INTRODUCTION

Parasomnias are undesirable motor or verbal phenomena that arise from sleep or sleep-wake transition.1–4 Parasomnias may include abnormal movements, behaviors, sensory phenomena, emotions, and autonomic activity.2 They are classified into arousal disorders (usually associated with nonrapid eye movement [NREM] slow-wave sleep), sleepwalking, sleep terrors, and confusional arousals), typically occur during the first third of the night, and usually involve no memory or poor recall of the event by the patient. Rapid eye movement (REM) sleep parasomnias, including REM sleep behavior disorder, nightmares, and isolated sleep paralysis, occur out of stage REM sleep and usually involve some recollection of the event or dream content related to the motor activity by the patient. Management of parasomnias includes identifying and treating other underlying sleep disorders (such as sleep apnea, periodic leg movements of sleep), educating the patient regarding safety precautions in their home, and possibly pharmacologic treatment specific to the parasomnia or sleep-related motor activity.

KEYWORDS
- Parasomnias
- Confusional arousals
- Sleepwalking
- Night terrors
- Nightmares
- REM sleep behavior disorder
- Sleep paralysis

KEY POINTS
- Parasomnias are abnormal and undesirable motor or verbal events that manifest during sleep or wake-to-sleep transition.
- Features of the clinical history and examination, along with findings from the overnight polysomnogram, are essential to differentiate between different parasomnias and their mimics.
- Disorders of arousal (ie, sleepwalking, sleep terrors, and confusional arousals) arise from nonrapid eye movement sleep (usually stage N3), typically occur during the first third of the night, and usually involve no memory or poor recall of the event by the patient.
- Rapid eye movement (REM) sleep parasomnias, including REM sleep behavior disorder, nightmares, and isolated sleep paralysis, occur out of stage REM sleep and usually involve some recollection of the event or dream content related to the motor activity by the patient.
- Management of parasomnias includes identifying and treating other underlying sleep disorders (such as sleep apnea, periodic leg movements of sleep), educating the patient regarding safety precautions in their home, and possibly pharmacologic treatment specific to the parasomnia or sleep-related motor activity.
The International Classification of Sleep Disorders, Second Edition lists 15 categories of parasomnias divided into disorders of arousal from NREM sleep (confusional arousals, sleepwalking, sleep terrors), parasomnias associated with REM sleep (REM sleep behavior disorder [RBD], recurrent isolated sleep paralysis, and nightmare disorder) and other parasomnias (sleep enuresis, sleep-related eating disorder [SRED], and several others that are not addressed here). Table 1 presents the key features of the parasomnias discussed in this article with regards to sleep stage propensity, semiology, and suggested treatment.

**PATHOPHYSIOLOGY**

The current understanding of the cause of parasomnias is that sleep and wakefulness are not mutually exclusive states of being. As one falls asleep and progresses through the various sleep stages, sleep stage shift is not a complete on or off switch phenomenon, but involves reorganization and transition of several neuronal centers for an equivocal stage to declare itself. It is during this period of reorganization (a unique state of sleep dissociation) that one encounters an admixture of different states of being. They may overlap or intrude into one another, resulting in complex behaviors, as shown in Fig. 1. Disorders of arousal are a consequence of intrusion of wakefulness into NREM sleep. REM parasomnias such as RBD are a corollary of an admixture of wakefulness and REM sleep. Fig. 1 shows the interesting corollary in that both patients with narcolepsy and RBD experience abnormalities in REM sleep control: abnormal expression of increased muscle tone during REM sleep, when it is expected to be inhibited in RBD. As is shown in Fig. 1, hypnagogic hallucinations are abnormal REM-related visual disturbances that occur during transition from wakefulness into sleep.

<table>
<thead>
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<th>Table 1</th>
<th>Key similarities and differentiating features between NREM and REM parasomnias as well as nocturnal seizures</th>
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<td><strong>Confusional Arousals</strong></td>
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<td><strong>Duration (min)</strong></td>
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<td>+</td>
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<tr>
<td><strong>Organic CNS lesion</strong></td>
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</table>

Abbreviation: SWA, slow wave activity or N3 sleep.

An alternative hypothesis is that central pattern generators (CPGs), as discussed in Fig. 2, lead to deafferentation of the locomotor centers from the generators of the different sleep states. Locomotor centers are present at both spinal and supra spinal levels and this dissociation can explain motor activity or ambulation especially in patients with disorders of arousals.

CPGs, which are located in the brain stem and spinal cord (shown in the yellow regions in Fig. 2), are believed to be responsible for involuntary motor behaviors classified into:

a. Oroalimentary automatisms, bruxism, and biting;
b. Ambulatory behaviors, ranging from the classic bimanual-bipedal activity of somnambulism to periodic leg movements; and
c. Various sleep-related events associated with fear, such as sleep terrors, nightmares, and violent behaviors.

CLASSIFICATION OF PARASOMNIAS

The International Classification of Sleep Disorders classifies 15 different parasomnias based on the sleep stage during which the parasomnia is most likely to manifest (Fig. 3). NREM sleep parasomnias, also known as disorders of arousal (Box 1), typically arise from stage N3 sleep, although they can also occur out of stage N1 or N2.
They tend to occur during the first third of the night, when stage N3 predominates. They also occur more frequently in childhood because of the higher percentage of stage N3 at these ages, and decrease in frequency with age. Patients do not typically remember the event or have dream recall corresponding to the motor activity, which distinguishes them from REM sleep parasomnias. Motor activity can be simple, benign movements, such as sitting in bed or sleep talking, or can be complex and dangerous, such as walking, driving, fighting, or eating. NREM sleep parasomnias consist of confusional arousals, sleepwalking, and sleep terrors. Box 2 lists several known factors known to predispose patients to experience these events.

CONFUSIONAL AROUSALS

Confusional arousals consist of mental confusion or confusional behavior during or after arousals from sleep. They occur most often out of stage N3 sleep and during the first third of the night, but can occur out of other stages of NREM sleep and at any time of the night.
Epidemiology and Risk Factors

There is no sex difference with confusional arousals, and like other NREM sleep parasomnias, they are more common in children and young adults. Prevalence rate in children 3 to 13 years of age is 17.3%. In children older than 15 years and in adults, prevalence is as high as 6.9%.12

Pathophysiology

Exact pathophysiology and localization in the brain have not been confirmed. Areas of the brain in control of arousal, such as the posterior hypothalamus, midbrain reticular area, and the periventricular gray matter, have all been implicated. Confusional arousals may represent a more intense version of symptoms that are experienced when awakening from deep sleep: decreased cognition, reaction time, and attention.13

Box 1

Key features common to disorders of arousal

- Typically occur from stage N3 sleep
- Predilection for the first third of the night
- More common in children
- Decrease in frequency with age
- Amnestic or poor recall of the event
- Events have varying behavior, not stereotypic
**Key Clinical Features and Diagnosis**

Patients experience mental confusion and disorientation to time and space lasting minutes to hours. They have a blunted response to questions and are less interactive with their environment. They have poor memory of the event, with only rare dream recall. They may speak unintelligibly or may perform more complex motor tasks, possibly clumsy, incorrectly, or inappropriately. They can be difficult to fully awaken and may become violent if this is attempted. This parasomnia can be termed sleep drunkenness or morning sleep inertia.\textsuperscript{14} Patients may appear awake with their eyes open during the event.\textsuperscript{15}

One form of confusional arousal is sexsomnia or sleep sex, where on awakening, the motor activity is sexual in nature. This activity can involve masturbation, molestation, or sexual assault. There is no recollection of the event by the patient. In addition to potentially leading to psychological and physical consequences to their bed partner, sexsomnia may result in shame, guilt, or medicolegal problems for the patient.\textsuperscript{16}

Diagnosis is typically made clinically and can be confirmed with overnight, attended polysomnography (PSG). Home videorecording can also be helpful. Overnight PSG can be helpful by either capturing an episode (\textbf{Fig. 4}) or by evaluating for causes of sleep fragmentation or arousals that could trigger confusional arousals, such as sleep apnea (\textbf{Fig. 5}) or periodic leg movements. It is also useful to evaluate for other possible causes for the motor behavior, such as seizures or RBD. Even if a typical event is not captured during the overnight study, many of these patients have frequent spontaneous arousals from stage N3 sleep or slow-wave sleep fragmentation, suggesting sleep stage instability or a propensity for NREM sleep parasomnias such as confusional arousals (\textbf{Fig. 6}). The cyclic alternating pattern (CAP) also may play a role in causing disorders of arousal. CAP is a measure of NREM instability with a high level of arousal oscillation. Patients who have disorders of arousal are found to have increases in CAP rate, number of CAP cycles, and arousals with electroencephalographic (EEG) synchronization.

**Treatment**

Efforts to wake up the patient or direct the patient are not typically successful during an event. It is recommended to let the episode run its course and ensure that the

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**Box 2**

Precipitating factors for disorders of arousal

- Sleep deprivation
- Forced arousal from sleep (noise, full bladder, pain)
- Circadian rhythm disturbances
- Fever or infection
- Medications (central nervous system [CNS] depressants, psychotropics)
- Sleep-disordered breathing
- Periodic leg movements/restless legs syndrome
- Stress
- Menstrual cycle

Patient is not putting themselves or others in a dangerous situation. To try to prevent episodes from occurring, treating any underlying sleep disorder is important. Ensuring adequate duration of sleep and following good sleep habits are also helpful. Avoiding CNS depressants, alcohol, or other triggers may be necessary. If pharmacologic therapy is warranted, tricyclic antidepressants (TCAs) (clomipramine 25–100 mg at bedtime) or benzodiazepines (clonazepam 0.5–2 mg at bedtime) can be initiated.17

Prognosis

In most cases, children outgrow confusional arousals. Many develop sleepwalking. If an underlying cause, such as sleep apnea, is treated or other triggers identified, confusional arousals decrease or disappear.

SLEEPWALKING

Sleepwalking is an arousal disorder culminating in walking around in an altered state with impaired judgment. Sleepwalking occurs most often out of stage N3 sleep and during the first third of the night, but can occur out of other stages of NREM sleep and at any time of the night.
Epidemiology and Risk Factors

No gender difference exists. Prevalence in childhood is as high as 17%, with a peak age of 12 years. Sleepwalking typically decreases in frequency until adulthood, and 3% of adults sleepwalk.14 Some of the adult sleepwalkers begin to have the condition after childhood (adult-onset sleepwalking). It does seem to run in families, suggesting a genetic component. About 80% of somnambulistic patients have at least 1 family member affected by this parasomnia, and the prevalence of somnambulism is higher in children of parents with a history of sleepwalking.14,18 A positive association with the HLA-DQB1*05 subtype was found in sleepwalking patients.19

Pathophysiology

The pathophysiology of sleepwalking is unknown. The brain is unable to fully awaken from slow-wave sleep. There seems to be incomplete cortical activation in response to an arousing stimulus. One study used transcranial magnetic stimulation to examine brain function in adult sleepwalkers. The study found alterations in sleepwalkers consistent with impaired efficiency of inhibitory circuits during wakefulness, possibly signifying immaturity of neural circuits, synapses, or receptors.20 Sleepwalkers have
difficulties maintaining consolidated slow-wave sleep. NREM sleep stage instability, or other sleep disorders such as sleep apnea, periodic leg movements, or restless legs syndrome, commonly cause awakenings, contributing to the development of sleepwalking.

**Key Clinical Features and Diagnosis**

Episodes usually occur from stage N3 sleep, but may occur out of any NREM sleep stage. They begin abruptly, with the patient having a blank expression with their eyes open. They may appear to be awake, but have decreased awareness of their surroundings and little reactivity. While ambulating, movements are clumsy and slow. Sleep talking or even conversations may be heard. It can be difficult to awaken the patient, and they may react violently to such attempts. At other times, they may listen to instructions and return to bed. They are usually amnestic of the event on awakening in the morning, although they may remember portions of the event.

Dreaming is sometimes reported in sleepwalkers, making it difficult to distinguish from RBD. Complex motor activities that have been reported in this state can vary, including driving, cooking, eating, and playing musical instruments.

Diagnosis is usually made clinically, and an overnight sleep study is indicated only in specific cases (Box 3). Video, arm leads, and additional EEG leads can be added...
during the study to improve chances of correctly identifying the parasomnia. Although full 18-channel EEG montage had best sensitivity at detecting seizures, a limited 7-channel or 8-channel montage with added temporal leads was superior to a traditional 4-EEG-channel sleep study (central and occipital leads), which had been routinely used in the past. When an overnight study is performed, it is unusual for a sleepwalking episode to be captured in the laboratory. However, the sleep study can be helpful in identifying underlying sleep disorders that may contribute to sleep fragmentation, such as sleep apnea or periodic leg movements. It may also reveal sleep stage instability, with frequent stage shifts or frequent spontaneous arousals from stage N3 sleep (see Fig. 6). If an event is captured on sleep study, it can show diffuse rhythmical $\delta$, diffuse $\delta$ with intermixed faster frequencies ($\theta$ and $\alpha$), or may be difficult to interpret because of motor artifact.

Treatment

Patients should be instructed to avoid the known triggers (reduce arousals) and any underlying sleep disorders (ie, sleep apnea, periodic leg movement disorder) should be treated. Safety measures and precautions should be put in place (Box 4). Often, reassurance is all that is required because sleepwalking may decrease in frequency and resolve in many patients as they get older. Medical treatment may be required if there is fear of injury to the patient or someone else, or if behaviors are disruptive to the family. Treatments include diazepam (2–5 mg) or clonazepam (0.5–2 mg) at bedtime. Trazodone and selective serotonin reuptake inhibitors (SSRIs) are also reported to help in some cases.

Prognosis

Sleepwalking decreases with age, which may be because of decreased amounts of slow-wave sleep as we get older, or may be because of full maturation of the brain and its sleep-controlling centers.
Sleep terrors, also called night terrors, are sudden arousals from NREM sleep associated with intense autonomic and motor symptoms, such as crying or screaming. These events can be dramatic and disturbing to the family, yet the patient may be unfazed by the events.

**Epidemiology and Risk Factors**

There is no gender difference, but they are more common in children, occurring in up to 6.5%. Prevalence in adults is approximately 2%.\(^{14}\) Obstructive sleep apnea and other sleep disorders can lead to awakenings from sleep and sleep terrors. Genetic factors play a role, as they do in other disorders of arousal.

**Pathophysiology**

Pathophysiology is unknown, but is believed to involve mechanisms that control sleep state stability, as in other disorders of arousal.

**Clinical Features and Diagnosis**

As depicted in Fig. 7, on awakening from sleep, patients with sleep terrors have intense autonomic discharge: tachycardia, tachypnea, flushing, diaphoresis, mydriasis, and increased muscle tone. In addition, there is typically screaming or crying initially on awakening and sitting up in bed. The patient does not respond normally and has amnesia for the episode. There can be brief dream fragments or vivid images, which they may remember. During an episode, the person is inconsolable and can be difficult to arouse.\(^{27}\)

![Fig. 7. Characteristic pattern of sleep terror. Sleep terrors are characterized by a sudden arousal associated with a scream, agitation, panic, and heightened autonomic activity (star). Inconsolability is almost universal. The child is incoherent and has altered perception of the environment, appearing confused. This behavior may be dangerous and could result in injury. (Modified after Avidan, AY and N Kaplish. The parasomnias: epidemiology, clinical features, and diagnostic approach. Clin Chest Med 2010;31(2):361; with permission.)](image-url)
Diagnosis is made clinically. However, a sleep study may be necessary to evaluate for causes of sleep fragmentation that may lead to sleep terrors, such as sleep apnea or periodic leg movements. A sleep study also helps evaluate for other parasomnias that are on the differential diagnosis such as RBD and seizures. Slow-wave sleep instability with spontaneous arousals is also noted in patients suffering from sleep terrors, as they are in other disorders of arousal. As shown in Fig. 8, during a sleep terror, EEG shows δ slowing, and there is an increase in muscle tone, with an increase in respiratory and heart rate.

Differentiation of sleep terrors from an easily confused, but semiologically and sleep stage different parasomnia (REM nightmare) is shown in Table 2.

**Treatment**

Safety precautions, similar to the ones advised in sleepwalking, are important to avoid injuries to the patient or others. Attempts to wake the patient should be avoided because of the possibility of upsetting or confusing them further. The patient should avoid any known triggers for sleep terrors, and underlying sleep disorders should

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**Fig. 8.** Two-minute minute epoch of a diagnostic PSG from a 9-year-old boy performed to evaluate arousals associated with screaming and inconsolable crying. The figure shows one of the patient’s representative spells showing an arousal with screaming arising out of slow-wave sleep with the patient’s arms flexed and held close to the chest (as if afraid and protecting himself). Channels are as follows: electro-oculogram (left, E1-M2; right, E2-M1), chin electromyogram (Chin1-chin2), electroencephalogram (right: frontal, F4; central, C4; occipital, O2; right mastoid, M2) 2 ECG channels, 2 limb EMG (LAT, RAT), snore channel, nasal-oral airflow (N/O), nasal pressure signal (NPRE), respiratory effort (thoracic [THOR], abdominal [ABD]) and oxygen saturation (SaO2). Modified after Avidan, AY and N Kaplish. The parasomnias: epidemiology, clinical features, and diagnostic approach. Clin Chest Med 2010;31(2): 358; Polysomnogram slide courtesy of Timothy Hoban, MD, Professor of Pediatrics and Neurology, University of Michigan, Ann Arbor, Michigan.
be treated. In most cases, these instructions along with reassurance are all that is necessary because the patient will likely outgrow the sleep terrors. If risk of injury or disruption is sufficient to require treatment, medications that have been shown to be effective include benzodiazepines and imipramine. There have also been reports of trazadone and paroxetine being helpful.29

Scheduled awakenings may also be useful if patients that have them at highly predictable times of the night. Parents or family members are instructed to wake the patient 30 minutes before the time that they have their sleep terrors. The patient should be awoken only briefly until their eyes are open or they vocalize, and then put back to sleep.29 Psychotherapy has also been helpful in patients who suffer from psychopathology.

**Prognosis**

Sleep terrors typically decrease in frequency as the child grows older, either because of the decreased total sleep time in stage N3 sleep, or because of maturation of the brain.

**OTHER PARASOMNIAS**

**SRED**

SRED consists of recurrent episodes of involuntary eating occurring during the main sleep period. This disorder typically involves eating peculiar forms or combinations of food, or possibly dangerous or toxic substances. Like disorders of arousal, most patients do not have full recall of the event. Eating can occur multiple times in 1 night, typically with high caloric foods. Foods may be prepared and cooked, although usually in a sloppy or inappropriate manner.5

SRED is commonly associated with sleepwalking, and many have had sleepwalking during childhood. The pathophysiology seems to be similar to the disorders of arousal. Other sleep disorders may also trigger SRED, including obstructive sleep apnea, restless legs syndrome, periodic leg movement disorder, and circadian rhythm disorders. Use of hypnotics along with cessation of smoking or alcohol have also been reported as triggers.30 There is a female predominance, with the mean age of onset at 22 to 29 years of age. SRED can be distinguished from night eating syndrome (NES), which is characterized by full recall of the eating and absence of bizarre or toxic ingestion. Many have also suggested that SRED and NES may exist along a common spectrum and have many overlapping features.31

Treatment includes appropriate management of any underlying sleep disorder. If the patient suffers from other disorders of arousal, recommended treatments for these

<table>
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<tr>
<th>Characteristic</th>
<th>Sleep Terror</th>
<th>Nightmare</th>
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<tr>
<td>Timing during the night</td>
<td>First third (deep slow-wave sleep)</td>
<td>Last third (REM sleep)</td>
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<tr>
<td>Movements</td>
<td>Common</td>
<td>Rare</td>
</tr>
<tr>
<td>Severity</td>
<td>Severe</td>
<td>Mild</td>
</tr>
<tr>
<td>Vocalizations</td>
<td>Common</td>
<td>Rare</td>
</tr>
<tr>
<td>Autonomic discharge</td>
<td>Severe and intense</td>
<td>Mild</td>
</tr>
<tr>
<td>Amnesia</td>
<td>Absent</td>
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<tr>
<td>State on waking</td>
<td>Confused/disoriented</td>
<td>Function well</td>
</tr>
<tr>
<td>Injuries</td>
<td>Common</td>
<td>Rare</td>
</tr>
<tr>
<td>Violence</td>
<td>Common</td>
<td>Rare</td>
</tr>
<tr>
<td>Displacement from bed</td>
<td>Common</td>
<td>Very rare</td>
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conditions can help reduce SRED (benzodiazepines and TCAs). Dopamine agonists, SSRIs, and topiramate have all been reported to help improve symptoms, although they lack the support of large trials.\textsuperscript{32,33}

**Catathrenia**

Catathrenia, also called sleep-related groaning, is characterized by expiratory groaning during sleep. Events usually occur nightly and during REM sleep, predominantly in the second half of the night. The sound occurs solely during expiration and sounds like groaning, although the patient does not show emotion or respiratory distress and does not recollect the event. The complaint is usually reported by a bed partner or the affected person may notice hoarseness on awakening. No otolaryngologic or vocal cord abnormalities are found in these cases. There have been some reports of successful use of continuous positive airway pressure (CPAP) in patients with sleep apnea and this condition.\textsuperscript{34,35} This is a rare condition with a prevalence of less than 1%, mostly made up of males. There have also been reports of catathrenia secondary to use of sodium oxybate in patients with narcolepsy.\textsuperscript{36}

On PSG, as shown in the example in Fig. 9, recording sound is required for identifying this condition. The episode generally lasts 2 to 49 seconds, appearing in clusters.
many times throughout the night during REM sleep. A sudden change in respiratory rhythm is seen in the respiratory channels, usually inspiration followed by a flat line during expiration, ending in arousal on EEG. It is important to distinguish this condition from a central apnea, which it closely resembles, except for the lack of inspiratory signal. No abnormal EEG activity should accompany the vocalizations.15

Dissociative Disorder

Dissociative disorders can occur from sleep during transitions between sleep and wake, or within several minutes from awakening from sleep after well-established EEG wakefulness. According to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, dissociative disorders are a disruption in the usually integrated functions of consciousness, memory, identity, or perception of the environment. Most patients with sleep-related dissociative disorders also have daytime events and have past history of abuse or psychiatric disease. The condition is more common in females. This condition has also been called pseudoparasomnia.37

During an event, patients may scream, run, or engage in violent behaviors lasting minutes to hours, typically longer than disorders of arousal. They may reenact previous sexual or physical abuse situations. The individual is usually amnestic of the event.

PSG shows EEG wakefulness present before, during, and after the episodes if captured during testing. If an event occurs after an arousal from sleep, there must be at least 15 seconds of wakefulness before the activity begins to be consistent with this condition, because many disorders of arousal occur immediately from sleep and have α frequency on EEG during the episode. Treatment typically consists of psychotherapy along with management of other underlying psychiatric disorders such as depression and anxiety.

Exploding Head Syndrome

Exploding head syndrome, also sometimes included in the group of sensory sleep starts, is characterized by a sudden loud noise or sense of explosion in the head either at wake-sleep transition or on waking during the night. The noise can be a loud bang, explosion, or a crash of cymbals, but sometimes less drastic. Patients are typically frightened by their symptoms and concerned about an impending serious health problem. Myoclonic jerks and flashes of light may accompany the sound. They occur more commonly in women, with the median age of onset of 58 years of age. Epidemiology and pathophysiology are unknown, although they are believed to represent a variant of sleep starts.38 The condition is rarely treated because of its benign nature, but clomipramine, nifedipine, and topiramate have been used.39,40

Other Motor Activity During Sleep

Sleep-related movement disorders can sometimes mimic parasomnias. They are different from parasomnias because they are simple, usually stereotyped movements that disturb sleep. One type of sleep-related movement disorder is sleep-related rhythmical movement disorder. In this condition, patients show repetitive, stereotyped, and rhythmical motor behaviors either during sleep or near a nap or bedtime. The behaviors include body rocking, head banging, or head rolling. As shown in Fig. 10, the frequency is usually 0.5 to 2 per second, generally lasting less than 15 minutes. The patient may stop the activity if spoken to or distracted. These behaviors mainly occur in infants and children, or adults with developmental delay or other neurologic or psychiatric diseases.15 No treatment is typically necessary, with the exception of safety precautions in the bedroom if the motor activity could cause
harm to the patient. If treatment is necessary, behavioral therapy, hypnosis, and rarely pharmacologic therapy with benzodiazepines have been reported to effective.

Sleep starts, or hypnic jerks, can also mimic parasomnias. These are sudden, brief, simultaneous contractions of the body occurring at sleep onset. They are sometimes associated with a subjective feeling of falling, sensory flash, or sleep-related hallucinations. Sleep starts are common in all ages and sexes. Excessive stimulant use, psychological stress, and sleep deprivation can all increase the frequency of sleep starts. Patients can be reassured regarding the benign nature of sleep starts and do not need medication therapy.

REM PARASOMNIAS

Nightmares

Epidemiology and risk factors
About 10% to 50% of children are affected by nightmares, and up to 75% of the population can remember at least 1 or a few nightmares in the course of their childhood. Prevalence of frequent nightmares, as defined by at least once per week, was 5.1% based on a large community-based cohort of middle-aged Hong Kong Chinese individuals. The frequency of nightmare experiences increases in patients diagnosed with posttraumatic stress disorder, those undergoing medical procedures, and in those with psychological stress caused by major catastrophic events.

Key clinical features and diagnosis
Nightmares manifest as a prolonged and vivid dream pattern that progressively becomes more complex and frightening to the sufferer, terminating in an arousal and vivid recall. Episodes may increase during times of stress, particularly after traumatic events. Certain medications such as β-adrenergic blockers, L-dopa, acetyl

Fig. 10. A 60-second PSG epoch showing unilateral rhythmical movement disorder (star) in a patient with untreated obstructive sleep apnea. Channels are as follows: electro-oculogram (left, E1-M2; right, E2-M1), chin electromyogram (Chin1-chin2), electroencephalogram (right: frontal, F4; central, C4; occipital, O2; right mastoid, M2) 2 ECG channels, 2 limb EMG (LAT, RAT), snore channel, nasal-oral airflow (N/O), nasal pressure signal (NPRE), respiratory effort (thoracic [THOR], abdominal [ABD]) and oxygen saturation (SaO2).
cholinesterase inhibitors, and abrupt discontinuation of REM-suppressant medications may induce nightmares. The PSG shows a sudden arousal pattern from REM sleep associated with an increased REM sleep density and variability in heart and respiratory rates.

**Treatment**
Reassurance is often the only management necessary. However, if offending agents are present, these medications may need to be changed, but for severe and refractory cases, the use of an REM-suppressing agent such as TCAs or SSRIs may be needed. Image rehearsal therapy and systematic desensitization and progressive deep muscle relaxation training have been categorized as level A by a recent American Academy of Sleep Medicine best practice guideline.

**Recurrent Isolated Sleep Paralysis**
Sleep paralysis is the inability to move on awakening and corresponds to lack of voluntary motor function at sleep onset or on awakening.

**Epidemiology and risk factors**
The episodes may occur at least once in a lifetime in as many as 40% to 50% of normal individuals.

**Key clinical features and diagnosis**
Sufferers recall a frightening arousal, during which they experience paralysis of skeletal muscles, with the possible exception of respiratory and extraocular movements, although cognition remains intact. Episodes generally last a few minutes and improve spontaneously or on external stimulation. Predisposing features include acute and chronic sleep deprivation and underlying circadian rhythm disturbances, such as jet lag and shift work disorder.

**Pathophysiology**
The underlying pathophysiology may be related to abnormalities in the mechanism controlling REM sleep muscle atonia.

**Treatment**
Treatment of sleep paralysis is mainly in the form of reassurance when episodes are infrequent. An example of conservative management is avoidance of an irregular sleep-wake schedule. However, when sleep paralysis is severe, the use of anxiolytic medications and antidepressants such as fluoxetine (as well as other REM-suppressing agents) may be helpful.

**RBD**

**Epidemiology and risk factors**
The prevalence of RBD is estimated to be 0.5% of the population. The disorder has a unique gender predilection, affecting male gender by a factor of 9, and has a higher prevalence in patients older than 50 years. Subjective data indicate that as many as 25% of patients with parkinsonism have abnormal dream enactment behaviors suggestive of RBD, whereas formal PSG reports RBD in as many as 47% of patients with Parkinson disease who experience sleep disturbances.

**Key clinical features and diagnosis**
The disorder is uniquely characterized by abnormal elevation of chin or limb muscle tone during REM sleep (Fig. 11) and by corresponding complex motor activity associated with elaborate dream enactment, which corresponds with the dream sequence.
Patients show a spectrum of abnormal dreams, mainly unpleasant and negative themes in which they need to protect themselves. The range of experiences consists of simple verbalizations to singing, yelling, shouting, and screaming to more complex motor phenomena such as walking, running, punching, kicking, jumping, and often agitated and violent behaviors synchronizing with the dream imagery. Often, it is injury to self or bed partner that brings the patient to the attention of the clinician. These complex and polymorphic motor phenomena, which are distinguished from the more stereotyped monomorphic nocturnal seizures, are associated with emotionally charged utterances.50–53 When awoken from an episode, some patients may have vivid recall and report dream mentation, which correlated with the observed behavior.

The case study in Box 5 shows a patient with dream enactment behavior and excessive abnormal limb movements that parallel the aggressive dream content. The PSG epoch in Fig. 11 shows the classic electrographic correlation of the behaviors enacted during dreaming highlighted by the arrows showing excessive chin (Chin1-chin2) and excessive anterior tibialis leg (LAT1-LAT2) electromyographic tone. Antecedal reports of sleep talking, yelling, or limb jerking may be present. With time, the dream content has the potential to become more violent, complex, action-filled, and unpleasant,
coinciding with the onset of RBD. If RBD leads to frequent arousals, sleep becomes fragmented, leading to other symptoms such as hypersomnolence. Potential for injury, including facial ecchymosis, skin lacerations, and skull fractures to patient or bed partner, is a major safety concern and warrants immediate and effective pharmacologic intervention.

RBD may be classified into an acute and a chronic form:

1. The acute form of RBD may be seen in the context of medication-related or substance-related, toxic, or metabolic derangements. The most common drug-related and substance-related form includes rapid withdrawal from alcohol, abrupt discontinuation of sedative-hypnotic agents (inducing REM rebound), and examples related to SSRIs, TCAs, monoamine oxidase inhibitors (MAOIs), biperiden, and cholinergic medications. Some reports also implicate excessive caffeine consumption (chocolate) as a cause. Acute neurologic disorders such as brainstem lesions caused by pontine stroke, multiple sclerosis, subarachnoid hemorrhage, and brainstem neoplasm have all been implicated in the acute form of RBD.

2. The chronic form of RBD is typically associated with advanced age as a predisposing factor. This form is idiopathic, generally more frequent, has an onset later in adulthood, progresses over time, and tends to stabilize.

Of the chronic form, about 60% of the cases are idiopathic; the remaining 40% of the cases are associated with underlying neurodegenerative disorders. A spectrum of dementias are implicated in RBD and include the synucleinopathies such as olivopontocerebellar atrophy and diffuse Lewy body disease, with a characteristic α-synuclein inclusion in the nerve cell bodies.

RBD typically begins after age 60 years and may precede clinical manifestation of the underlying neuropathologic lesion process by more than a 10 years. Patients with narcolepsy experience a higher incidence of RBD, and presence of RBD in children may indicate the potential onset of evolving narcolepsy. In addition, in patients with narcolepsy, psychiatric medications such as TCAs, SSRIs and MAOIs, which can be used to treat cataplexy, can sometimes trigger or exacerbate RBD in this cohort. As alluded to earlier, RBD is more predominant in men than in women (by a factor of 9), and the reason for this gender predilection is unclear. Recent data suggest that RBD may be the first sign of childhood narcolepsy in patients with a positive HLA-DQB1 *0602 positive, female gender, in whom cerebrospinal fluid hypocretin level (Hcrt-1) was extremely low.

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**Box 5**

**Case study**

An 80-year-old man presented with violent dreams reported by his wife of 55 years. He had been noted to move a lot in bed for the past 3 years, but had begun to show aggressive and swift movements against a presumed intruder in the last few months. His wife brought a tape-recorded message, which revealed screaming at a supposed intruder saying “You must leave, get out, move away!” A sample of the patient’s PSG after presenting to the sleep disorders clinic is shown in Fig. 11.

Patients who experience RBD most often experience their spells as soon as they enter REM sleep, which is as early as 90 minutes after sleep onset, but more commonly during the latter half of the night. The spells vary in frequency, from infrequent (ie, once a month) to as frequent as nightly episodes, which leads to significant sleep disruption and more likely results in a referral to the specialist.
**Pathophysiology**

RBD is a complex sleep phenomenon with a possible mechanism related to either a reduction of REM atonia or an abnormal augmentation of locomotor intermittent excitatory influences during REM sleep, or both.\(^5\),\(^6\)

**Fig. 11** shows the underlying pathophysiologic mechanism for RBD, which is proposed to be related to abnormal brainstem control of medullary inhibitory regions. An identical syndrome was reported in cats by the French investigator Jouvet,\(^7\) who experimentally induced bilateral lesions of pontine regions adjacent to the locus coeruleus, inducing absence of the REM-related atonia associated with REM sleep and abnormal motor behaviors during REM sleep. In this experimental model, the animal slept until its first REM sleep episode, during which it jumped, with eyes still closed, and ran around the cage making attack motions. In 1986, Schenck and colleagues\(^8\) described RBD in a new category of sleep disorders, reporting on a series of older patients, mainly men, who presented with aggressive sleep-related behaviors.

Data deduced from single-photon emission computed tomography (SPECT) neuroimaging reveal a possible mechanism relating to abnormalities in dopaminergic systems showing decreased striatal dopaminergic innervation as well as reduced striatal dopamine transporters.\(^9,\)\(^10,\)\(^11\) In patients with multiple system atrophy and RBD, positron emission tomography as well as SPECT studies indicate reduced nigrostriatal dopaminergic projections.\(^12\) In patients with idiopathic RBD, impaired cortical activation as determined by EEG spectral analysis supports the relationship between RBD and neurodegenerative disorders.\(^13\) **Fig. 12** shows the underlying pathophysiologic mechanism in RBD.

**Diagnostic evaluation**

PSG reveals abnormal muscle augmentation during REM sleep in excess of the normal REM sleep-related phasic electromyography (EMG) twitches (see **Fig. 11**). The results of the neurologic history and examination may indicate the need for other neuroimaging, looking for structural lesion of underlying neurodegenerative processes. This strategy is especially important if the episodes are acute, follow a neurologic insult, and occur in an otherwise younger patients.\(^6,\)\(^9,\)\(^10\)

**Differential diagnosis**

The differential diagnosis of RBD includes nocturnal frontal lobe seizures, confusional arousals, sleepwalking, sleep terrors, posttraumatic stress disorder, and nightmares. Patients with RBD are often distinguished based on the complex nonstereotypic nature of their episodes, timing later in the night when REM density is highest, and the characteristic patients who are older men.

**Treatment**

Patients with RBD should be assessed carefully, with meticulous attention to risk for injury during the nocturnal episodes. Environmental safety (level A evidence) is cornerstone and prudent in every patient with likely RBD, especially in those who experience displacement from bed and aggressive spells. Suggested level of pharmacotherapy for RBD is based on clonazepam and melatonin. The former is prescribed in dosages ranging from 0.25 mg to 1 mg by mouth every night at bedtime, achieving improvement in most (90%) patients, with little evidence of tolerance or abuse.\(^1,\)\(^9,\)\(^12\) Although clonazepam does not normalize abnormal limb EMG tone during the night, it acts to prevent the arousals associated with the REM sleep disassociation. Treatment of RBD with melatonin may restore REM sleep atonia and was effective in 87% of patients taking 3 to 12 mg at bedtime.\(^9,\)\(^13-\)\(^15\) Melatonin, a dietary supplement,
is not approved by the US Food and Drug Administration (FDA), has poor regulation in terms of pharmacologic preparation, and side effects have not been widely studied. Other agents that may be helpful for RBD include imipramine (25 mg by mouth every night at bedtime), carbamazepine (100 mg, by mouth 3 times a day) as well as pramipexole or levodopa.96–98 One recent study described successful amelioration of RBD with sodium oxybate when other treatments are ineffective or poorly tolerated.99 This finding also suggests that RBD and cataplexy may share a common pathophysiology.99 Recent data from the Minnesota group100 suggest the use of an innovative alarm to reduce episodes of RBD in those who may be refractory to traditional pharmacologic agents. Table 3 summarizes the level of evidence for treating RBD.

**Differentiating seizures and parasomnias**

Seizures occurring during sleep can closely resemble parasomnias and can pose a diagnostic challenge to the clinician. Nocturnal frontal lobe epilepsy (NFLE) is
Table 3  
Evidence-based pharmacotherapy for RBD

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Level of Recommendation</th>
<th>Special Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clonazepam</td>
<td>0.25–4.0 mg before bedtime (usual recommended dose is 0.5–2.0 mg)</td>
<td>Suggested&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Use with caution in patients with dementia, gait disorders, or concomitant obstructive sleep apnea. Side effects include sedation, impotence, motor incoordination, confusion, and memory dysfunction.</td>
</tr>
<tr>
<td>Melatonin</td>
<td>3 mg–12 mg before bedtime</td>
<td>Suggested&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Effective in patients with α-synucleinopathies, memory problems, and sleep-disordered breathing. Side effects include headaches, sleepiness and delusions/hallucinations.</td>
</tr>
<tr>
<td>Zopiclone</td>
<td>3.5–7.5 mg before bedtime</td>
<td>May be considered&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Side effects include rash and nausea.</td>
</tr>
<tr>
<td>Yi-Gan San</td>
<td>2.5 gm 3 tid</td>
<td>May be considered&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Studied mainly on patients who could not take clonazepam. No side effects were reported when used for the treatment of RBD.</td>
</tr>
<tr>
<td>Sodium oxybate</td>
<td>Unknown</td>
<td>May be considered&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
</tr>
<tr>
<td>Donepezil</td>
<td>10–15 mg</td>
<td>May be considered&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
</tr>
<tr>
<td>Rivastigmine</td>
<td>4.5–6 mg bid</td>
<td>May be considered&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Studied mainly on patients with dementia of Lewy body.</td>
</tr>
<tr>
<td>Temazepam</td>
<td>10 mg</td>
<td>May be considered&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
</tr>
<tr>
<td>Alprazolam</td>
<td>1–3 mg</td>
<td>May be considered&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
</tr>
<tr>
<td>Desipramine</td>
<td>50 mg every night at bedtime</td>
<td>May be considered&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
</tr>
<tr>
<td>Carbamazepine</td>
<td>500–1500 mg every day at bedtime</td>
<td>May be considered&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> Not FDA approved for the treatment of RBD.

<sup>b</sup> Supported by sparse high-grade evidence data, or a substantial amount of low-grade data or clinical consensus.

<sup>c</sup> Supported by low-grade data.


a good example of this because of its sudden, brief nature of predominantly stereotypic motor activity, which may occur only during sleep and may be missed easily on EEG. Table 4 shows the distinguishing and common clinical features of NFLE versus NREM and REM sleep parasomnias.\textsuperscript{101,102}
SUMMARY

It is essential for the clinician to understand how to differentiate between each parasomnia or look alikes using features from the history, physical examination, or the overnight sleep study. Historical features such as time of night, age of patient, and recollection of the event are helpful in making the diagnosis. Seizures, rhythmic movement disorder, and dissociative states should always be considered in the differential diagnosis. Sleep study findings may help identify the parasomnia or may uncover underlying sleep disorders that could be triggering the episodes.

Management includes educating the patient and bed partner about safety precautions as well as treating any underlying sleep disorder. Pharmacologic treatment may be necessary in some cases, although the risks of the medications should be considered when making this decision.

REFERENCES


