

Parasomnias

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The group of sleep disorders known as parasomnias are defined broadly as undesirable physical or experiential events that occur during sleep. Parasomnias have fascinated human beings for centuries and early explanations of these unusual behaviors often were based in beliefs of the supernatural. In the early twentieth century, Freud and other psychoanalysts debated the nature of parasomnias, often under the assumption that such behavior represented the expression of underlying conflict or latent desire [1]. For decades, parasomnias were believed to be the result of underlying psychopathology; however, more recently, the pendulum has swung so that the majority of parasomnias are believed not related to psychiatric illness [2]. As these disorders are behavioral and often co-occur with psychiatric illness, however, they challenge the arbitrary separation of disciplines within neuroscience.

Key to the understanding of parasomnias is that the mammalian brain cycles through three primary states—wakefulness, NREM sleep, and REM sleep. These states are generated via complex interactions of neural networks, and specific regions of the brain may be recruited and function in different roles that are state dependent. Just as specific regions are not necessarily exclusive to a particular sleep-wake state, nor are the states themselves mutually exclusive to one another. Often, the transition between states is blurred, and components of different states blend together or oscillate rapidly, leading to behaviors that represent an admixture of sleep and wakefulness [2,3]. Parasomnias are the unwanted motor, verbal, or experiential manifestations that may occur from such overlap between sleep states.

There are many classification schemas that exist to categorize the parasomnias, the most familiar being the *Diagnostic and Statistical Manual of Mental Disorders (DSM)* and the *International Classification of Sleep Disorders (ICSD)*. The *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision*

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(*DSM-IV-TR*) distinguishes only four separate parasomnias, whereas the *International Classification of Sleep Disorders: Diagnostic and Coding Manual, Second Edition (ICSD-2)* is much further delineated (Table 1) [4,5]. Still, both systems are imperfect in either reflecting current understanding of pathophysiology or being of pragmatic value for psychiatrists. Thus, this review focuses on those parasomnias seen most frequently in psychiatric settings or that have understood connections to neuropsychiatric illness, including NREM parasomnias of arousal (including sleep-related eating disorder [SRED]) and specific REM-related parasomnias (Table 2). The discussion involves diagnosis and treatment options, including pharmacotherapy, with the caveat that there are no medications approved by the Food and Drug Administration for the treatment of parasomnias.

NON-RAPID EYE MOVEMENT PARASOMNIAS: DISORDERS OF AROUSAL

The NREM parasomnias share many common features that are related to their underlying pathology. They tend to arise from slow wave sleep (SWS) (stages 3 and 4 of NREM sleep) and, thus, typically occur during the first 1 to 2 hours of

Table 1
Parasomnia classification of *DSM-IV-TR* and *ICSD-2*

<i>ICSD-2</i>			
	Disorders of arousal (from NREM sleep)	Parasomnias usually associated with REM sleep	Other parasomnias
<i>DSM-IV-TR</i>	<ul style="list-style-type: none"> • Sleepwalking • Sleep terrors • Confusional arousals 	<ul style="list-style-type: none"> • Nightmare disorder • REM-sleep behavior disorder • Recurrent isolated sleep paralysis 	<ul style="list-style-type: none"> • Parasomnia not otherwise specified/ unspecified^a • Sleep related dissociative disorders • Sleep enuresis • Sleep related groaning (catathrenia) • Exploding head syndrome • Sleep related hallucinations • Sleep related eating disorder

^aParasomnia not otherwise specified according to *DSM-IV-TR* includes those parasomnias not shaded; whereas parasomnia, unspecified, according to *ICSD-2*, includes parasomnias not listed. *ICSD-2* notes that parasomnia unspecified is intended for parasomnias that can not be classified elsewhere or for cases in which physicians have a clinical suspicion that an underlying psychiatric condition may cause the parasomnia. Omitted are parasomnias due to medical condition and parasomnia due to drug or substance (*ICSD-2*) that are classified as sleep disorder due to a general medical condition, parasomnia type and substance-induced sleep disorder, parasomnia type, respectively, under *DSM-IV-TR* [4,5].

Table 2
Characteristics of parasomnias

Parasomnia	N-REM parasomnias				REM parasomnias		
	Confusional arousals	Sleep walking	Sleep terrors	SRED	RBD	Sleep paralysis	Nightmare disorder
Stage of arousal	II, III, IV	III, IV	III, IV	II, III, IV	REM	REM	REM
Typical time of night	Anytime	First 2 h	First 2 h	Anytime	Anytime	Anytime (first 2 h)	Anytime
EEG during event	NA	Mixed	Mixed	Mixed	REM pattern	Wake pattern	NA
EMG activity during event	↑	↑	↑	↑	↑	↓	NA
Decreased responsiveness during event	+	+	+	+	+	–	+
Autonomic hyperactivity	–	–	+	–	+	±	+
Amnesia	+	+	+	± (partial)	– (Dream recall)	– (Experience recall)	– (Dream recall)
Confusion post episode	+	+	+	+	–	–	–
Family history	+	+	+	+	–	±	–

sleep (and seldom from napping). Although they typically may arise from SWS, it is important to note that these parasomnias are disorders of arousal, not sleep per se. Physiologically, arousals are transient events (typically a few seconds in duration) during which the electroencephalogram (EEG) shifts from a deeper stage of sleep, to a higher frequency (often alpha rhythm). As these arousals are brief, they do not represent full wakefulness, and the limited duration of the arousal is not sufficient to change the stage of sleep (as they are only a fraction of the 30-second epoch typically used to score sleep stages). Behaviorally, arousals are events in which wakefulness has not been fully re-established, and in the case of a NREM parasomnia, behaviors (eg, walking, eating, and sexual behavior) and mood states (eg, fear and anger) are expressed that are not fully (or at all) under conscious control or remembered on awakening [6]. These behaviors occur without complex mentation, ordered judgment, or full integration of environmental feedback. NREM parasomnias commonly are seen in childhood and classically diminish with increasing age. There often is a family history of NREM parasomnias in individuals who present for evaluation, and these disorders likely are expressed when environmental factors affect genetically predisposed individuals [7]. In susceptible individuals, precipitating events can be either endogenous factors (eg, apnea, periodic limb movements, or pain) or exogenous factors (eg, sleep deprivation or medications) that disturb sleep [3].

As a result of commonalities, the categorization of NREM parasomnias typically is based on the type of behavior that occurs. In the following sections, these disorders are presented along a continuum of behavioral arousal while acknowledging that clinical overlap can exist between these disorders.

CONFUSIONAL AROUSALS

Confusional arousals typically are brief, simple motor behaviors that occur without significant affective expression or responsiveness to the environment. They are associated with mental confusion on arousal or awakening [8]. The motor behaviors are simple and may be accompanied by indistinct vocalization. The episodes are brief and because of dense amnesia for the episode, without collateral information from a bed partner or parent, they often are unnoticed. Although self-report data likely underestimate the prevalence of the disorder, prevalence is estimated at 4.2%, with comparable rates in men and women and prevalence that decreases with age [8].

A variant of confusional arousals is described as “sleep drunkenness” or excessive sleep inertia [9,10]. What distinguishes confusional arousals from excessive sleep inertia is that the latter traditionally is referred to as a phenomenon that stems from final awakening; however it remains similar to confusional arousals in regard to its immediate development from sleep, impaired mentation, automatic behavior, and relative unresponsiveness to the environment. The duration during which sleep inertia can affect individuals is debated, with studies in the literature ranging from a few minutes to 4 hours [10]. There are interindividual differences on the impact on daily life, depending on the

severity and time course over which sleep inertia might deter desired activities or mental tasks. Excessive sleep inertia can occur from naps and full sleep periods, and its severity and duration likely are related to the depth of prior sleep. Studies that use self-report to explore the epidemiology of confusional arousals may in fact focus on excessive sleep inertia, because patients are more apt to recall these episodes as their mentation improves and they achieve full wakefulness. One such study finds that bipolar disorder was associated strongly with such self-reported confusional arousals, although the significance of this finding is unclear [8].

SLEEPWALKING

Sleepwalking, or somnambulism, exists along a continuum with confusional arousals; however, it is distinguished by greater complexity of behavior. Sleepwalking behaviors often enact simple motivations (eg, desire to urinate); however, these behaviors may become dangerous because of limited conscious control (eg, climbing out a window). Dreaming typically is not present, but sleepwalkers may recount limited mentation of their motivations for their behavior if awakened during an episode. Episodes typically arise from SWS during the first part of the sleep episode. Sleepwalkers typically have eyes open during an event and may be clumsy in their behavior [11,12]. If left alone, sleepwalkers often return to sleep, although they may do so in atypical places. If interrupted while sleepwalking, they may have a range of responses ranging from no response to agitation or violence. It is not uncommon for individuals to have somnambulism as children but not to present for evaluation until they are young adults, when their behavior becomes concerning to a bed partner. Further influencing presentation for evaluation is the frequency and dangerousness of the sleepwalking behaviors, which can vary widely from nightly severe episodes to rare benign events. As in other NREM parasomnias, full or partial amnesia for the episode is typical.

Somnambulism occurs in 10% to 20% of all children, with the greatest prevalence occurring between 3 and 10 years of age [13]. Because it is so common in young children and its prevalence tends to decrease with increasing age, sleepwalking often is considered to be a transiently disruptive phenomenon that resolves by adolescence. Sleepwalking occurs in 1% to 4% of adults, and within this population exist individuals who did not sleepwalk as children; however, approximately 80% of adults who have somnambulism have it as a continuation of childhood behavior [8]. The prevalence of sleepwalking does not seem to be associated with gender, race, or socioeconomic conditions [13]. There is significant evidence to suggest that there likely is a genetic component to this disorder, as evidenced by epidemiologic and twin studies [14]. In fact, the HLA gene, DQB1, may confer susceptibility to somnambulism [15]. Risk of sleepwalking is approximately double for individuals if one parent and triple if both parents have a history of sleepwalking.

The relationship between psychopathology and sleepwalking is a debated issue in the literature [16]. Although sleepwalking in childhood does not seem to

be related directly to psychiatric pathology, it is observed that psychopathology may be associated with sleepwalking in adolescence and adulthood [8,17]. Still, sleepwalking behaviors do not seem to represent unconscious motivations acted out during sleep in the vast majority of patients, with the caveat that psychologic conflict may not be detected readily in such individuals unless they concurrently are in psychotherapy [18]. Psychotropic medications may raise the risk of adult somnambulism because of their effects on sleep and wakefulness [19]. Also, because disrupted sleep and subsequent sleep deprivation are common among psychiatric patients, this may increase the risk of sleepwalking in these individuals [20].

SLEEP TERRORS

Sleep terrors have many of the same characteristics of sleepwalking; however, they are distinguished by more intense motor and autonomic activity and affective expression. Rather than construing sleep terrors and somnambulism as distinct disorders, it is more appropriate to consider them as related entities that can, in fact, evolve from one to the other during an episode. Similar to somnambulism, sleep terrors tend to occur in the first third of the sleep period and are believed caused by a confluence of genetic susceptibility with precipitating factors [21]. The prevalence of sleep terrors is 5% in children and 1% to 2% in adults [8]. In children, the presentation of sleep terrors typically is dramatic: a piercing scream followed by fear, crying, and inconsolability [22]. In adults, agitation is common and often individuals who have sleep terrors may injure themselves, others, or property during an episode. Dreaming typically is not reported; however, similar to somnambulism, simple thoughts may be recalled (eg, "I am in danger") that can be difficult to dispel even once awakened. If confronted during an episode, there is the real danger that individuals may incorporate the witness into the sleep terror leading to potential harm. Thus, it is recommended that individuals having a sleep terror be redirected gently in an attempt to raise their level of consciousness, although full or partial amnesia is typical.

SLEEP-RELATED SEXUAL BEHAVIOR AND SLEEP-RELATED VIOLENCE

Along the continuum of NREM parasomnias associated with disordered arousal are sleep-related sexual behavior and sleep-related violence. Both of these disorders are less well understood compared with those disorders discussed previously; however, their unique clinical dimensions merit brief discussion.

Sleep-related sexual behavior, or *sexsomnia*, is a recently described parasomnia in which sexual behavior occurs with limited awareness during the act, relative unresponsiveness to the external environment, and amnesia for the event [23]. The sexual behavior exhibited often may be distinct from that typical for the patient in terms of partner or type of sexual act (eg, forced sex with on a bed partner, repetitive masturbation, or anal intercourse) [24]. Although

poorly understood, it is proposed that distinguishing features of sexsomnia that differentiate it from sleepwalking include more widespread autonomic activation, sexual arousal, and duration of behavior that occasionally can exceed 30 minutes [23]. Not surprisingly, patients who have this parasomnia may present for evaluation for myriad reasons, ranging from complaint of a bed partner to criminal charges.

Sleep-related violence is another parasomnia considered distinct from those discussed previously. In many respects, it can be conceived as an overlap disorder of sleep terrors after sleepwalking [25]. The violent behavior occurs in a state consistent with night terrors, with anger or fear as the primary emotion, agitated resistance to the environment, a slow return to normal levels of alertness, and amnesia for the event. Like sexsomnia, the violent behavior often is atypical for the patient (eg, stabbing or bludgeoning a bed partner or homicidal attacks on others). The majority of cases are young to middle-aged men who have a previous history of sleepwalking [26]. Because sleep-related sexual and violent behaviors are the subject of recent forensic proceedings, there is an urgency in establishing a more comprehensive understanding of these parasomnias.

EVALUATION AND TREATMENT OF NON-RAPID EYE MOVEMENT PARASOMNIAS

Polysomnography (PSG) often is unnecessary for the evaluation of NREM parasomnias and, in any case, attempts to document somnambulism and sleep terrors by PSG often are unsuccessful. It long has been acknowledged that even nightly sleepwalkers may not exhibit the behavior when monitored [27]. Thus, PSG markers of susceptibility have been studied for their diagnostic usefulness and potential insight into pathogenesis. The vast majority of PSG studies of sleepwalkers demonstrate increased brief arousals from SWS with a preserved sleeping EEG, accompanied by autonomic activation after the arousal [28,29]. Similarly, multiple brief arousals with autonomic hyperactivity may be observed as a marker of sleep terrors [30]. PSG may be indicated when there is no history of childhood parasomnias, as their emergence in adulthood may be due to other disorders that can cause arousal from SWS, such as sleep-related breathing disorder, periodic limb movements of sleep (PLMS), or nocturnal seizures. The treatment of the parasomnia complaint in these instances entails the treatment of the underlying or precipitating sleep disorder.

In the evaluation of patients who have episodes of abnormal, unwanted nocturnal motor or affective behaviors, ideally, practicing psychiatrists consider a broad range of diagnoses and are able to refer to a sleep specialist when appropriate. Often the differential diagnosis may include nocturnal panic attacks, nocturnal dissociative episodes, frontal lobe seizures, delirium associated with medical or neurologic disorders, and REM sleep behavior disorder (RBD) (discussed later). Sleep-related dissociative disorders (SRDD) only recently have been considered a distinct sleep disorder, included and categorized as a parasomnia in the *ICSD-2*, thus meriting brief discussion [5]. SRDD occurs when

individuals who have a dissociative disorder have nocturnal episodes of dissociation during EEG established wakefulness (ranging from the transition from sleep to wakefulness to several minutes after achieving wakefulness). Myriad behaviors may manifest during these episodes that may be complex, violent, self-mutilating, abuse re-enactments, or fugue [31]. Prevalence is not known; however, 7 of 100 consecutive patients referred to a sleep disorders center for sleep-related injury were found to have SRDD [32]. Although nocturnal dissociative episodes tend to arise from more clearly established wakefulness than other parasomnias, their undesirable behavior, relatedness to sleep, and impaired awareness further challenge the delineation of sleep from wakefulness and consciousness from unconsciousness [33]. A history of similar daytime behaviors (eg, dissociation or panic attacks) makes the diagnosis of a NREM parasomnia less likely but does not rule out the diagnosis definitively. PSG may be indicated to rule out frontal lobe seizures or RBD or more clearly delineate a nocturnal dissociative episode.

The decision to treat NREM parasomnias is based on a risk-benefit analysis that considers the frequency of the parasomnia event, the risk of injury to self or others, and the distress the behavior is causing patients or family members [6]. The decision to treat also is complicated by the fact that the majority of parasomnias occur infrequently in adult patients; however, their appearance is unpredictable. Thus, adults who have problematic or high-risk parasomnias must decide whether or not chronic treatment for an episodic illness is desired.

For the majority of children, parasomnias do not require treatment because the behavior may be self-limited, poses little risk of harm to the child, and may have limited daytime sequelae because of amnesia for the event. Keeping regular sleep and waking times and avoidance of sleep deprivation often reduce the frequency of events. For children and young adults who sleepwalk, improving the safety of the sleeping environment (eg, locking doors and windows, keeping hallways and stairs well lit, removing dangerous objects from sleep area, placing mattress on floor, and, if possible, having bedroom on first floor) is important.

When treatment of sleepwalking or sleep terrors in adults is indicated, it is done following a three-step model: (1) modifying predisposing and precipitating factors, (2) improving the safety of the sleeping environment, and, if necessary, (3) using pharmacotherapy. As there are no controlled clinical trials that examine pharmacotherapy for NREM parasomnias, medication selection is based largely on anecdote and clinical experience. The agents used most commonly in the treatment of NREM parasomnias are from classes of medications that affect the γ -aminobutyric acid (GABA)-ergic system, although it is unclear whether or not these medications work by suppressing arousals during sleep or by decreasing SWS. The most data regarding treatment of NREM parasomnias exist for benzodiazepines (primarily clonazepam [0.5–2.0 mg at bedtime] but also alprazolam and other benzodiazepines), which are used successfully in moderate-sized open label trials in the treatment of injurious parasomnias for greater than 6 months [32,34]. Although these studies are limited in their ability

to detect tolerance, the majority of patients taking these agents do not have to escalate their dose to manage their parasomnia once at a therapeutic dose [34]. The long half-life of clonazepam increases the likelihood that patients may have lingering daytime effects of this medication; thus, if the parasomnia occurs in the first-half of the sleep period, selecting a shorter-acting benzodiazepine, such as triazolam (0.125–0.5 mg at bedtime), or benzodiazepine receptor agonist, such as zolpidem (5–10 mg at bedtime), may be tolerated better, keeping in mind that somnambulism-like behaviors can be rare side-effect of these medications and use of these medications is based largely on clinical experience and anecdote (Fig. 1).

SLEEP-RELATED EATING DISORDER

SRED is a NREM parasomnia that only recently has been described in the medical literature but has achieved notoriety in the popular media [35]. SRED is conceptualized best as a combination of the binge eating of bulimia nervosa with the disordered arousal, confusional behavior, and amnesia of a parasomnia [36,37]. SRED manifests as repetitive partial arousals from sleep, typically within 2 to 3 hours of sleep, with ingestion of food often in a hurried or “out-of-control” manner, despite frequent lack of hunger at the time of the episode. Foods consumed often are high-carbohydrate but also may consist of unusually combined foods, frozen foods, or non-nutritive substances. Patients often feel ashamed, gain weight as a result of the behaviors, and may attempt to

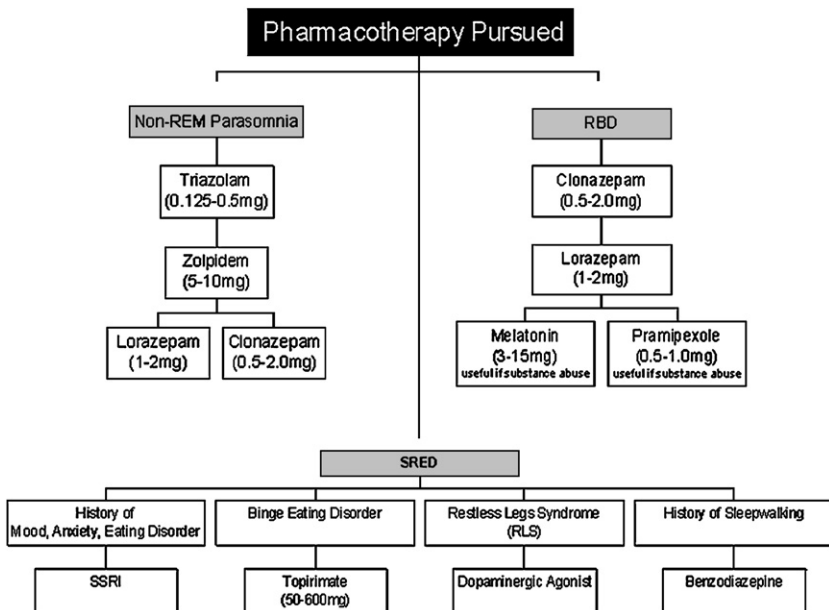


Fig. 1. Potential pharmacotherapy of parasomnias.

control their overall caloric intake forcibly via daytime anorexia. Many patients report reduced awareness at the time of the episode, stating they were mostly asleep or half-awake/half-asleep. Many patients who have SRED also report at least occasional amnesia for the episodes, and specifics may be elucidated via witnesses to the episodes or reconstructed from evidence on awakening (eg, food missing, messy eating place, or food in bed). The level of awareness may vary between episodes within the same night and from night to night over the longitudinal course of the disorder. Diminished awareness during an episode is what distinguishes SRED from nocturnal eating syndrome (NES), a disorder characterized by eating excessive amounts of food either before bed or during nocturnal awakenings while maintaining full consciousness [38]. Given that the criteria for SRED in the recently revised *ICSD-2* do not require impairment in consciousness during nocturnal eating episodes, but rather only “involuntary” eating, it is not clear whether or not NES and SRED are distinct disorders [5]. It may be most useful to consider NES and SRED as entities at opposite ends of a continuum of awareness during nocturnal eating. Although studies are limited, the prevalence of SRED is estimated to be 1% to 5% in the general population, to be 2 to 4 times more common in females, and tends to have onset in late adolescence or early adulthood; the prevalence of NES is found to be as high as 12.3% in psychiatric outpatients [39,40]. SRED seems to be a chronic disorder, so patients often do not present until years after they first develop symptoms. Patients who have SRED may have a history of sleepwalking; however, once eating behaviors during sleep become established, they tend to replace any other distinct sleepwalking behaviors. The pathophysiology of SRED and NES remains unclear; however, both theoretically could be related to abnormalities in the expression of hormones regulating appetite and the sleep-wake cycle [41,42]. Restless legs syndrome (RLS) may be comorbid with SRED in some patients, although the prevalence of co-occurrence is unknown, and treatment of RLS in these individuals may diminish nocturnal eating behaviors. There also are some case reports that benzodiazepine receptor agonists, and possibly atypical antipsychotics, can produce symptoms of SRED [43–45].

SRED seems to be more common in patients who have a daytime eating disorder; however, the vast majority of patients who have SRED do not have an eating disorder that manifests while awake [39,46]. Approximately one-third of patients who have SRED have a first-degree family member who has similar symptoms, comparable to familial patterns observed in sleepwalking and certain eating disorders [47,48]. PSG studies show that SRED behaviors can manifest from any time of night and from all states of NREM sleep [36]. The PSG features of SRED, however, are similar to other NREM parasomnias with frequent arousals from SWS [37].

Treatment of SRED is similar to other NREM parasomnias, with some notable exceptions. Besides avoidance of sleep deprivation and maintaining the safety of the sleeping environment, normalization of a daytime eating schedule is important. Pharmacotherapy should be tailored to individual patients. Those

who have a history of sleepwalking can be given short to intermediate acting benzodiazepines; however, this approach can worsen dissociated eating and amnesia. Alternatively, selective serotonin reuptake inhibitors (SSRIs) or topiramate are effective in some case series [49–51]. Treatment of patients who have SRED (in particular those who have symptoms of RLS) with dopamine agonists also is shown useful in an uncontrolled case series [46].

RAPID EYE MOVEMENT-RELATED PARASOMNIAS

During REM sleep, the body undergoes specific physiologic and experiential phenomena, including atonia of the voluntary muscles (except extraocular), elevated autonomic activity, and dreaming. The following REM-related parasomnias involve either the incoordination of these processes or the inappropriate admixture of REM sleep and wakefulness.

RAPID EYE MOVEMENT SLEEP BEHAVIOR DISORDER

RBD is characterized by the loss of coordination of dreaming and paralysis of the skeletal muscles during sleep. With the body free to move during REM sleep, individuals who have RBD act out their dreams, which can include complex motor behaviors, including screaming, punching, kicking, and so forth. In RBD, patients exhibit behavior with eyes closed and unresponsive to the environment around them. When awakened while acting out a dream, individuals achieve rapid full alertness and often report a dream to which their behavior corresponds. The impetus that often brings patients who have RBD to a physician's office is agitated or violent behavior leading to self-injury or injury to a bed partner.

The prevalence of RBD is estimated to be between 0.04% and 0.5% of the population [52]. It often is useful to separate RBD into two clinical forms, acute and chronic. Acute RBD typically is associated with medications, drugs of abuse, or withdrawal (in particular alcohol) [53]. The chronic form is seen most commonly in men who are over 50 years of age. Chronic RBD typically is subdivided into two types, idiopathic and secondary to a neurologic process. The diseases associated most commonly with RBD are the α -synucleinopathies, including Parkinson's disease, dementia with Lewy bodies, and multiple system atrophy—all of which are characterized by pathologic accumulation of the protein α -synuclein [54]. The three largest cohorts of patients who have RBD suggest that approximately 60% of chronic RBD is idiopathic, with the remainder secondary to a neurologic disease [52]. Follow-up studies suggest, however, that idiopathic RBD may be a prodromal syndrome for an underlying condition, leading some to question the nosology of idiopathic RBD [52,55]. Still, many patients initially diagnosed with idiopathic RBD never develop other neurologic illness, even decades after the onset of RBD.

Although the pathophysiology of RBD is not clear, the extrapyramidal and REM sleep systems in the brainstem share specific neuronal connections, which may be central to RBD pathogenesis. Animal models of RBD in which lesions in the brainstem in the region of the locus coeruleus produced REM sleep

without atonia, were developed many years before RBD was described clinically as a disorder in humans, implicating these brainstem areas in the control of motor activity during REM sleep [56]. Furthermore, reduced dopamine transporters in the striatum and diminished striatal dopaminergic innervation (using single photon emission CT and positron emission tomography, respectively) are demonstrated in RBD [57,58]. Similarly, a reduction of neurons in the peri-locus coeruleus is observed [59]. One intriguing hypothesis is that the pedunculopontine nucleus (PPN) may play a large role in the REM-atonia circuitry and its disruption in RBD, connecting clinical observations regarding the α -synucleinopathies/extrapyramidal system and RBD with observations in pontine-lesioned animal models [60].

Besides clinical history, PSG is necessary in confirming the diagnosis of RBD. PSG monitoring demonstrates elevated muscle tone or increased phasic muscle activity in the chin (submental) or limb (anterior tibialis) electromyogram during REM sleep [5]. At times, subtle or gross body movements may be recorded during the study. It is not unusual to see excess PLMS during REM and NREM sleep, but otherwise the PSG typically is normal. PSG may, however, be useful if another sleep disorder (eg, sleep-disordered breathing) is believed to contribute to the emergence of RBD. Nonspecific signs and symptoms that also may be found in RBD include general slowing of the waking EEG, subtle neuropsychologic dysfunction, autonomic dysfunction, subtle abnormalities of motor and gait speed, impairment in color discrimination, and olfactory dysfunction [61–64].

The management of RBD typically is behavioral and pharmacologic. From a behavioral standpoint, it is not uncommon for patients to devise their own home remedies to manage their behavior during sleep, such as sleeping in sleeping bags, tethering themselves to their beds, and so forth [53]. Although some of these may be problematic, they do adhere to management principles of other parasomnias—creating a safe sleeping environment for patients and bed partners.

First-line pharmacologic agents in the treatment of RBD are benzodiazepine receptor agonists (Fig. 1). The most commonly used of these agents is clonazepam (0.5 mg–2 mg), which is shown to decrease the frequency and extent of problematic dream-enacting behavior in open label trials [34,65]. In general, this agent is well tolerated; however, given that the majority of patients who have RBD are older individuals, the cognitive impairment and daytime sedation that may be associated with this long-acting benzodiazepine is of concern. In such instances, shorter-acting benzodiazepines (ie, lorazepam 1–2 mg) may be used. In cases where benzodiazepines are problematic because of daytime motor/cognitive effects or in patients who have substance abuse problems, small, uncontrolled case series suggest that melatonin (3–15 mg at bedtime) and pramipexole (0.5–1 mg at bedtime) may be efficacious in RBD [66,67].

Of particular importance to practicing psychiatrists is the role that REM-suppressing antidepressants (SSRIs), monoamine oxidase inhibitors (MAOIs), and tricyclic antidepressants (TCAs) can play in exacerbating RBD, as these agents are reported to cause or worsen RBD [65]. Furthermore, subclinical

RBD, in which motor tone is disinhibited, is associated with acute and chronic use of serotonergic antidepressants [68]. Thus, we (the authors) recommend the discontinuation of SSRIs, MAOIs, and TCAs if clinically feasible in patients who have RBD, particularly if RBD emerges as a result of these medications.

SLEEP PARALYSIS

Sleep paralysis (SP) refers to a conscious state at the onset or offset of sleep with associated paralysis of the voluntary musculature. Episodes may last from seconds to minutes, can be associated with hypnagogic or hypnopompic hallucinations, and thus, can be quite distressing. SP is believed to result from inappropriate REM intrusion into wakefulness or, conversely, the failure to maintain sleep during REM periods [69]. SP can occur at any time of night but tends to be clustered in the first 2 hours of the sleep period or at final awakening and may be worsened by sleep deprivation and supine positioning [69–71]. Episodes typically last seconds to minutes, and may disappear either spontaneously or when touched (eg, by a bed partner). The lifetime prevalence of SP is unclear, as estimates in the literature vary widely between 2.3% and 40%, with multiple episodes occurring in 1% to 10% of the population [69]. Not surprisingly, myriad cultural factors may influence reporting, subjective experience, and explanation of SP [72].

Although many patients who have SP may have it in isolation, the symptom of SP should prompt practitioners to inquire about other symptoms of associated neuropsychiatric illness. Primarily, SP is a classic symptom of narcolepsy; thus, symptoms of excess daytime sleepiness, cataplexy, and sleep attacks should be elicited [73]. Unless narcolepsy is suspected by history, PSG typically is not indicated. Furthermore, although the vast majority of patients who have SP likely do not have associated psychiatric illness, large epidemiologic studies find significantly higher rates of bipolar, depressive, and anxiety disorders among individuals who have SP [74]. Furthermore, some small studies find higher rates of SP in posttraumatic stress disorder (PTSD) versus non-PTSD individuals in specific cultural cohorts [75]. Treatment of underlying neuropsychiatric illness, when indicated, is important in the management of secondary SP; however, often times, reassurance and education are most useful in isolated cases. If the frequency of SP is bothersome to patients, there is a suggestion that SSRIs may be of some benefit, likely because of their REM-suppressing properties [76].

NIGHTMARE DISORDER

Dreams represent recall of mental activity that occurs during sleep, and although dreaming tends to occur predominantly during REM sleep, dreams occur during REM and NREM sleep. Nightmare disorder is characterized by recurrent dreams, followed by awakening with full and often detailed recall of the dream. Typically, fear is the predominant associated emotion recalled; however, anger, embarrassment, and sorrow also might occur. Nightmares

typically occur in the latter third of the night, which coincides with the increased proportion of REM sleep that occurs at this time [77]. Unlike RBD, nightmares typically are not associated with overt motor dream enactment. It often is difficult for individuals to return to sleep after a nightmare (unlike after sleep terrors), thus, frequent nightmares can lead to fear of going to sleep and subsequent insomnia.

Estimates of the epidemiology of nightmares and their association with psychiatric illness are hindered by inconsistent definitions. Still, it is estimated that 5% to 8% of adults in the general population have nightmares frequently enough to cause complaint, and nightmares are more common in women than men [78,79]. The majority of studies (but not all) find nightmares to be associated with myriad psychiatric diagnoses, including depression, substance abuse disorders, and personality disorders [80]. There also is some data to suggest a connection between nightmares and suicidality [81,82]. When distress and frequency of nightmares are assessed independently, distress seems to be tied more closely to psychopathology [83].

The psychiatric illness associated most commonly with nightmares is PTSD, as nightmares are a diagnostic feature of PTSD as part of the re-experiencing cluster [4]. It is notable that the *DSM-IV-TR* excludes the diagnosis of the parasomnia nightmare disorder if the nightmare is believed secondary to another psychiatric illness; however, the *ICSD-2* considers PTSD a predisposing factor for nightmare disorder [4,5]. The prevalence of nightmares in PTSD seems to be related in part to the study population and the specific trauma. Studies examining the general population, however, find individuals who have PTSD report nightmares at nearly fivefold higher rates relative to non-PTSD individuals [84]. Nightmares associated with PTSD often have similar thematic content or literal associations to the trauma history, and recurrent nightmares often occur [85]. A tendency to experience “bad dreams” and interrupted sleep preceding a trauma was found to be a risk factor for PTSD and depressive symptoms after Hurricane Andrew, which suggests premorbid nightmares may be a marker for posttrauma psychopathology [86].

The diagnosis of nightmare disorder is clinical, as PSG is generally of little value, unless needed to rule out another parasomnia. In PTSD, nightmares rarely are observed in sleep laboratories, but when seen, they occur from REM and NREM sleep. Several medications are found effective in the treatment of PTSD-related nightmares, including prazosin (small, controlled trial), topiramate (open-label, add-on trial), and atypical antipsychotics (case reports); however, the majority of studies are limited by small sample size [87–89]. The behavioral intervention of imagery rehearsal therapy, in which alternative versions of nightmares with better outcomes are rehearsed while awake, also shows benefit for the treatment of nightmares, trauma and nontrauma related [90].

SUMMARY

Parasomnias represent undesirable behaviors that arise from sleep but are not fully under voluntary control. Different classification schemas exist for the

categorization of these disorders, however parasomnias can broadly be grouped according to whether they arise from NREM or REM sleep. NREM parasomnias are best conceptualized as disorders of arousal that occur along a continuum of behavioral, affective, and autonomic activation. When indicated, treatment with benzodiazepines has the widest clinical support, however no controlled studies have been performed. SRED is a NREM parasomnia that has recently been described and is best conceptualized as an overlap disorder between an eating disorder and a parasomnia. REM-related parasomnias include RBD, sleep paralysis, and nightmare disorder, which can be idiopathic or secondary to underlying neuropsychiatric disorders. Treatment with benzodiazepines has shown some benefit for RBD, while SSRIs may be useful if the often-normative experience of sleep paralysis is treated pharmacologically. Both medication and cognitive therapies may be of value in nightmare disorder.

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