Parasomnias are a group of disorders exclusive to sleep and wake-to-sleep transition that encompass arousals with abnormal motor, behavioral, or sensory experiences. Sensory experiences often involve but are not limited to perceptions, dreamlike hallucinatory experiences, and autonomic symptoms. When accompanied with excessive motor activity and other complex motor behaviors, these parasomnias can be disruptive to the patient and bed partners. Motor behaviors may or may not be restricted to bed but can become dangerous when the subject ambulates or is agitated. In some parasomnias, it may be injury or concerns for physical injury to the patient or bed partner that brings them to the attention of physicians. The other presentations include disrupted nocturnal sleep of patients, bed partners, or family members sharing the sleeping quadrant. The behaviors are inappropriate for the time of occurrence but may seem purposeful or goal directed. In general, most parasomnias are more common in children and decrease in frequency as they get older. Parasomnias have been reported in approximately 4% of the adult population.\(^7\)

These complex motor behaviors occurring during sleep may have medicolegal implications, as violence could be a prominent component as documented in the case of Canadian Supreme Court case *Her Majesty the Queen v Kenneth Parks* and in the State Supreme Court case *State of Arizona v Scott Falater*. The incidence of violent behavior during sleep is generally presumed to be low, but recent reports indicate a prevalence of up to 2% in adults. Sexual differences have also been noted in other parasomnias such as rapid eye movement (REM) sleep behavior disorder (RBD) and sleep-related eating disorder. Complex behaviors with sexual acts have been implicated in cases of sexual assault and rape.

**PATHOPHYSIOLOGY**

Sleep can be broadly divided into non-REM (NREM) and REM sleep. NREM sleep is further divided into stage N1, stage N2, and stage N3 (slow wave sleep). Sleep stage shift is not a complete on-off switch phenomenon, but involves reorganization and transition of various neuronal centers for an equivocal stage to declare itself.\(^1,2\) During this period of reorganization (a unique state of sleep dissociation) an admixture of 2 or 3 different states of being is observed. Fig. 1 depicts the conceptualization of the overlapping states of being leading to parasomnias.\(^9\) It is usually an arousal during these periods of
reorganization that leads to complex motor behavior during sleep.2,10

Another hypothesis is the deafferentation of the locomotor centers from the generators of the different sleep states. Locomotor centers are present at spinal and supraspinal levels and this dissociation can explain motor activity or ambulation, especially in patients with disorders of arousals.11

Central pattern generators, which are located in the brain stem and spinal cord, 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.11

CLASSIFICATION OF PARASOMNIAS

NREM Parasomnias

The International Classification of Sleep Disorders 2nd Edition (ICSD-II) categorizes NREM parasomnias (disorders of arousals) into 3 broad categories

(Fig. 2): confusional arousals, sleepwalking, and night terrors. These share several common features (such as having increased predilection in children, decreasing with age, and occurring in the first half of the night, typically within the first 2 hours of sleep) but have certain unique features: inconsolable crying and autonomic hyperactivity in night terrors and ambulation in sleep terrors, which helps differentiate them.

Confusional Arousals

Case history: A 56-year-old man with history of traumatic brain injury and noncompliance with positive airway pressure therapy for his sleep-disordered breathing, began experiencing nocturnal spells as frequently as multiple times per night, primarily during the first half of the night. These spells were characterized by sudden arousals associated with confusion and singing behavior.

During the diagnostic nocturnal polysomnogram (PSG) video recording (Fig. 3), multiple similar spells of arousals with confusion, along with side-to-side head movements, arm flapping, and talking occurred exclusively from NREM sleep, and in 1 event he was reported by the sleep technicians to be “quacking like a duck.”

Fig. 1. Overlapping states of being. Parasomnias are explainable on the basic notion that sleep and wakefulness are not mutually exclusive states but may dissociate and oscillate rapidly. The abnormal admixture of the 3 states of being (NREM sleep, REM sleep, and wakefulness) may overlap, giving rise to parasomnias. REM parasomnias occur because of the abnormal intrusion of wakefulness into REM sleep and likewise NREM parasomnias such as sleepwalking occur because of abnormal intrusions of wakefulness into NREM sleep. Other nocturnal spells that may be confused with parasomnias include NFLE and psychogenic spells such as posttraumatic stress disorder (PTSD), dissociated disorders. (Modified from Mahowald MW, Schenck CH. Non-rapid eye movement sleep parasomnias. Neurol Clin 2005;23(4):1078, vii; with permission.)
Approximately 20 of these nocturnal episodes were recorded with and without preceding respiratory effort related arousal. No epileptiform activity was recorded on the limited electroencephalogram (EEG) recording during these episodes. The events were clinically suspected of being consistent with the diagnosis of confusional arousals and were resolved with the application of continuous positive airway pressure during the titration phase of the study. This suggested that sleep-disordered breathing was probably a precipitating factor for the patient’s nocturnal events.

Confusional arousals (also known as sleep drunkenness or excessive sleep inertia) consist of arousals originating from NREM sleep (usually slow wave sleep) associated with an arousal linked to confusion and disorientation. The associated behaviors may be inappropriate and patients have slow mentation in responding to questioning from the observer. Usually these behaviors are noted in the first half of the night but forced and anticipatory awakenings during slow wave sleep may result in confusional arousal induction. Aggression or violent behavior is atypical, but may follow forced awakening from sleep. Confusional arousals are associated with amnesia of the event, and recollection in the morning is absent or sketchy. Associated motor behavior may be simple and nongoal oriented and, less commonly, complex and associated with aggression, violence, or inappropriate sexual activity. When sexual behavior is encountered along the spectrum of confusional arousals, the parasomnia is further defined as a sexsomnia.

There is no sexual predilection in confusional arousals. High prevalence is observed at a prevalence of 17.3% in children 3 to 13 years of age, after which the prevalence decreases. Prevalence among adults 15 years and older is estimated at 2.9% to 4.2%. Genetic factors play a significant predisposing role, as there may be a familial history of similar childhood nocturnal behaviors. In adults, a variety of factors that lead to arousals from sleep have the potential to precipitate confusional arousals. These factors include sleep deprivation, fever, infections, centrally active medications (hypnotics, antidepressants, and tranquilizers), sleep-disordered breathing, and periodic limb movements of sleep.

**Confusional arousal variants**

ICSD-II describes 2 variants of confusional arousals in adults and adolescents: sleep-related sexual behaviors and severe morning sleep inertia.

- Abnormal sexual behavior has been reported to occur during sleep and is now classified in ICSD-II as a variant of confusional arousals in adults and adolescents. In 2002 Guilleminault and colleagues described 11 subjects (7 men and 4 women) with atypical sexual behaviors during sleep. The range of abnormal

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**Fig. 2.** ICSD-II classification of parasomnias showing all 11 of the 15 important parasomnias. Disorders from NREM sleep are also known as disorders of arousal. Parasomnias categorized as “other parasomnias” do not show a strong predilection for NREM or REM sleep. Other parasomnias also include: sleep-related hallucinations, parasomnias due to drug or substance, parasomnias due to medical conditions, parasomnias unspecified.

<table>
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<td>Sleep Related Dissociative Disorders</td>
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sexual behaviors is wide and includes violent masturbations, sexual assaults and loud sexual vocalizations, fondling the bed partner, sexual intercourse with or without orgasm, and agitated sexual behaviors, which may have considerable medicolegal implications. In addition to occurring in confusional arousals, sexsomnias have been reported with other NREM parasomnias such as somnambulism. Treatment with medications such as clonazepam with simultaneous psychotherapy is an effective treatment combination in these patients. Another variant in adults of confusional arousals is severe morning sleep inertia. This variant remains clinically similar to typical confusional arousal and arises from light NREM sleep, but does not occur out of slow wave sleep.

**Sleepwalking**

Case history: A 67-year-old woman presented with a history of “difficulties with her sleep since she was a toddler.” She complained of multiple arousals in the night with associated “uncomfortable feeling in her legs.” She reported that the symptoms have been there for many years but gradually this has been getting worse. The patient’s niece, who accompanied her to the clinic, said that the patient wakes up in multiple spots in the house, calling these her “little nests.” She does not remember moving around the house at night and does not have any idea why she wakes up in different locations. In the morning, she also reports finding bed sheets, pillows, and covers at different spots in her house. The sleep disorders clinic managed her for restless leg syndrome, a possible factor contributing
to her sleepwalking behaviors, and the therapy completely resolved her spells, which were clinically believed to represent somnambulism. Her sleep and daytime functioning improved. She reported that her sleep was more consolidated and she slept in her bed throughout the night.

Sleepwalking (also known as somnambulism) arises out of slow wave sleep and is noticed usually in the first third of the nocturnal sleep, as there is more abundance of slow wave sleep, which serves as a substrate for these spells.\textsuperscript{4,19} The episodes are characterized as an ambulatory phenomenon during sleep. Patients with sleepwalking behaviors are typically calm; however, more complex behaviors such as eating, cooking, cleaning the house, unlocking doors, sexual activities, and even driving have been reported.\textsuperscript{20,21} If awakened, patients are observed to be confused and can become violent or agitated.\textsuperscript{22} The ambulation usually terminates spontaneously but may occur in unusual places such as the bathroom or kitchen. Most often, patients return to their bed and have no recollection of the episodes the following morning.

Sleepwalking is common in children, with prevalence as high as 17%, decreasing to about 3% in adults.\textsuperscript{23,24} Sleepwalking in men can be associated with injury and violence.\textsuperscript{25} Children between 4 and 6 years of age form the major group exhibiting sleepwalking behavior. With advancing age, especially after puberty, sleepwalking decreases significantly.\textsuperscript{4,5} Patients are at risk of injury from falls while going downstairs, running into closed doors or windows, or jumping out of windows.\textsuperscript{26}

Genetic factors play a significant risk for sleepwalking, especially if a first-degree relative is affected with this parasomnia, as the risk increases 10-fold when compared with the general population.\textsuperscript{27} In a twin study, monozygotic twins exhibited 6 times greater concordance for sleepwalking compared with dizygotic twins.\textsuperscript{28} Precipitating factors for sleepwalking include sleep deprivation, fever, centrally active substances, hypnotic medications (such as zolpidem), stress, and sleep-disordered breathing, sharing a similar predisposing factor as in patients with confusional arousals.\textsuperscript{29} Genetic susceptibility to develop sleepwalking has been linked to \textit{DQB1} genes, with 35% of White sleepwalkers testing positively compared with only 13.3% of controls.\textsuperscript{30}

Delta power density during spectral analysis is reduced in children who sleepwalk and adults with persistent sleepwalk.\textsuperscript{31–33} Patients who experience sleepwalking had increased power of low delta just before the confusional arousal in the first sleep cycle, suggesting a chronic inability to sustain slow wave sleep.\textsuperscript{32,34} Increased blood flow in the thalamus and cingulated cortex has been seen on a single photon emission-computed tomography (SPECT) scan during sleepwalking in a 16-year-old boy.\textsuperscript{35}

### Sleep Terrors

**Case history:** A 4-year-old boy experienced episodes of nighttime arousals with severe agitation and a piercing scream occurring within the first 2 to 3 hours of sleep onset, which lasted approximately 5 to 10 minutes. The frequency of these spells was once to twice per night. The episodes were first noted when he went on an overseas trip and his sleep-wake cycle was altered. Following an arousal with loud scream, his crying was inconsolable but had a waxing and waning pattern. At the time, he was also suffering from symptoms of upper respiratory infection (URI). On consultation with the pediatrician, his parents were given reassurance, and symptomatic treatment of his URI was initiated. In addition, better sleep hygiene, including attaining a regular sleep-wake schedule, turning off the lights, and limiting physical activity close to bedtime were discussed, including measures on how to best readjust the child’s altered circadian rhythm. Gradually as the child recovered from URI and his circadian rhythm became more synchronized, his nocturnal episodes resolved completely.

Sleep terrors, also known as night terrors or pavor nocturnes, are parasomnias arising out of slow wave sleep. Affected children are usually 4 to 12 years old and the estimated prevalence is between 1% and 6.5% of children.\textsuperscript{12} Although sleep terrors tend to resolve spontaneously during adolescence, they may persist and can be seen in 4% of adults as well.\textsuperscript{36} Psychopathology is rare in the affected children but may have a more significant factor in adults with sleep terrors.\textsuperscript{7}

Sleep terrors are characterized by a sudden arousal associated with a piercing scream or cry in the first few hours of sleep onset (Fig. 4).\textsuperscript{19} During a sleep terror, the patient may act in an afraid, agitated, anxious, and panicky manner.\textsuperscript{37} Inconsolability is a striking feature. Typically, the child does not want to be touched or comforted during the event, to the surprise of the parents. Verbalization during the episode is incoherent and perception of the environment seems altered. The child may run into walls or in circles and even run outside, possibly as a result of altered perception and panic. These events can be potentially dangerous, when ambulation is present, and may result in physical harm to self or their bed partners.\textsuperscript{25,38}

Sleep terrors typically last from 30 seconds up to 5 minutes. Most patients with sleep terrors are amnesic to the event in the morning but some...
maintain dream imagery or fragments of dream. A strong component of sympathetic activation characterized by tachycardia, tachypnea, sweating, flushed skin, or mydriasis is present in almost all patients, and is typically a key distinguishing factor. In adults, associated behaviors may be violent and may result in injury to the patient or the bed partner. Injury to self may appear as an apparent suicide (pseudosuicide) in some cases.

**Somniloquy**

Somniloquy, also known as sleep talking, is not listed under the category of traditional parasomnias in ICSD-II. In this manual it is classified under “sleep disorders associated with conditions classified elsewhere”. However, it is a common sleep-related behavior involving vocalization of sounds, speech, or at times conversations without any awareness of the event, and hence it is reminiscent of a parasomnia in its semiology. Events of somniloquy have the potential to lead to legal ramification if any confidential material is uttered. Diagnostic polysomnography reveals that the events most commonly occur during stage N1, N2, and REM sleep. The prevalence of this condition is not certain; however, it is estimated to be 4.9% in Chinese children between 2 and 12 years of age. In older adults, somniloquy is also associated with obstructive sleep apnea, other disorders of arousals, or RBD. No specific treatment exists but if there is suspicion for coexistent sleep disorder, diagnostic work-up and management of the underlying comorbid conditions is warranted.

**Nocturnal Frontal Lobe Epilepsy**

Nocturnal frontal lobe epilepsy (NFLE), including supplementary motor area seizures, are episodes
which may closely mimic the NREM parasomnias and pose a tough diagnostic challenge. The episodes are sudden, brief, spanning less than a minute in duration with little or no ictal confusion and occur exclusively or predominantly during sleep. The main distinguishing features between parasomnias and nocturnal seizures are shown in Table 1. The semiology suggests a frontal lobe origin involving the orbitofrontal or mesial frontal regions. There is often vocalization of variable complexity, frequent warning, usually nonspecific, the attacks seem to be bizarre and hysterical, the unique feature is a stereotypical pattern, and the interictal and ictal surface EEG are often normal. These episodes can be misdiagnosed during wakefulness as pseudoseizures and during sleep as movement disorders.

NFLE is a clinical example of localization-related epilepsy, which shares a strong interface with sleep. Three major clinic semiologies have been described in patients with NFLE: paroxysmal arousals, characterized by brief and sudden recurrent motor paroxysmal behavior; nocturnal paroxysmal dystonia (NPD) (discussed in further detail in the next section) and motor attacks with complex dystonic-dyskinetic features; and episodic nocturnal wanderings (stereotyped, agitated somnambulism).

An NFLE with strong genetic predisposition, autosomal-dominant NFLE (ADNLE) is a channelopathy with a defect in the neuronal nicotinic acetylcholine receptor. Video electroencephalography monitoring and video PSG with full EEG are useful in distinguishing NFLE and ADNFLE from other conditions such as parasomnias. Treatment with carbamazepine is effective in many patients with frontal lobe seizures.

### NPD

NPD was listed as a motor disorder of sleep in the earlier version of the ICSD, and the most recent edition classifies it as a form of frontal lobe epilepsy: sleep-related epilepsy. NPD consists of a sudden arousal, occurring during NREM sleep, associated with a complex sequence of movements, repeated dystonia or dyskinetic (ballistic or chorioathetotic). Patients may also move their legs and arms with cycling or kicking movements, rock their trunks, and show tonic asymmetric or dystonic posture of the limbs. A few cases are characterized by a violent ballistic pattern with flaying of the limbs. Differential diagnosis includes REM sleep behavior disorder and sleep terrors. Treatment with carbamazepine is often effective.

### REM PARASOMNIAS

The ICSD-II distinguishes among 3 separate REM parasomnias: nightmares, recurrent isolated sleep paralysis, and RBD.

#### Nightmares

Nightmares are common, affecting between 10% and 50% of children and up to two-thirds of the general population can recall at least 1 or a few nightmares in the course of their childhood. Half of all adults recall an occasional nightmare, whereas 1% report more than an occasional nightmare a week. Nightmares present as a vivid and prolonged dream sequence that tends to become progressively more intense, complex, and anxiety provoking, eventually terminating in an arousal and vivid recall. Episodes may increase during times of

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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>
</tr>
<tr>
<td>Movements</td>
<td>Common</td>
<td>Rare</td>
</tr>
<tr>
<td>Severity</td>
<td>Severe</td>
<td>Mild</td>
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<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>
<td>Present</td>
</tr>
<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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stress, particularly following traumatic events.\textsuperscript{53,54} Some medications such as levodopa, β-adrenergic blockers, and abrupt withdrawal of REM-suppressing medications may precipitate nightmares. The PSG shows an abrupt awakening from REM sleep associated with an increased REM sleep density and variability in heart and respiratory rates.\textsuperscript{55} Reassurance is often the main management necessary, but when episodes are severe and refractory, the use of REM-suppressing agents such as tricyclic antidepressants (TCAs) or selective serotonin reuptake inhibitors (SSRIs) may be needed.\textsuperscript{56–58} Differentiation between sleep terrors and nightmares is shown in Table 1.

**Recurrent Isolated Sleep Paralysis**

Sleep paralysis is defined as an inability to perform voluntary motor function at sleep onset or on awakening. The disorder occurs at least once in a lifetime in 40% to 50% of normal subjects. Patients report frightening episodes in which movements of the skeletal muscles are not possible, although ocular and respiratory movements and cognition usually remain intact. Episodes last a few minutes and may be aborted spontaneously or on external stimulation (vigorous eye movements). Episodes may occur in isolation, in healthy patients, as a genetically transmitted familial form, and as 1 of the narcolepsy symptoms. Depending on the meaning given to and cause of the sleep paralysis experience, which is largely culturally determined, patients may react to the event in specific ways. Predisposing factors include acute sleep deprivation and sleep-wake cycle disturbances (jet lag, shift work). In 1 study of first-year medical students, sleep paralysis episodes (predormital, postdormital, or both types of sleep paralysis) occurred in up to 16.25% of individuals.\textsuperscript{59} The underlying cause for sleep paralysis may be attributed to abnormalities in the mechanism controlling REM sleep muscle atonia and it is probably a result of abnormal activation of limbic system structures.\textsuperscript{60} Pharmacotherapy for sleep paralysis is often unnecessary when episodes are infrequent and in most cases reassurance is all that is needed. Management is most successful when patients avoid irregular sleep schedules but when sleep paralysis is severe, the use of anxiolytic medications and fluoxetine may be indicated.\textsuperscript{61,62}

**RBD**

RBD is characterized by abnormal elevation of limb or chin electromyography tone during REM sleep and by complex and elaborate motor activity associated with agitated and violent dream mentation. Patients with RBD report a wide spectrum of abnormal dream experiences, ranging from simple and relatively benign verbalizations, singing, yelling, shouting, and screaming to more complex motor phenomena such as walking, running, kicking, punching, jumping, and violent agitated behaviors that correlate with the reported aggressive dream imagery experience.\textsuperscript{53–65} The injury associated with the spells is often what brings the patient to the care and attention of the physician.

The prevalence of RBD is estimated to be 0.5%.\textsuperscript{8} The disorder has an increased gender predilection in that it affects men more than women (9:1 ratio), and has a higher prevalence in older age, usually in men more than 60 years old.\textsuperscript{63,65} The specific reason for the gender predilection in men is a mystery.\textsuperscript{56–68} Subjective reports indicate that about 25% of patients with Parkinsonism have dream enactment behaviors suggestive of RBD, and sleep evaluation in patients with Parkinson disease who experience sleep disturbances found RBD to be present in up to 47%.\textsuperscript{59,70}

Case history: A 68-year-old man presented with violent dreams reported by his wife. His dreams became extremely aggressive and “dramatic” in the last few months before presentation for sleep medicine evaluation. His wife reported that there is not 1 night that her husband wakes up without “kicking and cursing as if in an intense argument.” One episode led to his punching a lamp and sustaining severe cuts in his wrist, necessitating surgical repair. He had purchased 2 sleeping bags which he tied to the mattress to prevent himself from moving and hurting himself and his wife.

Patients with RBD may experience their spells as early as 90 minutes after falling asleep and more frequently during the second half of night, as REM sleep is denser in the latter part of the night. The frequency of episodes varies from once a month to nightly episodes, as in this case history, which result in more significant sleep disruption and are more likely to be brought to medical attention. RBD may be further classified into an acute and a chronic form. The acute form of RBD may be seen in the setting of substance- or medication-related cases, injury of the central nervous system (CNS) (stroke, demyelination), or metabolic derangements. The most common drug-related forms include rapid withdrawal from alcohol, abrupt discontinuation of sedative-hypnotics agents (which result in REM rebound), and cases related to TCAs, biperiden, monoamine oxidase inhibitors (MAOIs), cholinergic agents, and SSRIs (resulting in loss of REM atonia).\textsuperscript{71–81}
Exposure to caffeine (sometimes in the form of chocolate) has also been implicated in RBD. A prodromal history of other, and sometimes milder, forms of nocturnal spells such as sleep talking, yelling, or limb jerking may be present. With time, the dream content and enactment often become increasingly more aggressive, complex, action-filled, violent, or unpleasant, coinciding with the onset of RBD. Hypersomnolence may emerge if sleep becomes disrupted with dream enactment spells, leading to frequent arousals and nonrestorative sleep. The potential for bodily injury, skin lacerations, face ecchymoses, and skull fractures to patients and bed partners are a major safety concern and may necessitate preemptive pharmacologic interventions. Safety measures such as protective barricades around the bed, heavy curtains over the windows, removing sharp objects from the bedroom area, and sleeping on the ground floor in a sleeping bag (as in this case history) should be recommended until the disorder is fully managed.

Older age is a predisposing factor for the chronic or idiopathic form of RBD, which is generally more frequent, occurs later in life, becomes progressively more severe with time, and eventually stabilizes. Approximately 60% of patients who present with RBD are classified as idiopathic; the remaining cases are associated with underlying neurologic diseases and injury such as neurodegenerative disorders, cerebrovascular accidents, and multiple sclerosis. Dementias implicated in RBD include the synucleinopathies such as olivopontocerebellar atrophy and Lewy body disease with a characteristic \( \alpha \)-synuclein inclusion in the nerve cell bodies. The condition has also been reported in Machado-Joseph disease (spinocerebellar ataxia type 3) and the Guillain-Barré syndrome. Acute neurologic insult such as brainstem lesions caused by demyelination in multiple sclerosis, subarachnoid hemorrhage, pontine stroke, and brainstem neoplasm have been all implicated in the acute form of RBD. RBD typically begins in the sixth or seventh decade of life and may precede clinical manifestation of the underlying neuropathologic lesion process by several years to more than a decade. Patients with narcolepsy experience a higher incidence of RBD, and RBD may be the first sign of disease onset in children. Medications that act on the CNS and psychiatric medications such as TCAs, SSRIs, and MAOIs, which can be used to treat cataplexy, can sometimes exacerbate or trigger RBD in these patients.

The underlying pathophysiology of RBD may be related to abnormal brainstem control of medullary inhibitory regions. An identical syndrome was reported by Jouvet 4 decades ago. The studies involved an animal preparation, in cats, in which experimentally induced bilateral lesions of pontine regions adjacent to the locus coeruleus.
produced absence of REM-related muscle atonia associated and abnormal motor behaviors during REM sleep. In this experimental preparation, the cat slept until its first REM period, when it was noted to jump, with eyes still closed, and run around the cage, making attack motions. Two decades later, in 1986, the condition was eventually characterized as a new parasomnia by Schenck and colleagues, reporting a series of patients, mainly older men, with aggressive nocturnal behaviors. RBD is currently viewed as 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.

Recent brain imaging data, based on SPECT studies, show a possible mechanism relating to dopaminergic abnormalities and decreased striatal dopaminergic innervation as reduced striatal dopamine transporters. Positron emission tomography and SPECT studies further confirm reduced nigrostriatal dopaminergic projections in patients with multiple system atrophy and RBD. RBD is believed to be mediated neither by direct abnormal α-synuclein inclusions nor alone, by striatonigral dopaminergic deficiency as it probably reflects a more complex multiple neurotransmitter dysfunction involving GABAergic (γ-aminobutyric acid-mediated), glutamatergic, and monoaminergic systems. In patients with idiopathic RBD, impaired cortical activation as determined by EEG spectral analysis supports the relationship between RBD and neurodegenerative disorders. The potential pathophysiology of RBD is depicted in Fig. 6.

Evaluation of Parasomnias

A detailed history from the patient and especially from the witness of these events can provide valuable insight and lead to correct diagnosis, rewarding management, and preventive and safety interventions. Home-made video recordings if available can be used to lend support the presumptive diagnosis. Education and counseling the patient and family are key components in evaluation and management of most parasomnias. Any physical injury sustained to self or to the bed partner should be well documented.

The major differential diagnosis in patients with NREM parasomnias is sleep-related epilepsy. Differentiating parasomnias from sleep-related epilepsy can be challenging, especially when the patient lives alone or these nocturnal behaviors are unwitnessed. However, even when these behaviors are recorded during long-term video EEG monitoring, diagnosis can still remain uncertain. Up to 44% of patients with a diagnosis of NFLE can have a normal ictal EEG during video EEG recording. Similarly, a substantial number of patients with NFLE can have a personal and a family history of parasomnias.

A detailed personal history (sleep and nonsleep related) is critical to the diagnosis of NREM parasomnias. Family history, if available, can be helpful to support the clinical diagnosis. Recently a Frontal Lobe Epilepsy and Parasomnias (FLEP) scale has been reported to have higher accuracy in differentiating NFLE from parasomnias. The scale consists of several key questions based on the semiologic features of NFLE and parasomnias. The FLEP scale is designed to discriminate between features that are universally problematic based on clinical experience and the medical literature. Responses favoring nocturnal epilepsy (spells that are of brief duration, occurring several times per night, with a high degree of stereotypy) are scored positively, whereas those favoring parasomnias (amnesia, recall) are scored negatively. A score of 3 or higher is highly suggestive of epilepsy, whereas a positive score is more likely to represent a parasomnia. The FLEP scale may have some limitations in differentiating sleep-walking from episodic nocturnal wanderings (a form of NFLE) and RBD from seizures. For nocturnal frontal lobe seizures, the FLEP scale has a sensitivity of 71.4%, a specificity of 100%, a positive predictive value of 100%, and a negative predictive value of 91.1%. Factors that may precipitate NREM parasomnias as mentioned earlier should be explored, including comorbid obstructive sleep apnea, periodic limb movements of sleep, and temporal association with certain medications. Neuroimaging is typically not required in most patients with parasomnias, but may be necessary in patients with focal findings on neurologic examination or atypical features (eg, younger women with dream enactment behavior). The nocturnal PSG is not required for diagnosing NREM parasomnias but should be considered if there is concern about injury to the patient or spouse, or if there is an unusual presentation, suspicion for coexistent sleep disorders, or a high level of suspicion for an underlying seizure disorder.

Diagnostic Evaluation of RBD

Polysomnography is obligatory for RBD, revealing abnormal muscle augmentation during REM sleep; exceeding the normal REM sleep-related phasic electromyography twitches (Fig. 7). These motor phenomena may be simple (talking, laughing) or
complex (limb and trunk movements, repeated punching, kicking, or yelling) and are sometimes associated with emotionally charged utterances. When the patient wakes up from the episode they may have vivid recall and report dream mentation, which sometimes correlates with the observed behavior (trying to kick or protect themselves from an aggressive intruder). As in the NREM parasomnias the results of the neurologic history and examination may further indicate the need for other neurologic testing, including computed tomography or magnetic resonance imaging of the brain to identify structural lesion of underlying neurodegenerative processes. This procedure is especially important if the episodes are acute, are temporally related, follow neurologic injury, or occur in younger patients and women. The differential diagnosis of RBD includes nocturnal frontal lobe seizures, somnambulism, sleep terrors, confusional arousals, posttraumatic stress disorders, and nightmares. RBD is often distinguished based on the complex nature of the spells, occurrence later in the night when REM density is highest, and the characteristic patient profile (ie, older men). Differentiation among the key parasomnias and nocturnal seizures is shown in Table 2. Table 3 summarizes the differentiation between NREM parasomnias, REM parasomnias, and nocturnal seizures.

**Treatment of Parasomnias**

Successful management of disturbing parasomnias depends on establishing an accurate diagnosis. Reassurance, nonpharmacologic therapy,
and intervention measured to address safety issues are important for the less dramatic parasomnias. When the patient’s parasomnias are associated with displacement from the bedroom, the patient and the family need to maintain a safe bedroom environment, relocating the bedroom to the ground floor of the house and if possible, blocking all windows with heavy drapes. The patient should be warned to avoid precipitating events of parasomnias, including unusual stress, sleep deprivation, and ingestion of substances believed to promote arousal disorders such as alcohol (which is known to increase slow wave sleep and may promote parasomnias such as sleepwalking) and caffeine-containing substances (which have been implicated in parasomnias such as RBD).

**Treatment of NREM Parasomnias**

Counseling and support are the key elements for the treatment of NREM parasomnias. Improving sleep hygiene (keeping a regular sleep-wake cycle and limiting exercise, caffeine, alcohol, and exposure to bright light before bedtime) should be stressed to the patient and bed partners. Educating the patient and family members about taking necessary safety precautions when these episodes occur is helpful in most cases. Removing dangerous objects from the immediate vicinity, locking medication cabinets, locking doors and windows, hiding car keys, and placing the mattress on the ground floor are a few examples that limit the risk of injury. Forced awakenings, which risk precipitating some parasomnias during slow wave sleep, should be discouraged.

Pharmacotherapy is typically not needed for patients who present with mild or infrequent episodes. However, when there is a concern for physical injury or if the episodes involve potentially dangerous complex activities, pharmacotherapy is a necessity. Low-dose clonazepam and other benzodiazepines such as temazepam or...
### Table 2
Key similarities and differentiating features between NREM and REM parasomnias as well as nocturnal seizures

<table>
<thead>
<tr>
<th></th>
<th>Confusional Arousals</th>
<th>Sleep Terrors</th>
<th>Sleepwalking</th>
<th>Nightmares</th>
<th>RBD</th>
<th>Nocturnal Seizures</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time</strong></td>
<td>Early</td>
<td>Early</td>
<td>Early-Mid</td>
<td>Late</td>
<td>Late</td>
<td>Any</td>
</tr>
<tr>
<td><strong>Sleep stage</strong></td>
<td>SWA</td>
<td>SWA</td>
<td>SWA</td>
<td>REM</td>
<td>REM</td>
<td>Any</td>
</tr>
<tr>
<td><strong>EEG discharges</strong></td>
<td>/C0</td>
<td>/C0</td>
<td>/C0</td>
<td>/C0</td>
<td>/C0</td>
<td>/C0</td>
</tr>
<tr>
<td><strong>Scream</strong></td>
<td>–</td>
<td>+++++</td>
<td>–</td>
<td>++</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td><strong>Autonomic activation</strong></td>
<td>+</td>
<td>+++</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td><strong>Motor activity</strong></td>
<td>–</td>
<td>+</td>
<td>+++</td>
<td>+</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td><strong>Awakens</strong></td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td><strong>Duration (minutes)</strong></td>
<td>0.5–10</td>
<td>1–10</td>
<td>2–30</td>
<td>3–20</td>
<td>1–10</td>
<td>5–15</td>
</tr>
<tr>
<td><strong>Postevent confusion</strong></td>
<td>+</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>Child</td>
<td>Child</td>
<td>Child</td>
<td>Child-Young Adult</td>
<td>Older Adult</td>
<td>Young Adult</td>
</tr>
<tr>
<td><strong>Genetics</strong></td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>–</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td><strong>Organic CNS lesion</strong></td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>++</td>
<td>+++</td>
</tr>
</tbody>
</table>

*Abbreviations: REM, rapid eye movement; SWA, slow wave arousal*
Diazepam have been used to limit nocturnal arousals. Trazodone and certain SSRIs such as paroxetine are effective as well. Other co-morbid sleep disorders such as obstructive sleep apnea and periodic limb movements of sleep may be viewed as possible precipitating factors. Management of comorbid sleep disturbances in these patients results in a significant decline in frequency or disappearance of the NREM parasomnias. Cognitive behavioral therapy, biofeedback, relaxation therapy, and hypnosis are helpful in children and adults. Interventions such as anticipatory arousal therapy are beneficial, especially in children. Anticipatory awakening is believed to work by either preventing or interrupting the altered underlying electrophysiology of partial arousal, interrupting the disturbing behavioral features of the parasomnia.

Management of RBD

Patients with RBD should be carefully assessed for risks for injury, particularly given the aggressive nature of the events and the potential for displacement and violent behaviors. Active and passive safety measures are a necessity in every patient with possible RBD who experiences aggressive spells and affliction of harm to the bed partner. Practice guidelines are currently being written, and are not yet available in publishable form; the most extensive studies to date include pharmacotherapy with clonazepam (0.25–1 mg by mouth every bedtime), which achieved improvement in most (90%) patients with little evidence of tolerance or abuse.

Although clonazepam does not normalize abnormal muscle augmentation during REM sleep, it probably exerts its function in preventing the arousals associated with the REM sleep disassociation at the level of the pons. Treatment of RBD with melatonin may help normalize or perhaps even restore REM sleep electromyography atonia and may be effective in 87% of patients taking doses of between 3 and 12 mg at bedtime. Over-the-counter melatonin is not regulated or approved by the US Food and Drug Administration (FDA), is considered a dietary supplement, may have poor regulations in terms of pharmacologic preparation, and side effects have not been widely studied. Research into treatment with melatonin receptor agonists in patients with RBD is under way, but results are not yet available in publishable form. Other agents that may be helpful for RBD include imipramine (25 mg by mouth every bedtime), carbamazepine (100 mg by mouth 3 times a day) as well as dopamine agonists and precursors (ie, pramipexole and levodopa, respectively). One recent study described successful amelioration of RBD with sodium oxybate when other treatments are ineffective or poorly tolerated. This finding also suggests that RBD and cataplexy may in part share a common underlying mechanism.

SUMMARY

Much has been learned in the last few decades about pathogenesis of parasomnias, especially RBD. However, the role of central pattern generators as progenitors for disorders of arousal remains unclear. Although there are pharmacologic and nonpharmacologic management strategies, well-researched standard of practice guidelines, including strategies for evaluation and management, are not yet available in this domain of sleep medicine. Violent behaviors associated with parasomnia probably remain underreported and the forensic implications will undoubtedly continue to challenge the sleep physician as an expert witness.

### Table 3
Differentiating patterns between NFLE and parasomnias: discriminatory components on history

<table>
<thead>
<tr>
<th>Duration</th>
<th>NFLE</th>
<th>Parasonmia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration</td>
<td>&lt;2 min</td>
<td>&gt;10 min</td>
</tr>
<tr>
<td>Timing at night</td>
<td>Events in first 30 minutes</td>
<td>Events later in the night</td>
</tr>
<tr>
<td>Number of events</td>
<td>Multiple events per night</td>
<td>One or 2 events per night only</td>
</tr>
<tr>
<td>Complexity</td>
<td>Complex behavior uncommon</td>
<td>Often wandering and complex behavior</td>
</tr>
<tr>
<td>Semiology</td>
<td>Highly stereotyped</td>
<td>Variable semiology</td>
</tr>
<tr>
<td>Recall</td>
<td>Often full recall of event and speech</td>
<td>Event and speech during event not recalled</td>
</tr>
</tbody>
</table>

REFERENCES

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