriptions of events may be inaccurate, especially when the events make them anxious or frightened.

Pathophysiology

DoAs result from an NREM sleep-wake state dissociation because patients appear to be simultaneously awake (with retention of their motor and behavioral functions) and asleep (with impairment of cognition, judgment, and memory for the events).[4]

Table 1
Classification of parasomnias based on the International Classification of Sleep Disorders, Third Edition

NREM-Related Parasomnias	REM-Related Parasomnias	Other Parasomnias
Confusional arousals	RBD	Exploding head syndrome
Sleepwalking	Recurrent isolated sleep paralysis	Sleep-related hallucinations
Sleep terrors	Nightmare disorder	Sleep enuresis
Sleep-related eating disorder	—	—

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A cranial single-photon emission computed tomography (SPECT) study captured a sleepwalking event in a 16-year-old boy characterized by activation (increased regional cerebral blood flow) of thalamocingulate (motor coordination) pathways, with simultaneous deactivation in the frontal lobe.[5] The stereotactic electroencephalogram (EEG) recordings of NREM parasomnias showed local activations of the motor cortex, with an EEG pattern of wakefulness in the motor and central cingulate cortices paralleled by a concomitant increase in slow waves in the dorsolateral prefrontal cortex.[6,7] This finding confirmed the coexistence of simultaneous wakelike and sleeplike activities in different brain regions during an episode, which explains the motor and emotional activation along with clumsiness, uninhibited behavior, and amnesia of the event. The variable level of awareness during an NREM parasomnia could depend on the quantity and position of the local persistence of these slow waves.[4]

EEG studies showed increased arousals and cyclic alternating pattern rate during slow wave sleep, reflecting an alteration of NREM sleep continuity and different dynamics of slow wave activity throughout the night.[8] An EEG spectral analysis study showed a significantly greater number of arousals during stage N3 in sleepwalkers, particularly during the first NREM sleep cycle.[9]

Some behaviors observed in DoAs resemble stereotyped, archaic behaviors (such as defensive postures, violent gestures, and feeding) that result from the activation of neural circuits (mainly subcortical), namely central pattern generators (CPGs). CPGs are functional neural organizations located in the spinal cord, mesencephalon, pons, and medulla regulating innate behavioral automatisms and survival behaviors.[10] Some manifestations of DoAs could result from CPG disinhibition, as a result of prefrontal cortex dysfunction, permitting the expression of complex behaviors without conscious control.[11]

NREM parasomnias can also occur or worsen when specific factors are present: (1) conditions that increase slow wave sleep, such as sleep deprivation and drugs (zolpidem, lithium, and sodium oxybate); (2) conditions that cause repeated cortical arousals determining sleep fragmentation, such as sleep disorders (obstructive sleep apnea, periodic limb movements of sleep, chronic pain, narcolepsy), noise, fever, physical activity late in the day, emotions, stress, and anxiety; and (3) impaired arousal mechanism and persistence of sleep drive resulting in a failure of the brain to fully transition into wakefulness.[3]

In addition to the precipitating factors mentioned earlier, there is a strong genetic contribution to DoAs. Positive family history is found in up to 80% of children presenting with DoA[12] (ie, precipitating factors act on an underlying genetic predisposition). The contributions of both determine the frequency and severity of the parasomnia events.

Epidemiology

NREM parasomnias are common pediatric sleep disorders that tend to decrease across development and in adulthood, possibly because of decreased slow wave sleep with aging. Laberge and colleagues[13] found that about 17% of children between 3 and 13 years old experienced occasional or frequent episodes of confusional arousals.

Ohayon and colleagues[14] observed that confusional arousal affects 4.2% of the general population, decreasing from 6.1% among those 15 to 24 years old to 3.3% among those aged 25 to 34 years old, and stabilizing around 2% after age 35 years. The prevalence of sleepwalking in children ranges from 3% to 14.5%, and most episodes resolve after age 10 years. Sleep terrors have the greatest incidence in preschool children, with a reported overall prevalence of 17.3% in children between 3 and 13 years old. The peak

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prevalence of sleepwalking (13%) occurred at age 10 years. One-third of children who had sleep terrors went on to develop sleepwalking.[15]

Clinical Features

NREM parasomnias occur along a continuum, ranging from confusional arousals with low motor and autonomic activation at the lower end of the spectrum up to night terrors characterized by intense motor activity and autonomic activation at the high end of the spectrum. There is an age-related evolution with different presentations: a child might present a sequence of confusional arousals in early childhood and sleep terrors later on, followed by sleepwalking in late childhood and adolescence. These 3 disorders show common and distinct features (**Table 2**).

Confusional arousal

Confusional arousals are characterized by mental confusion or confused behavior that occurs while the patient is in bed, in the absence of terror or ambulation outside the bed.[1] These events often begin with the individual sitting up in bed with eyes open and staring or looking around: the child looks awake but is confused, disoriented, does not respond adequately to orders, can be engaged in conversation with slowed speech, and shows blunted response to questioning.

Episodes usually start with a moan and some movements and then progress toward crying; the expression of terror typical of pavor nocturnus is missing. The duration varies from a few minutes to 40 to 60 minutes. The exact prevalence is unknown because of the difficulty of identification, in part because symptoms can be too mild to be recognized, or because of inaccurate descriptions by distressed parents. Onset is before the child turns 5 years old.[3]

Sleepwalking

Sleepwalking is characterized by complex behaviors that are usually initiated during arousals from slow wave sleep and culminate in leaving the bed in an altered state of consciousness. The child acts out more or less complex, automatic movements that vary from simply standing by the bed to walking around the house in an agitated manner associated with semipurposeful behaviors such as eating, drinking, or leaving home. These episodes can be concomitant with vocalization, often in an

Table 2
Clinical features of disorders of arousal

	Confusional Arousal	Sleepwalking	Night Terrors
Age (y)	2–10	4–12	1.5–10
Onset	First third of night	First third of night	First third of night
Agitation	Mild	No/poor	Marked
Autonomic Activity	Mild	Mild	Marked
Motor Activity	Low	Complex	Rarely complex
Ictal Behavior	Whimpering, some articulation, sitting up in bed, inconsolable	Walking around, quiet or agitated, unresponsive to verbal commands	Screaming, agitation, flushed face, sweating, inconsolable
Amnesia	Yes	Yes	Yes
Threshold of Arousal	High	High	High
Familiality	High	High	High

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incomprehensible language, and aggressive acts can occur, usually in relation to attempts to block or awaken the child.

The average duration is around 10 minutes. Typically, parents report that the episode ends after the child has gone to the bathroom to urinate; this has led to the supposition that bladder repletion may contribute to these episodes by triggering a partial arousal.

The age of onset is generally between 4 and 8 years, with a peak at 10 years and resolution during adolescence. Prevalence is between 15% and 30% for sporadic episodes and 3% for frequent episodes without differences between boys and girls.[13]

A positive family history was found in up to 80% of subjects; other family members might be affected by sleepwalking or other arousal disorders, with a chance of recurrence of 45% if 1 parent is affected and 60% if both parents are affected.[8]

Association with mental disorder has only been noted when sleepwalking persists into adulthood.[8]

Sleep terrors

Sleep terrors are distinguished from other DoAs by their prominent autonomic activation and distinct expression of terror. During the episode, the child presents with a sudden onset of partial awakening, a loud cry, intense agitation, autonomic symptoms (pallor, sweating, tachycardia, tachypnea, increased arterial pressure, mydriasis) and increased muscle tone. The child is not very responsive to environmental stimulation and does not recognize those close to them; the child may seem to look beyond, appears terrified, and is inconsolable; less often, the child may also get out of bed and walk around. Some children show prolonged inconsolability without awareness. If awakened, the child will be confused and disoriented, although they usually return to sleep quickly and do not remember the episode in the morning. The episodes are short (but can range from 30 seconds to 30 minutes) and rarely occur more than once in a night.[16]

Prevalence varies between 1% and 6% and is slightly higher among boys; typical age of onset is from 2 to 4 years, with a peak between 5 and 7 years. There is a high overlap between sleep terrors and other parasomnias. Precipitating factors are stress, fever, bladder distension, and sleep deprivation, and there is no apparent relationship with mental disorder.[17]

Differential Diagnosis

The diagnosis of DoA is mainly based on clinical descriptors; the availability of a videorecording of an episode is extremely useful. A clear clinical history can be sufficient to diagnose an NREM parasomnia in most cases, but in others only vPSG recording can clarify the nature of the disorder. Typical PSG features of sleep architecture of patients with NREM parasomnia include hypersynchronous delta waves, irregular slow wave activity, and NREM sleep instability. NREM parasomnia needs to be distinguished from other parasomnias and sleep-related seizures.[18]

Although the diagnosis can be made clinically, some situations warrant further evaluation. A sleep study (vPSG) should be ordered if a concomitant sleep disorder is suspected (obstructive sleep apnea, periodic leg movement disorder, and so forth). Consider referral to child psychiatry if posttraumatic stress disorder, anxiety, depression, or a neurodevelopmental disorder is suspected, and referral to neurology if there are frequent, stereotypic, and brief episodes suspicious for seizures.

Nightmares can resemble sleep terrors but occur during REM sleep in the second half of the night. After a nightmare, children become fully alert quickly, respond positively to comforting, may report the dream content after awakening, and show lower

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levels of autonomic activation and mobility. Nocturnal panic attacks are characterized by waking from sleep in a state of panic, with intense fear or discomfort, but are more frequent in adults; immediately after the episodes, children appear oriented, can vividly recall the attack, and have difficulty returning to sleep.[19]

A challenge in differential diagnosis is represented by sleep hypermotor epilepsy (SHE), previously called nocturnal frontal lobe epilepsy, in which seizures occur almost exclusively during sleep with different sleep-related motor attacks of increasing complexity and duration. Seizures are usually brief, abrupt, can occur at any time in the night (or several times), and the presentation ranges from stereotypic movements to dystonic positions and nocturnal wandering". However, there are some similarities and possible coexistence of parasomnias in children with SHE, and the differential diagnosis between these disorders can be complicated. Further evaluation by neurology and vPSG study is recommended if SHE if suspected.[20]

Non–rapid Eye Movement Parasomnia Treatment

Parasomnia episodes are often benign and normally require no treatment. General management considerations include prevention, safety measures, and bystander intervention guidelines (**Table 3**). Relaxation techniques before sleep and hypnosis can also be helpful. Another technique is anticipatory or scheduled awakenings, which consist of awakening the child about 15 minutes before the presumed time when the episode will occur (usually within 2 hours of falling asleep). This technique may shift the child into a lighter state of sleep, thereby aborting the event. The Lully Sleep Guardian is a vibrating alarm placed under the mattress and connected to the parent's smartphone that can be set to activate at predetermined times, resulting in arousal.[21]

Pharmacotherapy should be considered only when episodes are frequent or dangerous to the patient or others, or when they cause undesirable secondary consequences, such as excessive daytime sleepiness, or distress to the patient or their family. Parents should be advised that prescribed drugs are considered off-label.[16,22] L-5-Hydroxytryptophan, a precursor of serotonin that may modify central serotoninergic system dysfunction or enhance production of sleep-promoting factors, can be effective for treating sleep terrors.[23] Melatonin has also been reported to be helpful for patients with sleepwalking and sleep terrors.[24] A referral to sleep medicine specialists may be indicated when a coexisting sleep disorder is suspected.

Sleep-related eating disorder

SRED consists of "recurrent episodes of involuntary eating and drinking during arousals from sleep, associated with diminished levels of consciousness and

Table 3
Prevention and safety approach for non–rapid eye movement parasomnias

Safety Measures	Prevention	Bystander Guidelines
Remove furniture or objects near bed	Good sleep hygiene	Silently observe
Lock doors and windows	Avoid sleep deprivation	Permit episodes to run course
Security alarm to warn family members	Avoid environmental stimulation (ie, light, sound, touch)	Intervene only to prevent injury
Stairwell barriers and night light to prevent falls/injuries	Address comorbid sleep disorders (obstructive sleep apnea, periodic limb movement disorder)	Avoid physically restraining the child

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subsequent recall, with problematic consequences."[1] Episodes typically occur during partial arousals from sleep during the first third of the night, with impaired subsequent recall.[25] SRED occurs predominantly in young women, with average age of onset approximately 22 to 27 years. However, this disorder is often hidden or overlooked and, on average, patients receive treatment of it after 12 to 16 years from onset.[16]

RAPID EYE MOVEMENT–RELATED PARASOMNIAS

RBD, sleep paralysis, and nightmare disorders are the parasomnias of REM sleep, according to the ICSD-3.[1]

The main differences between NREM parasomnias and REM-related parasomnias are (1) the linkage to specific REM stage; (2) the occurrence during the second half of the night, when REM is more prevalent; (3) dream enactment behaviors; and (4) absence of mental confusion on awakening. On very rare occasions, some patients meet the diagnostic criteria for both NREM and REM parasomnia and are diagnosed with parasomnia overlap disorder.[26]

Rapid Eye Movement Sleep Behavior Disorder

RBD is characterized by complex and violent behaviors with enactment of dreams that are often unpleasant, action-filled, and violent and that can cause sleep disruption and injuries to the patients or to their bedpartners.

Pathogenesis is linked to the absence of the typical REM elimination of muscle tone (atonia). In the absence of normal REM atonia, patients present with recurrent episodes of enacting their dreams, behaviors that can vary from small hand movements to violent activities, such as punching, kicking, or leaping out of bed.[26] The patient usually remembers the dream. For instance, patients with RBD may dream of being chased and run out of the bed, or that they are fighting and punch their bed partner.

RBD in childhood and adolescence is rare and is usually associated with narcolepsy or idiopathic hypersomnia, neurodevelopmental-neurodegenerative disorders, or structural brainstem abnormalities; it can also represent a side effect of pharmacologic agents, such as selective serotonin reuptake inhibitor agents.[27] Further evaluation is always indicated in children presenting with symptoms suspicious of RBD. Evaluation should include referral to a sleep specialist or a neurologist, vPSG, and brain imaging.

Nightmare Disorder

Nightmare disorder is characterized by "recurrent, highly dysphoric dreams, which are disturbing mental experiences that generally occur during REM sleep and that often result in awakening."[1] On awakening from the dysphoric dreams, the person rapidly becomes oriented and alert.

Occasional nightmares are common in children, ranging from 60% to 75%, but the prevalence of nightmare disorder is estimated to be 1.8% to 6%. Nightmares can also occur more frequently in children with posttraumatic stress disorder.[28] A child with nightmare disorder may be scared but usually manages to report the dream and is well oriented, with an intact sensorium; parental intervention is accepted well. During the nightmare, there is little motor activity and the child does not move out of bed (because of REM atonia) and there is no dream enactment.

Emotional contents of nightmares are characteristically negative, with anxiety and fear but also anger, rage, embarrassment, and disgust. Exposure to violent content in electronics (television or computer programs) may contribute to nightmares and should be avoided as part of the sleep hygiene and bedtime routine education.

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Monsters or other fantastical images often characterize the dreams of young children, whereas adolescents may experience more realistic images linked to daytime stressors or traumatic events.[29]

Recurrent Isolated Sleep Paralysis

Recurrent isolated sleep paralysis is defined as a period of inability to perform voluntary movements that occurs at the beginning of sleep of a sleep period (hypnagogic) and/or after waking up (hypnopompic). Each episode lasts seconds to a few minutes and causes clinically significant distress, including bedtime anxiety or fear of sleep. The disturbance is not better explained by another sleep disorder (especially narcolepsy), mental disorder, medical condition, medication, or substance use.[30] Prevalence estimates vary widely, between 6% to 40%. Isolated episodes are exacerbated by sleep deprivation, stress, and sleep-wake rhythm irregularities.[31]

The individual experiencing sleep paralysis is conscious and alert, but feels paralyzed; all muscle groups are involved, with the exception of the diaphragm and the extrinsic muscles of the eye. The attacks usually end spontaneously, although they may occasionally be stopped intentionally if the individuals move their eyes rapidly or are administered tactile stimuli. These episodes commonly begin in adolescence, but can also appear in childhood.[32] Hallucinations can also occur during paralysis and commonly include sensing the presence of others nearby, pressure on the chest, or hearing footsteps.

The pathogenesis of this disorder is linked to the persistence of REM atonia into wakefulness, thus normal mental activity occurs in the presence of body paralysis.

First-line treatment is reassurance that the episodes are benign. Because sleep deprivation exacerbates sleep paralysis, recommendation of an adequate amount of sleep per age and education about sleep hygiene should be implemented. Pharmacologic treatment is rarely needed but may be considered in severe, debilitating cases with significant daytime consequences. REM-suppressing agents such as low doses of tricyclics, clonidine, or clonazepam could be tried.[31] Other sleep disorders that interrupt sleep (such as obstructive sleep apnea) can contribute to sleep deprivation and exacerbation of sleep paralysis. If snoring, gasping, or witnessed apneas are reported, a sleep study should be ordered. Sleep paralysis is also a symptom commonly seen in narcolepsy. If other symptoms, such as excessive daytime sleepiness, cataplexy, or hallucinations, are reported, referral to a sleep specialist or neurologist is recommended.

SLEEP ENURESIS

Sleep enuresis (SE) is characterized by recurrent involuntary voiding that occurs during sleep. In primary SE, recurrent involuntary voiding occurs at least twice a week during sleep after 5 years of age in a patient who has never been consistently dry during sleep for 6 consecutive months. SE is considered secondary in a child or adult who had previously been dry for 6 consecutive months and then began wetting at least twice a week. Both primary and secondary enuresis must be present for a period of at least 3 months.[33]

SE is one of the most common problems in pediatrics, with a general prevalence of 3% to 15%. SE is more frequent in boys less than 11 years of age, although after 11 years of age no sex differences are reported. Spontaneous remission during childhood occurs in around 15%. There is a strong genetic predisposition for primary SE.

SE is defined as monosymptomatic when the child has no associated daytime symptoms of bladder dysfunction (such as wetting, increased voiding frequency, urgency, jiggling, squatting, and holding maneuvers).[34]

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The most accepted hypothesis for the pathogenesis of SE is that it involves 3 systems: excessive nocturnal urine production, nocturnal bladder overactivity, and failure to awaken in response to bladder sensations.[35] Other pathophysiologic mechanisms are mostly related to sleep fragmentation, which can be secondary to sleep-disordered breathing[36] or periodic limb movements during sleep.[37]

From a developmental point of view, complete control of the bladder at night is usually achieved by age 5 years; thus, bed-wetting in toddlers is considered physiologic.

Children with SE are often described as deep sleepers, with higher arousal thresholds.[38] Enuretic events happen mainly during the first part of the night, and can occur in all sleep stages.[33] One study reported that patients with enuresis are subjectively sleepier than healthy controls and more difficult to awaken[39]; this was attributed to sleep fragmentation, which might be responsible for the higher arousal threshold and is consistent with a large body of research.[40]

Secondary SE is more commonly associated with urinary tract infections, malformations of the genitourinary tract, medical conditions that result in an inability to concentrate urine (diabetes mellitus or insipidus, sickle cell disease), and increased urine production secondary to excessive evening fluid intake (caffeine ingestion, diuretics, or other agents). Furthermore, neurologic diseases (spinal cord abnormalities with neurogenic bladder or seizures) and psychosocial stressors (parental divorce, neglect, physical or sexual abuse, institutionalization) should be considered.[33]

Enuresis can lead to a reduction of a child's self-esteem and to personality problems. Behavioral therapy is based on general hygiene measures (moderate restriction of evening drinks), elimination of negative family habits (eg, repeated intimate care, excessively careful attitudes toward sphincter control), sphincter conditioning exercises and bladder gymnastics training, behavioral and conditioning measures (keeping a diary, scheduled awakenings), and motivational techniques with positive reinforcement.

The most effective treatment of enuresis is a bed-wetting alarm, a system consisting of a small sensor clipped to the underwear, which is connected to a small battery-powered speaker that is activated when the sensor becomes wet. The resulting alarm awakens the wearer (or parents), alerting them that enuresis has begun. Controlled studies have shown that this approach is superior to all other methods, including pharmacologic or psychotherapeutic treatment. The drug options include desmopressin, imipramine, and oxybutynin.[41]

SUMMARY

Pediatricians and psychiatrists have multidisciplinary tasks when managing parasomnia sleep disorders in children. Education to encourage a regular lifestyle, adequate sleep hygiene, avoid sleep deprivation, create a personalized sleep ritual with a regular bedtime even on weekends, and provide quiet sleeping conditions are the first steps in the management of these disorders.

Understanding of the pathogenesis and key diagnostic testing for parasomnias is still a work in progress; a critical step in the correct diagnosis will be to transcend phenomenological categorization of these disorders and to establish a pathophysiologically defined classification scheme. Such an approach might incorporate candidate biomarkers such as hypersynchronous slow waves and genetic links, which should provide improved ways to classify these mysterious events and to identify their precise management.

Cognitive behavior therapy or relaxation training may be helpful and possibly lead to a long-term benefit to these children or adolescents if they learn how to recognize the

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signs of the condition and how to cope with it. Even if benign, parasomnia events can have a substantial negative repercussion on quality of life.

Pharmacologic treatment currently focuses primarily on sedative medications, which are obviously not the first choice in children. The development of pathophysiologically based categorization is essential to support further research showing the efficacy of clinical therapies and identifying the key characteristics for optimal therapeutic outcomes, leading to tailoring treatments, whether nonpharmacologic or pharmacologic, for individual patients.[42]

CLINICS CARE POINTS

- Parasomnias are usually benign and self-resolving.
- Parasomnias can be exacerbated by sleep deprivation or another sleep disorders, such as obstructive sleep apnea or periodic leg movements of sleep.
- Treatment options often include sleep extension, sleep hygiene, and safety measures to avoid injuries.
- Most parasomnias resolve by adulthood, but they can persist or coexist with another sleep disorder.
- If another sleep disorder is suspected, referral for vPSG is warranted.
- Stereotypic movements with repetitive, brief episodes can be suspicious of seizures and a neurology referral is recommended.

DISCLOSURE

The authors have nothing to disclose.

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