Parasomnias

John A. Fleetham MB, Jonathan A.E. Fleming MB

S leepwalking, sleep terrors, sleeptalking and sleep paralysis are some of the behavioural manifestations associated with the partial arousals from sleep known as parasomnias—a group of sleep disorders defined as undesirable physical events or experiences that occur during the initiation of sleep, during sleep or during arousal from sleep.¹ Although more common in children, parasomnias can occur at any age.

During sleep, the brain cycles regularly between wakefulness, nonrapid eye movement (NREM) sleep and rapid eye movement (REM) sleep. Nonrapid eye movement sleep is subdivided into four stages: stage I, a transitional stage between wake and sleep; stage II, which makes up most of the sleep period; and stages III and IV, which typically occur in the first half of the night and during which more profound stimulus is required to wake the sleeper. Parasomnias occur when transitions between these stages are blurred (commonly between stages III/IV and the awake state), causing behaviours that lack the complete awareness and mentation associated with wakefulness.¹

There are two criterion-based classifications of sleep disorders: the *Diagnostic and Statistical Manual of Mental Disorders, fifth edition* (DSM-V) classification² and the International Classification of Sleep Disorders (ICSD) classification. The DSM-V uses a "lumping versus splitting" approach and is less comprehensive than the ICSD classification. In this review, we use the ICSD classification (Box 1). The ICSD classification divides parasomnias into six groups of disorders that can emerge from either NREM or REM sleep, be related to drug or substance use or be a symptom of another medical disorder. Of parasomnias, REM sleep behaviour disorder is most commonly associated with an underlying neurologic condition.¹

In this review, we address parasomnias in adults;³ sleep disorders in children have recently been reviewed elsewhere.⁴ Nonrapid eye movement parasomnias are relatively rare, episodic, often self-limiting events that may not be captured during a polysomnogram. Consequently, their systematic study is often limited to case reports and small case series. Because there have been no large randomized controlled trials of interventions and there are no guidelines available (with the

exception of nightmares), the level of evidence for management options is low and is largely limited to the opinion of experts. The sources of evidence used in this review are shown in Box 2.

Rapid eye movement sleep behaviour disorder, an REM parasomnia, is a more prevalent condition associated with both iatrogenic causes and medical conditions. It can be reliably confirmed by polysomnography, which has resulted in a higher level of evidence and a best practice guideline.⁵

How do parasomnias commonly present?

All parasomnias occur from sleep, and NREM parasomnias usually occur among individuals aged 5–25 years with a family history of similar parasomnias (e.g., sleepwalking or sleep terrors) and involve physical and verbal activity of varying complexity. Typically, the sleeper returns to sleep and is amnestic in the morning, with the behaviour being reported by others or discovered because items have been moved or used during the night or the events have resulted in injury.

External (e.g., noise, temperature change) or internal (e.g., apnea, periodic limb movement) events may cause an arousal or partial awakening, usually from stage III and IV sleep, which occurs in the first 90 minutes of the sleep period. Arousal or partial awakening can also occur during stage II sleep later in the night.² Sleep deprivation or disruption from any cause (e.g., restless legs syndrome, obstructive sleep apnea) can precipitate these events in susceptible individuals.

In contrast, REM sleep behaviour disorder is characterized by verbalizations and actions consistent with dream enactment (e.g., fight or flight behaviours) that usually awaken the sleeper who, **Competing interests:** None declared.

This article has been peer reviewed.

Correspondence to: John Fleetham, john.fleetham@vch.ca

CMAJ 2014. DOI:10.1503 /cmaj.120808

KEY POINTS

- Parasomnias are very common and are not related to underlying psychiatric disease.
- Parasomnias may be complex and prolonged.
- Evaluation and treatment of parasomnias is suggested for patients whose activities are potentially harmful or very disturbing to others.
- Medication effects or central nervous disease should be considered for REM sleep behaviour disorder in younger patients.

unlike those with NREM parasomnias, can often recall the event, including the dream and its associated actions.¹

The characteristics of NREM and REM parasomnias are summarized in Table 1. Distinguishing between NREM and REM parasomnias is important because the treatment and prognosis are different. Nonrapid eye movement parasomnias are more common in younger people and are usually "outgrown" by young adulthood, whereas REM parasomnias present in late adult life and are associated with degenerative brain diseases.²

The major differential diagnoses for both NREM and REM parasomnia include nocturnal seizures, intoxicated states, medication-induced complex behaviours, nocturnal panic attacks and dissociative disorders.²

Are investigations required for diagnosis?

Parasomnias are typically diagnosed by obtaining a careful clinical history that assesses the timing, expression and form of the behaviour in the home environment (Box 3). A sleep diary (to exclude sleep deprivation as a precipitant) and a partner's log of the events are useful tools. (See http://yoursleep.aasmnet.org/pdf/sleepdiary.pdf for an example of a sleep diary.)

Referral to a specialist in sleep disorders should be considered for patients whose activi-

Box 1: International Classification of Sleep Disorders for parasomnia

- 1. Disorders of arousal from nonrapid eye movement sleep
 - a. Confusional arousals
 - b. Sleepwalking
 - c. Sleep terrors
- 2. Parasomnias usually associated with rapid eye movement sleep
 - Rapid eye movement sleep behaviour disorder
 - b. Recurrent isolated sleep paralysis
 - c. Nightmare disorder
- 3. Other parasomnias
 - a. Sleep-related dissociative disorders
 - b. Sleep enuresis
 - c. Sleep-related groaning
 - d. Exploding head syndrome
 - e. Sleep-related hallucinations
 - f. Sleep-related eating disorder
- 4. Parasomnia, unspecified
- 5. Parasomnia due to drug or substance use
- 6. Parasomnia due to medical condition

ties are potentially harmful or very disturbing to others, or if polysomnography is required.

It is not cost-effective to obtain a polysomnogram for patients with NREM parasomnia, except to exclude other causes of additional sleep instability (e.g., obstructive sleep apnea, periodic limb movement disorder, narcolepsy). Because one of the ICSD diagnostic criteria for REM sleep behaviour disorder requires the demonstration of REM sleep without atonia, polysomnography is needed to confirm this diagnosis and to rule out comorbid sleep disorders, such as obstructive sleep apnea or periodic limb movement disorder, which are more prevalent in adults and may need to be a focus of management.

How are specific parasomnias managed?

Although incompletely understood, the pathophysiology of NREM and REM parasomnias differ; however, there are commonalities in their management.

There have been no large, randomized controlled treatment trials for all parasomnias, and guidelines are limited to the management of nightmares⁸ and REM sleep behaviour disorder.⁵

Parasomnias commonly associated with NREM sleep

There are three main types of NREM parasomnias: confusional arousals, sleepwalking and sleep terrors (Table 1). We have included sleeprelated eating disorders and sleeptalking in this section because they emerge from NREM sleep. The DSM-V classifies sleep-related eating as a subtype of sleepwalking, and the ICSD notes that sleeptalking can occur during any stage of sleep.

Confusional arousals

Confusional arousals can occur any time sleep is interrupted⁹ and can accompany sleep-disruptive conditions, such as narcolepsy, obstructive sleep apnea, periodic limb movement disorder or by forced awakenings, especially in sleep-deprived individuals. Drugs and medications⁶ that disrupt sleep can also cause confused arousals.¹⁰

Box 2: Evidence used in this review

We searched Web of Science and MEDLINE for reviews and guidelines on the assessment and treatment of parasomnias, as well as for specific parasomnias (from 2008 to 2012). We also reviewed the citations for parasomnias in the International Classification of Sleep Disorders¹ and the Diagnostic and Statistical Manual of Mental Disorders, fifth edition.²

Table 1: Characteristics of parasomnias	of parasomnias						
		Nonrapid eye mov	Nonrapid eye movement parasomnia		Rapid e	Rapid eye movement parasomnia	ıia
Characteristic	Confusional arousal	Sleepwalking	Sleep terrors	Sleep-related eating disorder	REM sleep behaviour disorder	Sleep paralysis	Nightmare disorder
Emerges from	Stages II, III and IV*	Stages III and IV	Stages III and IV	Stages II, III and IV	REM sleep	REM sleep	REM sleep
Time of night	Anytime during sleep	First third of sleep	First third of sleep	Anytime during sleep	Anytime but most frequently last third of sleep	Anytime	Anytime but most frequently last third of sleep
Vocalizations	Yes	Yes	Marked (screaming or crying)	Yes	Marked	Slight (moaning or groaning)	Sometimes
Getting out of bed	Rare	Usual	Sometimes	Always (food seeking)	Yes	No	No
Responsiveness on awakening	Decreased	Decreased	Decreased	Decreased	Responsive	Responsive	Responsive
Autonomic activity	Normal	Normal	Increased	Normal	No	No	Yes
Post-event confusion	Yes	Yes	Yes	Yes	No	No	No
Prevalence	4.2% ¹⁰	1%–4% in adults, 80% of adults have sleep walked as children¹º	1%-2% in adults ¹⁰	Unknown; estimated to be 1%–5% in general population but higher among patients with eating disorders; 2–4 times more common among women ⁴	Unknown; estimated to be 0.38% in the general population and 0.5% among elderly people; about 33% of patients with newly diagnosed Parkinson Disease', more common among men	Unknown. 15%–40% has been reported among students under 30 years for at least one episode ¹ and 1%–10% of the population for multiple episodes ⁴	5%-8% of adults;4 more common among women
Amnesia of event	Yes	Yes	Yes	Variable	No	No	No
Injury risk	Low if undisturbed	Low if undisturbed; may strike out if disturbed or intoxicated	More common; may injure self trying to escape; may strike out if disturbed or intoxicated	Self-injury from food preparation (cuts, burns, poisoning)	May injure self or partner as part of dream enactment	None	None
Family history of parasomnias	Yes	Yes	Yes	Yes	Occasionally	Yes	Yes; twin studies suggest a genetic predisposition and co-occurrence with other parasomnias!
Note: REM = rapid eye movement.	ement.						

*The scoring of sleep has changed recently with Stage II sleep being renamed N2, and stages III and IV being named N3. Because most clinicians are familiar with the old staging nomenclature, this is what we have used in this review.

Typically, these events are brief and may include sleeptalking and simple motor behaviours that occur without responsiveness to the environment.³ Because of the sleeper's amnesia, they may only be noted by his or her bed partner. The frequency of events decreases with age;¹¹ persistence into the twenties is usually associated with comorbidities or medications.¹²

Two variants of confused arousals have been described: severe morning sleep inertia (sleep drunkenness, similar to the confusion, disorientation and slowed responses seen on awakening from slow wave sleep) and sleep-related sexual behaviours (commonly referred to as sexsomnia). Sexual behaviours (e.g., vocalizations, fondling, masturbation, intercourse and sexual assault) emerge as part of a confused arousal from sleep, although partial seizures and other pathologies should be considered.¹³

Isolated confusional arousals in adults rarely come to clinical attention. For patients who present with confusional arousals, important measures to maximize sleep stability include improving sleep hygiene, avoiding centrally acting medications or drugs, and preventing sleep deprivation. The use of benzodiazepines¹⁴ and specific serotonin reuptake inhibitors (SSRIs),¹⁵ in conjunction with education about sleep and stress management and taking appropriate precautions (Box 4), may be effective. Anecdotal evidence suggests that scheduled awakenings (setting the alarm for 1 hr after sleep onset) can be helpful.¹⁶

Patients with confusional arousals (as well as those with other parasomnias) should be counselled to not co-sleep with children, because even a single event has the potential for very serious consequences. It is also useful for patients to let others know of the disorder if they are going on a group trip (e.g., business trip, camping).

Box 3: Clinical questions to aid in the diagnosis of parasomnia

- 1. Rule out sleep deprivation
 - Time in and out of bed and total sleep time, ideally from a one-week sleep diary
 - b. Exclude external causes of sleep loss
 - i. Elective
 - ii. Shift work
 - iii. Caregiving: young children, ill family member, pets
- 2. Rule out effects of intoxication or withdrawal
 - a. Alcohol use
 - i. Current pattern of use
 - ii. Any binge drinking?
 - b. Substance use
 - i. Current pattern of use
 - ii. Intoxication with stimulants
 - iii. Withdrawal from sedatives
- 3. Rule out sleep disorders causing sleep instability
 - a. Sleep apnea
 - b. Periodic limb movement disorder
 - c. Restless legs syndrome
 - d. Narcolepsy
- 4. Rule out medical disorders or treatments associated with sleep instability
 - a. Syndromes causing pain or discomfort at night
 - b. "Alerting" medications (e.g., second-generation antidepressants [selective serotonin reuptake inhibitor, serotonin–norepinephrine reuptake inhibitor], stimulants, dopamine agonists)
- 5. Confirm presence of NREM parasomnias in other family members and during the patient's childhood
 - a. In the absence of such a history, expand the screen to include other pathologies, including nocturnal seizures
- 6. Timing of events
 - a. NREM parasomnias predominate in the first third of the night, REM parasomnias in the last third
- 7. Morphology of events
 - a. Stereotypical, brief events are more likely to be ictal
 - b. Marked autonomic arousal is characteristic of sleep terrors
 - c. Confusion on awakening is likely with NREM parasomnias but absent with REM parasomnias
 - d. Eyes tend to be open in NREM parasomnias but closed in REM parasomnias
 - e. Amnesia of the event is characteristic of NREM parasomnias, but REM parasomnias are characterized by recall of dreams or dream fragments

Note: NREM = nonrapid eye movement, REM = rapid eye movement.

Sleepwalking

Sleepwalking is the result of a series of complex behaviours that occur while the patient is in an altered state of consciousness. A spectrum of activity may be seen; the most simple events include sitting up in bed in a confused manner before walking. Usually the individual's eyes are open (in contrast, the eyes remain closed in REM parasomnias).¹

Agitated ambulation is more common in older sleepwalkers, is often associated with a perceived threat or dreams promoting "fight or flight" behaviour, and may result in injury to bystanders. Rarely, sleepwalking may lead to injury or death for the sleeper¹⁷ or others.¹⁴

The co-occurrence of predisposing factors is required to precipitate sleepwalking episodes, and sleepwalking is unlikely in the absence of one or more of these factors. ¹⁸ Genetic susceptibility is the predominant predisposing factor, and sleep deprivation, touch, noise, stress, alcohol, medications and fever are potential triggers.

The cause of sleepwalking is unknown, although the pathophysiology clearly involves slow wave sleep. Although the macroarchitecture of sleep in people who sleepwalk is essentially normal, there may be more fragmentation of slow wave sleep. Single photon emission computed tomography during an episode of sleepwalking supports the co-occurrence of sleeping

and waking states caused by selective activation and hypoactivation of neural circuits.²⁰

In addition to the usual safety precautions (Box 4), the patient can be counselled to avoid sleep deprivation, and comorbid sleep disorders (e.g., obstructive sleep apnea²¹) can be treated via nonpharmacologic and pharmacologic interventions. Scheduled awakenings, psychotherapy, relaxation exercises and hypnosis may be helpful,²² but pharmacotherapy is often required for patients with frequent or hazardous episodes (arbitrarily defined as more than 50% of nights). Clonazepam is commonly used, with more limited evidence for other benzodiazepines, imipramine, paroxetine and melatonin.¹⁶

Sleep terrors

Sleep terrors — events typically lasting between 30 seconds and 5 minutes²³ — commence with the sleeper sitting up in bed, emitting a loud scream and showing the typical autonomic manifestations of intense fear. Associated dream imagery may be reported and incoherent vocalizations can occur; the sleeper may bolt from the bed and run in an apparent attempt to avoid harm, thereby injuring themselves or others.²⁴

The pathophysiology of sleep terrors is unknown, but it is assumed that slow wave sleep instability accounts for these disturbances. There is an association between sleep terrors and psychiatric disorders; 10,25 sleepers with night terrors exhibit a high level of depression, anxiety, and obsessive—compulsive and phobic traits on the Minnesota Multiphasic Personality Inventory. 25 Tricyclic antidepressants and benzodiazepines may be effective and should be considered if the sleep terrors are potentially harmful. 4

Sleeptalking

Sleeptalking or somniloquy, classified in ICSD under "sleep disorders associated with conditions classified elsewhere," is a common parasomnia that ranges from isolated speech to full conversations without recall. It occurs in all sleep states and can be associated with obstructive sleep apnea, other disorders of arousal and REM-related parasomnias.²³ There are no specific treatments, and the clinical approach is to eliminate or reduce the conditions with which it is associated.

Sleep-related eating disorder

This parasomnia is classified in DSM-V under sleepwalking,² whereas ICSD classifies it as "other parasomnias." Sleep-related eating disorder is best conceptualized as the combination of the binge eating of bulimia nervosa with the disordered arousal, confused behaviour and amnesia (partial or full) of a parasomnia arising from NREM sleep.¹⁰ Food and

beverages are consumed in a hurried and out-ofcontrol manner, with high-calorie foods being preferred. Sometimes unusual (e.g., slabs of butter) or toxic foodstuffs (e.g., uncooked chicken) may be ingested. Variable, diminished awareness and recall distinguishes sleep-related eating disorder from nocturnal eating syndrome,²⁶ in which excessive amounts of food are eaten before or during the sleep period in full consciousness.

The pathophysiology of sleep-related eating disorder is unclear, but it is associated with other sleep disorders²⁷ including sleepwalking, restless legs syndrome, periodic limb movement disorder, obstructive sleep apnea and narcolepsy²⁸ and medications, including zolpidem.²⁹ Successful treatment of the associated sleep disorder can substantially improve this parasomnia.

Management of sleep-related eating disorder usually requires specialist care with attention to safety (because of the potential ingestion of toxic foods). First steps include normalizing the patient's eating schedule, preventing sleep deprivation and minimizing alcohol and substance use. If medication is required, the current preferred choice by experts is topiramate, with SSRI agents and dopamine agonists being second-line options.³⁰

Parasomnias usually associated with REM sleep

There are 3 parasomnias associated with REM sleep: REM sleep behaviour disorder, recurrent isolated sleep paralysis and nightmare disorder.

REM sleep behaviour disorder

Two clinical forms of REM sleep behaviour disorder are seen: acute and chronic.³¹ The acute form is associated with specific medications and substance abuse or withdrawal, and the chronic form is subdivided into either idiopathic or secondary to a neurologic disease.⁵

Box 4: Safety precautions for patients with parasomnia

- 1. Identify, then reduce or eliminate known precipitating factors
- 2. Avoid sleep deprivation: maintain a regular sleep—wake schedule with a constant waking time
- 3. Limit, preferably eliminate, the use of alcohol and recreational drugs
- 4. Maximize safety of the sleeping environment
 - i. Sleep on the lowest floor in the house
 - ii. Use a mattress on the floor
 - Sleep solo or use two queen-sized mattresses pushed together for co-sleeping
 - iv. Minimize or pad bedside furniture
 - v. Secure bedside light above bed but out of reach
 - vi. Use plastic cups or bottles, if bedside water is required
 - vii. Consider child-proof door knobs, door wedges or alarms
 - viii. Remove or lock any weapons or dangerous household items

Rapid eye movement sleep behaviour disorder is characterized by activity in REM sleep causing injury or sleep disruption. Polysomnography is required to confirm the associated electromyographic abnormalities. In REM sleep behaviour disorder, there is a loss of REM atonia with excessive muscle tone, twitches on electromyography, and movements that often appear to be dream enactment, potentially causing injury to the sleeper or bed partner. Episodes usually occur about once a week, but may be as often as four times nightly over consecutive nights. In contrast to all NREM parasomnias, the sleeper awakens rapidly without confusion and often with good recall of a dream that corresponds with the enacted behaviours that can include talking, laughing, swearing, shouting, reaching, grabbing, flailing, punching, hitting or running.⁵

Because REM sleep behaviour disorder emerges from REM sleep, which is maximal in the last half of the night, this is the time when events are most likely to occur. Patients with narcolepsy, a condition characterized by REM periods at sleep onset, are at risk of REM sleep behaviour disorder, and their events can occur earlier in the night.⁵

Rapid eye movement sleep behaviour disorder is considered to be a long-standing and progressive disorder, although an acute form can emerge during REM sleep rebound states (e.g., the first night on continuous positive airway pressure for obstructive sleep apnea), withdrawal from alcohol, sedative hypnotic agents or REM suppressant medications.⁵

The major predisposing factors are male sex, age greater than 50 years, and an underlying neurologic disease, particularly Parkinson disease, Lewy body dementia and stroke.⁵ Rapid eye movement sleep behaviour disorder is a wellestablished risk factor for neurodegenerative disease, with studies indicating that 40%–65% of patients with this disorder develop a defined neurodegenerative phenotype within the subsequent 10 years.³² Narcolepsy and post-traumatic stress disorder are associated with REM sleep behaviour disorder,¹ as are the use of SSRI and serotonin norepinephrine reuptake inhibitors.³³

The exact pathophysiology of REM sleep behaviour disorder is unclear, but it may be caused by reduced dopamine transporters in the striatum³⁴ and diminished striatal dopaminergic innervation.³⁵ The onset may be gradual or rapid and the course is progressive; spontaneous remissions are very rare, although dream enactment may subside during the late stages of a neurodegenerative disorder.

Rapid eye movement sleep behaviour disorder can be diagnosed based on clinical history, which should include the bed partner's account. This is the only parasomnia that can be readily

confirmed by polysomnography, which shows an excessive amount of sustained electromyographic activity in REM sleep. Of patients with REM sleep behaviour disorder, 70% will have periodic limb movements during NREM sleep.³⁶

As with other parasomnias, there have been no large trials of treatment for this condition, but case series have shown that clonazepam is most commonly used for this condition.⁵ Screening for obstructive sleep apnea should be undertaken before starting treatment with clonazepam. The most troublesome adverse effect associated with clonazepam therapy is morning confusion and memory impairment.⁵ Melatonin in high doses is an alternative to clonazepam, and there is some anecdotal support for the use of dopamine agonists such as pramipexole.⁵ A novel alarm system for patients unresponsive to medications has been described recently.³⁷

Sleep paralysis

Sleep paralysis is the inability to perform voluntary movements at sleep onset or on awakening.1 Although consciousness is preserved and recall is present, the patient is unable to speak or move at first. Respiration is unaffected, although patients may feel as if they are unable to breathe because the accessory respiratory muscles are atonic, but diaphragmatic breathing is maintained. Episodes last seconds to minutes and resolve spontaneously, but they can be aborted if the patient is touched or spoken to. The frequency can vary from once-ina-lifetime to several times in a year. During the first few episodes, intense anxiety accompanies the paralysis. Auditory, visual or tactile hallucinations can occur and may accompany a perception of an ominous presence in the room.1

Sleep deprivation, irregular sleep—wake schedules (e.g., from jet lag or shift work) and stress are predisposing factors, and the condition is more likely to occur when the sleeper is in the supine position. These factors may facilitate a state dissociation in which elements of REM sleep persist into wakefulness causing the phenomenon.¹

Causes of secondary sleep paralysis (narcolepsy and other neuropsychiatric illnesses) should be excluded through a detailed physical examination, medical, psychiatric and sleep history, and appropriate investigations. Patient education and ensuring adequate, regular sleep periods are important. If the episodes are frequent, the patient should practise moving his or her little finger during drowsy sleep, because this action, if performed during an episode, may abort it. There have been no trials of psychologic or pharmacologic interventions, but SSRIs may be helpful because they suppress REM sleep and are well tolerated. 38,39

in better recall and reflect the admixture of REM sleep with its associated dream imagery, voluntary muscle atonia and wakefulness. In this group of parasomnias, clinical experience has shown that the same advice about sleep hygiene, avoiding substance use and preventing sleep deprivation used in NREM is helpful. Clonazepam, pramipexole and melatonin are the medications commonly used to manage REM sleep behaviour disorder.

Parasomnias are not benign conditions and potentially place the sleeper and bed partner at risk of injury and may be associated with other disorders that require careful consideration. Parasomnias can be successfully managed by appropriate investigation, education and the strategic use of psychologic and pharmacologic therapies.

References

- International classification of sleep disorders: diagnostic and coding manual. 2nd ed. Westchester (IL): American Academy of Sleep Medicine; 2007.
- Diagnostic and statistical manual of mental disorders, 5th edition: DSM-5, Arlington (VA): American Psychiatric Association, 2013.
- Plante DT, Winkelman JW. Parasomnias. Psychiatr Clin North Am 2006;29:969-87.
- Kotagal S. Parasomnias in childhood. Sleep Med Rev 2009;13: 157-68.
- Aurora RN, Zak RS, Maganti RK, et al. Best practice guide for the treatment of REM sleep behavior disorder (RBD). J Clin Sleep Med 2010;6:85-95.
- Lange CL. Medication-associated somnambulism. J Am Acad Child Adolesc Psychiatry 2005;44:211-2.
- Iber C, Ancoli-Israel S, Chesson A, et al.; the American Academy of Sleep Medicine. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. Westchester (II): American Academy of Sleep Medicine; 2007.
- Aurora RN, Zak RS, Auerbach SH, et al. Best practice guide for the treatment of nightmare disorder in adults. J Clin Sleep Med 2010:6:389-401.
- Zucconi M, Oldani A, Ferini-Strambi L, et al. Arousal fluctuations in non-rapid eye movement parasomnias: The role of cyclic alternating pattern as a measure of sleep instability. J Clin Neurophysiol 1995;12:147-54.
- 10. Goldstein CA. Parasomnias. *Dis Mon* 2011; 57:364-88.
- Ohayon MM, Guilleminault C, Priest RG. Night terrors, sleepwalking, and confusional arousals in the general population: their frequency and relationship to other sleep and mental disorders. J Clin Psychiatry 1999;60:268-76.
- Ohayon MM, Priest RG, Zulley J, et al. The place of confusional arousals in sleep and mental disorders — findings in a general population sample of 13 057 subjects. J Nerv Ment Dis 2000;188:340-8.
- Andersen ML, Poyares D, Alves RS, et al. Sexsomnia: abnormal sexual behavior during sleep. *Brain Res Rev* 2007;56:271-82.
- Siclari F, Khatami R, Urbaniok F, et al. Violence in sleep. *Brain* 2010;133:3494-509.
- Béjot Y, Juenet N, Garrouty R, et al. Sexsomnia: an uncommon variety of parasomnia. Clin Neurol Neurosurg 2010;112:72-5.
- Attarian H. Treatment options for parasomnias. Neurol Clin 2010; 28:1089-106.
- Seeman MV. Sleepwalking, a possible side effect of antipsychotic medication. *Psychiatr Q* 2011;82:59-67.
- Pressman MR. Factors that predispose, prime and precipitate NREM parasomnias in adults: clinical and forensic implications. Sleep Med Rev 2007;11:5-30.
- Espa F, Ondze B, Deglise P, et al. Sleep architecture, slow wave activity, and sleep spindles in adult patients with sleepwalking and sleep terrors. Clin Neurophysiol 2000;111:929-39.
- Bassetti C, Vella S, Donati F, et al. SPECT during sleepwalking. Lancet 2000;356:484-5.
- Guilleminault C, Kirisoglu C, Bao G, et al. Adult chronic sleepwalking and its treatment based on polysomnography. *Brain* 2005;128:1062-9.
- Harris M, Grunstein RR. Treatments for somnambulism in adults: assessing the evidence. Sleep Med Rev 2009;13:295-7.
- Avidan AY, Kaplish N. The parasomnias: epidemiology, clinical features, and diagnostic approach. Clin Chest Med 2010;31:353-70.

- Pressman MR. Disorders of arousal from sleep and violent behavior: the role of physical contact and proximity. Sleep 2007; 30:1039-47.
- Kales JD, Kales A, Soldatos CR, et al. Night terrors. Clinical characteristics and personality patterns. Arch Gen Psychiatry 1980;37:1413–7.
- O'Reardon JP, Peshek A, Allison KC. Night eating syndrome: diagnosis, epidemiology and management. CNS Drugs 2005; 19:997-1008.
- Howell MJ, Schenck CH. Treatment of nocturnal eating disorders. Curr Treat Options Neurol 2009;11:333-9.
- Palaia V, Poli F, Pizza F, et al. Narcolepsy with cataplexy associated with nocturnal compulsive behaviors: a case-control study. Sleep 2011;34:1365-71.
- Hoque R, Chesson AL Jr. Zolpidem-induced sleepwalking, sleep related eating disorder, and sleep-driving: fluorine-18flourodeoxyglucose positron emission tomography analysis, and a literature review of other unexpected clinical effects of zolpidem. J Clin Sleep Med 2009;5:471-6.
- Howell MJ, Schenck CH. Treatment of nocturnal eating disorders. Curr Treat Options Neurol 2009;11:333-9.
- Schenck CH, Mahowald MW. REM sleep behavior disorder: clinical, developmental, and neuroscience perspectives 16 years after its formal identification in SLEEP. Sleep 2002;25:120-38.
- Postuma RB, Gagnon JF, Montplaisir J. Rapid eye movement disorder as a biomarker for neurodegeneration: the past 10 years. Sleep Med 2013;14:763-7.
- Hoque R, Chesson AL Jr. Pharmacologically induced/exacerbated restless legs syndrome, periodic limb movements of sleep, and REM behavior disorder/REM sleep without atonia: literature review, qualitative scoring, and comparative analysis. J Clin Sleep Med 2010;6:79-83.
- 34. Eisensehr I, Linke R, Tatsch K, et al. Increased muscle activity during rapid eye movement sleep correlates with decrease of striatal presynaptic dopamine transporters. IPT and IBZM SPECT imaging in subclinical and clinically manifest idiopathic REM sleep behavior disorder, Parkinson's disease, and controls. Sleep 2003;26:507-12.
- Albin RL, Koeppe RA, Chervin RD, et al. Decreased striatal dopaminergic innervation in REM sleep behavior disorder. *Neurology* 2000:55:1410-2.
- 36 Fantini ML, Michaud M, Gosselin N, et al. Periodic leg movements in REM sleep behavior disorder and related autonomic and EEG activation. *Neurology* 2002:59;1889-94.
- Howell MJ, Arneson PA, Schenck CH. A novel therapy for REM sleep behavior disorder (RBD). J Clin Sleep Med 2011;7: 639-644A.
- Koran LM, Raghavan S. Fluoxetine for isolated sleep paralysis. Psychosomatics 1993;34:184-7.
- McCarty DE, Chesson AL Jr. A case of sleep paralysis with hypnopompic hallucinations. Recurrent isolated sleep paralysis associated with hypnopompic hallucinations, precipitated by behaviorally induced insufficient sleep syndrome. J Clin Sleep Med 2009;5:83-4.
- Gehrman PR, Harb GC. Treatment of nightmares in the context of posttraumatic stress disorder. J Clin Psychol 2010;66:1185-94.
- Krakow B, Zadra A. Clinical management of chronic nightmares: imagery rehearsal therapy. Behav Sleep Med 2006;4:45-70.
- Hansen K, Höfling V, Kröner-Borowik T, et al. Efficacy of psychological interventions aiming to reduce chronic nightmares: a meta-analysis. Clin Psychol Rev 2013;33:146-55.
- Calohan J, Peterson K, Peskind ER, et al. Prazosin treatment of trauma nightmares and sleep disturbance in soldiers deployed in Iraq. J Trauma Stress 2010;23:645-8.
- 44. Nevéus T. Sleep enuresis. Handb Clin Neurol 2011;98:363-9.
- von Gontard A, Baeyens D, Van Hoecke E, et al. Psychological and psychiatric issues in urinary and fecal incontinence. *J Urol* 2011; 185:1432-6.
- 46. Fleming J. Dissociative episodes presenting as somnambulism: a case report. *Sleep Res* 1987;16:263.
- Pearce JM. Clinical features of the exploding head syndrome. J Neurol Neurosurg Psychiatry 1989;52:907-10.
- Palikh GM, Vaughn BV. Topiramate responsive exploding head syndrome. J Clin Sleep Med 2010;6:382-3.

Affiliations: Department of Medicine (Fleetham) and Department of Psychiatry (Fleming), University of British Columbia; Sleep Disorders Program (Fleetham), University of British Columbia Hospital, Vancouver, BC.

Contributors: Jonathan Fleming performed the literature review. Both authors drafted the article, revised it critically for important intellectual content and approved the version submitted for publication.