BMJ Best Practice

Parasomnias in adults

Straight to the point of care

Last updated: Jan 26, 2024

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Summary

Parasomnias are undesirable events occurring during sleep or during the transition into or out of sleep. They may occur during non-rapid eye movement or rapid eye movement sleep, depending on the type of parasomnia.

They include abnormal behaviours (e.g., sleep-related eating) or dreams (e.g., nightmare disorder).

Diagnosis relies mainly on careful history from the patient or, more commonly, the bed partner or other household members.

Treatment is indicated if the parasomnia may result in injury to patient or bed partner or if sleep is significantly disrupted.

Management depends on the type of parasomnia and includes reassurance, sleep environment modification, cognitive therapy, and pharmacotherapy.

Definition

Parasomnias are undesirable sleep-related events that may occur during sleep or during the transition into sleep or out of sleep. They include abnormal behaviours (e.g., sleepwalking) and dreams (e.g., nightmares). Parasomnias may be transient and have no significant consequences on the patient's sleep health. However, in some cases they are severe or persistent enough to cause significant sleep disruption and distress or injury to the patient or bed partner.[1]

Epidemiology

In general, non-rapid eye movement (NREM) parasomnias are more prevalent in children and occur in people less than 50 years old.[1]

NREM sleep parasomnias

- Confusional arousals have a lifetime prevalence reported as 18.5% and among adults over 15 years prevalence is 2.9% to 4.2%.[2]
- Sleepwalking is a common parasomnia in childhood, occurring in up to 2% of the population.[3] In adults, the prevalence is about 1% to 4%.[4] [5] Most children outgrow the parasomnia in their mid-teens. Sex does not appear to play a role in European studies, and this is also believed to hold true for the US population.[6]
- Sleep terrors affect approximately 6.5% of children.[5] Prevalence in adults has been reported at 2.3% to 2.6% in people aged 15-64 years, and 1% in people aged 65 years and older.[2] Among children, sleep terrors are more common in boys than in girls but no sex difference is reported in adults.[1]
- Sleep-related eating disorder has been recorded in 5% of a sample 700 people, including outpatients and inpatients with an eating disorder, obese people in a trial for an anorexic agent, depressed patients in an antidepressant trial, and a group of unselected college students.[7] Randomly picked controls (who did not have an eating disorder history, were not part of the cohort in a trial for an anorexic agent, and were not depressed patients taking an antidepressant) formed the unselected college student group in the trial. The highest prevalence was noted in the inpatient eating disorders group (16.7%), followed by the outpatient eating disorders group (8.7%); 4.6% of the unselected student population also reported the problem.[7]

Rapid eye movement (REM) parasomnias

- Rapid eye movement sleep behaviour disorder (RBD) is a type of REM parasomnia. It has an estimated prevalence of 0.68% (0.29% to 1.15%) when confirmed by polysomnography (PSG). The prevalence of probable RBD (by history and not PSG-confirmed) is higher at 4% to 6%, but the higher estimate may be due to false positives.[8] Exact incidence is unknown. This parasomnia typically affects older men, but when it occurs in younger people, men and women are equally affected.[2]
- Recurrent isolated sleep paralysis prevalence rates have been noted to differ across nationalities in comparative studies.[2] One study reported a prevalence of 11.4% in American students, compared with 19.2% and 20.7% in students from Kuwait and Sudan, respectively.[9] Cultural differences may explain some of the variability in prevalence. In Asian cultures, the 'ghost oppression' phenomenon (kanashibari) corresponds to the symptoms recognised in the western hemisphere as sleep paralysis. More accurate prevalence estimates can be obtained by accounting for such differences when study questionnaires are prepared.[10] Although often reported by younger subjects, isolated sleep paralysis may be under-recognised in older people. A prevalence of almost 18% for isolated sleep paralysis symptoms has been reported in a cohort of Chinese patients >70 years of age.[11] One study documented the overall prevalence to be between 2.3% and 40% and reported a 2 to 1 female to male ratio.[12]
- Nightmare disorder is more prevalent in children. It also occurs more frequently in women than in men, with an overall prevalence of 5% to 8% in adults.[13] [14]

Other parasomnias

- Exploding head syndrome has been reported at all ages, but seems to be particularly prevalent in younger adults; occurring in about 16.6% of college students. It is more common among those who

also experience sleep paralysis. Overall prevalence in the general population and global trends in epidemiology are unknown. Whether or not it is more common in women remains controversial.[15]

- Sleep-related hallucinations prevalence estimates are available from studies in non-institutionalised European people, and prevalence in the US is likely to be similar. A large cohort of >15,000 people in the UK, Germany, and Italy, aged ≥15 years, reported hallucinations at sleep onset (24.8%) and on awakening (6.6%). Younger patients are more likely to be affected, and women may be slightly more at risk. There are no known global trends in epidemiology.[16]
- The prevalence of enuresis varies widely with age, from 2% in older adults to 30% in school-age children.[17]

Aetiology

The following parasomnias do not have a known aetiology but are considered disorders of arousal:

- Confusional arousals
- Sleep terrors
- Sexsomnia
- Exploding head syndrome
- Sleep-related hallucinations
- Sleep-related eating disorder

Nightmares are postulated to be simulations of threatening events and to serve a rehearsal function important for survival.[2][18] Recurrent nightmares are thought to occur due to a combination of impaired fear extinction and hyperarousal, which result in a nightmare script that is replayed and causes distress.[2][18][19]

Enuresis is associated with central nervous system immaturity, disorders of arousal, increased fluid intake, urinary problems both functional and structural, male sex, maternal smoking, mother's age less than 20 at the time of the child's birth, psychosocial stressors, and attention deficit hyperactivity disorder.[20] Sleepwalking has been associated with the human leukocyte antigen gene DQB1.[21]

Rapid eye movement sleep behaviour disorder (RBD) can occur in an isolated form or secondary to other aetiologies such as neurodegenerative diseases, narcolepsy, and other disorders that impact the brainstem nuclei involved in rapid eye movement (REM) sleep. The isolated RBD often precedes the development of neurodegenerative disorders, especially the synucleinopathies, in which insoluble protein (alpha-synuclein) is deposited in brain tissue.[22] The prototype condition in this category is Parkinson's disease.[23]

RBD symptoms may also be precipitated by medicines including tricyclic antidepressants, selective serotonin-reuptake inhibitors, cholinergic agents, and monoamine oxidase inhibitors.[24] Recurrent isolated sleep paralysis does not have a known aetiology and may be associated with sleep deprivation (e.g., irregular sleep-wake schedules, mental stress, anxiolytic medication).[2] [25]

Pathophysiology

The precise pathophysiology is unknown for the following:

- Exploding head syndrome
- Nightmare disorder
- Sleep-related eating disorder
- Enuresis

Non-rapid eye movement (NREM) sleep parasomnias

- Confusional arousals and sleepwalking are characterised by NREM sleep instability, noted even on the non-sleepwalking nights. In electroencephalographic-neuroimaging studies, sleepwalking motor events are also associated with arousal-related activation of cingulate motor area.[26] This is similar to the instability noted in patients with the upper airways resistance syndrome.[27]
- High levels of fragmentation of slow-wave sleep underlie the pathophysiology of sleep terrors and sleepwalking.[28]
- Patients with sleep-related eating disorder tend to share psychological characteristics of people with daytime eating disorders, and they tend to have mildly fragmented sleep and reduced sleep efficiency.[29]
- Patients with sleepwalking have simultaneous wakefulness originating from the motor and cingulate cortices and persistent sleep in associative cortical regions. There is a decline in gray matter volume in the dorsal posterior and posterior midcingulate cortices in sleepwalkers representing a neuroanatomical correlate to this pathophysiological finding.[30]

Rapid eye movement (REM) sleep parasomnias

- RBD is a type of REM sleep parasomnia and its pathophysiology is complex, but early research supports the hypothesis that lesions of the tegmentum of the pons may result in loss of atonia and motor activity during REM sleep.[31] It is postulated that the pontine tegmentum accommodates an atonia mechanism and a mechanism that suppresses brainstem motor pattern generation. Different behaviours (atonia alone versus atonia with more complex, unsuppressed motor activity) could therefore be expected depending on the precise location of injury.
- Recurrent isolated sleep paralysis arises from REM sleep, and may be viewed as a dissociated state in which REM sleep elements persist into wakefulness.[2]

Other parasomnias

- Although REM dream intrusion into the wake-sleep-wake transition is presumed to occur in sleep-related hallucinations, the exact pathophysiology is also unknown.[2]

Classification

The international classification of sleep disorders (3rd edition), text revision (ICSD-3-TR)[2]

1) Non-rapid eye movement (NREM) parasomnias

- Confusional arousals: characterised by confusion noted when the patient arouses or is aroused from sleep, usually slow-wave sleep.
- Sleepwalking: results in patient ambulation during sleep. Sleep or an altered level of consciousness will persist during the episode.
- Sleep terrors: presents with manifestations of fear and autonomic hyperactivity (e.g., tachycardia, pupillary dilation, increased blood pressure) during an arousal from slow-wave sleep. Often heralded by a loud vocalisation sometimes described as a blood-curdling scream.
- Sleep-related eating disorder: patients eat and/or drink after sleep arousal. The behaviour is involuntary and may or may not be subsequently recalled.

2) Rapid eye movement (REM) parasomnias

- RBD is a type of REM parasomnia manifested by vigorous and even violent behaviour occurring during REM sleep. Associated with abnormal tone during REM sleep and seems to represent vivid dream enactment. Patients often develop neurodegenerative disorders that are usually synucleinopathies, such as Parkinson's disease. A variant of RBD also exists that presents with both dream-enacting behaviour out of REM sleep and either confusional arousal or sleepwalking, and tends to happen in a younger group of subjects without a clear association with synucleinopathies. It is called parasomnia overlap syndrome.
- Recurrent-isolated sleep paralysis: results in the inability to speak or move at the time of sleep onset or on waking, typically from REM sleep. Diagnosis implies that narcolepsy is not also present and that no other disorders that could result in transient paralysis are present. Typically occurs during REM sleep.
- Nightmare disorder: patients report recurrent unpleasant dreams resulting in sleep disruption. The nightmares usually occur during REM sleep.

3) Other parasomnias

- Exploding head syndrome: patients describe a loud noise or a sensation of explosion in the head occurring during the wake-sleep or sleep-wake transitions.
- Sleep-related hallucinations: may occur at sleep onset (hypnagogic) or on waking from sleep (hypnopompic) and are usually visual experiences.
- Enuresis or bedwetting tends to occur more in children, but adults can suffer from it as well.

Diagnostic and statistical manual of mental disorders, 5th ed., text revision (DSM-5-TR) classification[1]

DSM-5-TR classifies parasomnias as follows:

- Non-rapid eye movement sleep arousal disorders
- Nightmare disorder
- Rapid eye movement sleep behaviour disorder

For further details, see Diagnostic criteria .

Case history

Case history #1

At the insistence of his wife and daughter, a 70-year-old white man presents with abnormal sleep behaviour. The wife reports that the patient punches her vigorously and shouts loudly in his sleep. The patient believes that the problem stems from acting out dreams in which he is chased by a group of men and has to defend himself when cornered by 'throwing punches at them'. After a severe episode in which he dived out of bed and sustained a forehead laceration, he decided to tie himself to the bed and his family insisted that he see his physician. His examination reveals decreased facial expression, some cogwheel rigidity and bradykinesia of the upper extremities (right greater than left), and shuffling of gait. Polysomnography with additional forearm EMG electrodes noted rapid eye movement (REM) sleep without atonia. The patient was diagnosed with REM sleep behaviour disorder and Parkinson’s disease.

THEORY

Case history #2

A 30-year-old man presents with his wife, who complains that he walks in his sleep. She reports that about 1 to 2 hours into sleep she is woken up by her husband leaving the bed and walking around the room. This happens once or twice a month. On 2 occasions he tripped on furniture in the room. She reports that she has been unable to abort these episodes by redirecting him and talking to him because he seems to be dazed and unresponsive. The patient himself goes back to bed and falls asleep after about 10 minutes of aimless wandering around the room. The patient has no recollection of any of these events. However, he does report that sleepwalking is extremely common in his family, and when he was younger he was observed walking in his sleep more commonly than at present. The patient underwent polysomnography which did not show sleep apnoea or epileptiform abnormalities. The patient was diagnosed with sleepwalking and improved with bedroom environment safety measures, avoiding sleep deprivation, and cognitive behavioural therapy.

Other presentations

Parasomnias may be transient and have no significant consequences on the patient's sleep health. In other cases they are severe or persistent enough to cause significant sleep disruption and distress or injury to the patient or bed partner.

Approach

The diagnosis of a parasomnia is based on a thorough history, usually from the bed partner. The diagnosis will help to guide the subsequent careful selection of tests.

Presence of risk factors

In general, parasomnias are more common in children than in adults, especially non-rapid eye movement (NREM) parasomnias.[1] Rapid eye movement sleep behaviour disorder (RBD) and sleep-related cramps are more common at an older age. RBD is more common in men, while nightmare disorder is more common in women.[13] [32] Parasomnias are frequently reported to occur after a forced awakening from sleep.

It is important to enquire about a family history of sleep disorder, as this is a very common finding in sleepwalking, and also occurs with confusional arousals and sleep terrors.[1] The presence of the human leukocyte antigen gene DQB1 is strongly associated with sleepwalking.[21]

Other primary sleep disorders, such as obstructive sleep apnoea, sleep deprivation, and periodic limb movement disorders should be screened for, as these typically cause parasomnias.

Nightmare disorder is strongly associated with chronic or acute stress and psychiatric disorders, particularly post-traumatic stress disorder, anxiety, bipolar disorder, and depression.[18] [46]

Certain medicines and pharmacological agents are strongly associated with particular disorders: venlafaxine and mirtazapine with RBD; noradrenaline, serotonin, and dopamine with nightmares and other sleep disturbances.[18] [40][43] [44] [47] Neurodegenerative disorders such as Parkinson's disease are strongly associated with RBD, as is narcolepsy, and sleep-related eating disorders have been reported to occur more frequently in people receiving treatment for a known eating disorder.[39] People with sleep-related eating disorders are also more likely to have a history of sleepwalking, sleeptalking, and periodic limb movements of sleep.[45]

History

The history is usually obtained from the bed partner when available. Patients with parasomnias may be reported to arouse from sleep and exhibit disturbed cognition, behaviour, or both. Mental slowing, disorientation, and memory problems, as well as speech disturbances, are noted in confusional arousals.

Vigorous or even violent behaviour may be present in confusional arousals, sleepwalking, sleep terrors, and RBD.[1] [5] The sensation of a sudden loud noise in the head in exploding head syndrome may be virtually diagnostic.[48] [49]

There are some features that can help distinguish NREM parasomnia and RBD. With NREM parasomnia, the events are typically in the first 1-2 hours of sleep onset, the eyes are often open during the event, and there is little to no dream recall. In contrast, RBD behaviours can happen throughout the sleep period but are typically after the first hour and most often late in the sleep period, when rapid eye movement sleep predominates. The eyes are closed, and there are often vivid dreams associated with the more vigorous events.

Witnesses of a person experiencing sleep terrors or nightmares may describe how frightened the patient appears to be. This is manifested in signs of autonomic hyperactivity such as tachycardia, tachypnoea, and pupillary dilation.

In recurrent isolated sleep paralysis, episodes of inability to move and chest heaviness are described. Careful questioning for symptoms of narcolepsy such as cataplexy and hypnagogic and hypnopompic hallucinations is warranted before a diagnosis of recurrent isolated sleep paralysis is made.

Other important behavioural data in parasomnias include the eating behaviour noted in sleep-related eating disorder. The patient may recall eating behaviour during the night, or the parasomnia may be described by a spouse, bed partner, or other household member.

In community-based surveys with RBD screening questionnaires, the chance of probable RBD was higher in those with lower educational level and lower socioeconomic status, as well as in those who had more risk factors for cardiovascular disease.[50]

Physical examination

In most parasomnias, the examination in the surgery is normal. However, there may be evidence of external injuries pointing towards a violent parasomnia such as RBD. People with RBD often develop neurodegenerative disorders that are usually synucleinopathies, such as Parkinson's disease.[37] [51] Therefore, people with RBD may have manifestations of these disorders, including the following:

- Slowness of thought
- Memory difficulties
- Decreased facial expression
- Extra-ocular movement abnormalities
- Resting tremor
- Bradykinesia
- Rigidity of the extremities, possibly with cog-wheeling
- Gait abnormalities such as shuffling and instability
- Chorea

Overnight polysomnography (PSG)

If any investigations are required to confirm the diagnosis of a particular type of parasomnia, the first step would most frequently be overnight PSG with extended electroencephalographic (EEG) montage to rule out atypical presentation of seizures. PSGs for NREM parasomnias are higher yield if leg electrodes are included as well as psychotropic medications stopped.[52] Expanded EEG electrodes during the PSG are useful to evaluate for sleep-related hypermotor epilepsy. In some parasomnias, the diagnosis is not obvious from the history alone, and many would suggest that if the behaviour is bothersome enough to treat, then PSG evaluation is indicated. PSG is also indicated if there is a suspicion of a primary sleep disorder such as obstructive sleep apnoea or periodic limb movement disorder. PSG should be requested when a complaint of leg kicking is voiced by the patient's bed partner. PSG is essential if RBD is suspected.[53] In RBD, PSG shows evidence of increased electromyography tone during episodes of rapid eye movement sleep and evident abnormal behaviour documented on video monitoring during an episode. This may include shouting and swearing, kicking, punching, and even jumping out of bed. PSG is not required for the diagnosis of confusional arousals, sleepwalking, sleep terrors, or sleep-related eating disorder, but can help. It is not required for nightmare disorder, sleep-related hallucinations, or exploding head syndrome.[2] [54]

PSG can aid diagnosis in unusual cases and may reveal comorbid conditions that increase the likelihood of parasomnia events. Although PSG is not necessarily required for the diagnosis of confusional arousals, sleepwalking, sleep terrors, or sleep-related eating disorder, it can have a very high diagnostic yield.[55]

In recurrent isolated sleep paralysis, no tests are necessary unless narcolepsy is suspected. In enuresis, a PSG is required if obstructive sleep apnoea is suspected.

Further investigations

PSG with expanded EEG can be used to determine the presence of epilepsy.[1] However, if the test is not diagnostic, a prolonged EEG for 24-48 hours should be considered if nocturnal seizures are suspected in the differential diagnosis of confusional arousals, sleepwalking, sleep terrors, and RBD.[56] Epileptiform spikes may be noted. In some cases, clinical seizure activity during EEG monitoring may be accompanied by an electrographic seizure. EEG may also be required in enuresis to rule out nocturnal seizures. The frontal lobe epilepsy and parasomnia scale, a validated questionnaire for the diagnosis of nocturnal events, can aid diagnostic confidence between NREM parasomnia and sleep-related hypermotor epilepsy, but it cannot distinguish either of these from RBD.[56]

A urine drug screen is also indicated if drug misuse is suspected.

In sleepwalking episodes, EEG shows increased slow-wave activity and slow oscillation density.[57]

History and exam

Key diagnostic factors

known condition causing sleep fragmentation/deprivation (common)

- Individual types of parasomnia have particular risk factors strongly associated with them. Obstructive sleep apnoea (OSA), enuresis, and periodic limb movement disorder have been found to contribute to sleep fragmentation, sleep deprivation, and the occurrence of parasomnias (mostly non-rapid eye movement parasomnias).[1] OSA and narcolepsy are also associated with rapid eye movement sleep behaviour disorder (RBD); narcolepsy usually occurs in younger adults with RBD.[39] Antidepressants such as venlafaxine (or other serotonin- and noradrenaline-reuptake inhibitors) and fluoxetine (or other selective serotonin-reuptake inhibitors [SSRIs]) can also trigger RBD.[40] [41]

normal physical examination between episodes (common)

- In most parasomnias, the examination in the surgery is normal; however, in rapid eye movement sleep behaviour disorder signs of parkinsonism may exist.

cognitive disturbance during event (confusional arousals, sleep terrors, and sleepwalking) (common)

- Mental slowing, disorientation, memory problems, and speech disturbances are noted in confusional arousals, sleep terrors, and sleepwalking.[5]

cognitive disturbances in between episodes (rapid eye movement sleep behaviour disorder) (common)

- Impaired cognition noted between episodes (during a clinic visit) is a feature of the neurodegenerative disorders that often accompany rapid eye movement sleep behaviour disorder.

sensation of a sudden loud noise in the head (exploding head syndrome) (common)

- Virtually diagnostic of exploding head syndrome.[48] [49]

vigorous or violent behaviour during episode (confusional arousals, sleepwalking, sleep terrors, and rapid eye movement sleep behaviour disorder) (common)

- May accompany confusional arousals, sleepwalking, sleep terrors, and rapid eye movement sleep behaviour disorder.[1] [5]

episodes of inability to move during episode (recurrent isolated sleep paralysis) (common)

- Described in recurrent isolated sleep paralysis.

eating behaviour during the night (sleep-related eating disorder) (common)

- Patient may recall eating behaviour during the night or the parasomnia may be described by a spouse, bed partner, or other household member.

evidence of external injuries (rapid eye movement sleep behaviour disorder) (common)

- Points towards a violent parasomnia such as rapid eye movement sleep behaviour disorder; in such cases, patients may have cognitive problems.

evidence of fear during episode demonstrated by autonomic hyperactivity (sleep terrors, nightmare disorder) (common)

- Tachycardia, tachypnoea, and pupillary dilation are noted in sleep terrors.
- Tachycardia and tachypnoea are common in nightmare disorder.

Other diagnostic factors

abnormal facial expression during episode (confusional arousals, sleepwalking, sleep terrors, rapid eye movement sleep behaviour disorder) (common)

- A dazed, confused look is typical in confusional arousals, sleepwalking, and sleep terrors.
- Decreased facial expression noted between episodes (during a clinic visit) is a feature of the neurodegenerative disorders that often accompany rapid eye movement sleep behaviour disorder.

parkinsonian signs (rapid eye movement sleep behaviour disorder) (common)

- People with rapid eye movement sleep behaviour disorder often develop neurodegenerative disorders that are usually synucleinopathies such as Parkinson's disease.[37] [38]

Risk factors

Strong

history of childhood parasomnias (non-rapid eye movement parasomnias)

- In general, non-rapid eye movement parasomnias are more prevalent in children.

age >60 years (rapid eye movement sleep behaviour disorder)

- Rapid eye movement sleep behaviour disorder is a disorder of older adults, mainly of men.[32]

male sex (rapid eye movement sleep behaviour disorder)

- Men are more commonly affected by rapid eye movement sleep behaviour disorder than women.[32]

female sex (nightmare disorder, isolated recurrent sleep paralysis)

- Nightmare disorder occurs more frequently in women than in men, with an overall prevalence of 5% to 8% in adults.[13] It is more prevalent in children.

family history (confusional arousals, sleepwalking, sleep terrors)

- A familial tendency is often noted in confusional arousals, sleepwalking (very common), enuresis, isolated recurrent sleep paralysis, and sleep terrors.[2]

presence of human leukocyte antigen gene DQB1 (sleepwalking)

- The human leukocyte antigen gene DQB1 has been linked to a sleepwalking tendency.[21]

obstructive and central sleep apnoea (mainly non-rapid eye movement sleep parasomnias)

- Contributes to sleep fragmentation, sleep deprivation, and the occurrence of parasomnias.
- It is likely that sleep-related breathing disorders facilitate the appearance of all parasomnias, especially in otherwise predisposed people.
- Screening and treatment for obstructive sleep apnoea is recommended to decrease the frequency of these parasomnias.
- Central sleep apnoea could also be a precipitating factor, although it is much less common. Screening detects this as well.

enuresis

- Contributes to sleep fragmentation, sleep deprivation, and the occurrence of parasomnias.

periodic limb movement disorder (mainly non-rapid eye movement sleep parasomnias)

- Contributes to sleep fragmentation and the occurrence of parasomnias.
- Screening and treatment for periodic limb movement disorder is recommended to decrease the frequency of parasomnias.

sleep deprivation (sleepwalking and sleep paralysis)

- Strong risk factor for sleepwalking and sleep paralysis, especially in people who are genetically predisposed to these parasomnias.[33]

stress (nightmare disorder)

- People with acute and chronic stress are more likely to develop nightmare disorder.[18]

psychiatric disorders (nightmare disorder, sleep paralysis)

- Patients with nightmare disorder often have an associated underlying psychopathology, particularly PTSD (also, e.g., anxiety, bipolar disorder, depressive disorder).[18]
- PTSD and anxiety are also risk factors for developing isolated sleep paralysis.[34]

neurological disorders (rapid eye movement sleep behaviour disorder)

- People with rapid eye movement sleep behaviour disorder often develop neurodegenerative disorders that are usually synucleinopathies, such as Parkinson's disease.[35] [36] [37] [38] Early-onset rapid eye movement sleep behaviour disorder, occurring prior to age 50, comprises a substantial minority of cases and is typically associated with narcolepsy.[39]

medicines such as venlafaxine, SSRIs (rapid eye movement sleep behaviour disorder, nightmare disorder)

- Acute rapid eye movement sleep behaviour disorder may be precipitated by venlafaxine, SSRIs, and other antidepressants.[40] [41]
- Rapid eye movement sleep behaviour disorder has also been observed with mirtazapine prescribed for insomnia and depression in people with parkinsonism.[42]
- Pharmacological agents affecting the neurotransmitters noradrenaline, serotonin, and dopamine are associated with nightmares and other sleep disturbances.[18] [43] [44]

forced awakenings

- Parasomnias are commonly reported to occur after forced awakening from sleep.

eating disorder (sleep-related eating disorder)

- People with a known eating disorder are at higher risk than controls of developing sleep-related eating disorder.[7]

history of sleepwalking, sleeptalking, and periodic limb movements of sleep (sleep-related eating disorder)

- Associated with sleep-related eating disorder.[45]

Investigations

1st test to order

Test	Result
polysomnography (PSG) (in rapid eye movement sleep behaviour disorder [RBD]) • PSG should be requested if RBD is suspected.[53] • PSG may also be considered when the patient's bed partner reports leg kicking.[58]	**evidence of increased electromyographic tone during rapid eye movement sleep and abnormal behaviour documented on video (e.g., shouting and swearing, kicking, punching, and even jumping out of bed)**

Other tests to consider

Test	Result
PSG (in confusional arousals) • PSG is not required for the diagnosis of confusional arousals and should be ordered only if there is some uncertainty about the history or physical examination.	**arousal from slow-wave sleep on EEG; delta or theta activity may be noted**
PSG (in sleepwalking) • PSG can be helpful if the differential diagnosis includes sleepwalking precipitated by sleep-disordered breathing (obstructive sleep apnoea), other parasomnias causing sleepwalking, or rapid eye movement sleep behaviour disorder mimicking sleepwalking. • However, PSG is not required for diagnosis of sleepwalking.	**arousal from slow-wave sleep on EEG with video recording showing aimless ambulation occurring during sleep and often preceded by sitting up in bed**
PSG (in nightmare disorder) • PSG is not required unless nightmare disorder must be differentiated from other parasomnias.	**an arousal from rapid eye movement sleep noted on EEG**
PSG (in sleep terrors) • PSG can be helpful if the differential diagnosis includes sleep terrors precipitated by sleep-disordered breathing (obstructive sleep apnoea), or other parasomnias causing sleep terrors. • However, PSG is not required for diagnosis of sleep terrors.	**arousal from slow-wave sleep on EEG with video recording demonstrating patient fear, agitation, and confusion**
PSG (in all other parasomnias) • Indicated whenever the diagnosis is not obvious from the history. • Also indicated if there is a suspicion of a primary sleep disorder such as obstructive sleep apnoea or periodic limb movement disorder. • PSG may also be considered when the patient's bed partner reports leg kicking	**normal or may demonstrate evidence of other primary sleep disorder**
electroencephalogram (EEG) • Performed if nocturnal seizures are suspected in the differential diagnosis of confusional arousals, sleepwalking, sleep terrors, and rapid eye movement sleep behaviour disorder.	**normal; epileptiform spikes may be noted or clinical seizure activity during EEG monitoring may be accompanied by an electrographic seizure if nocturnal epilepsy present; in sleepwalking episodes, EEG shows increased slow-wave activity and slow oscillation density**
urine drug screen • A urine drug screen is also indicated if drug misuse is suspected.	**normal; drugs of misuse may be present**

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
seizures (sleep related)	• Sleep-related hypermotor seizures may be accompanied by incontinence, tongue biting, tonic-clonic movements, and drooling; these symptoms are not noted in the parasomnias. • Seizures may also occur when the patient is awake and asleep; parasomnias are always related to sleep.[59]	• An electroencephalogram may show abnormal electrical activity supporting a diagnosis of epilepsy.[56]
narcolepsy	• May present with similar features to recurrent isolated sleep paralysis. • In addition to daytime sleepiness, narcolepsy patients may have other symptoms including cataplexy (sudden focal or generalised loss of tone in the body associated with strong emotional experiences), hallucinations at sleep onset or on awakening.	• A multiple sleep latency test (MSLT) following an overnight polysomnogram differentiates narcolepsy from recurrent isolated sleep paralysis. • In an MSLT, patients are monitored in the sleep laboratory while having the opportunity to nap. • A short sleep latency (<8 minutes) and ≥2 sleep-onset rapid eye movement episodes with these naps indicate a diagnosis of narcolepsy.[60]
nocturnal dissociative disorder	• Often have underlying psychopathology.[61] The condition is identical to dissociative disorders occurring during the awake state, which result in the disruption of the integrated function of consciousness, memory, identity, or perception of the environment.[1] • Bizarre behaviour and experiences, such as pelvic thrusting, may be reported by eyewitnesses.[48] Examination may reveal evidence of previous self-harm, such as wrist cut marks or self-mutilation.[48]	• Structured clinical interview. • Polysomnography (PSG) is not required unless the clinical presentation is very atypical and cannot be differentiated clinically from rapid eye movement sleep behaviour disorder, sleepwalking, or sleep terrors. PSG reveals that the behaviour associated with the disorder emerges from an awake state, which was indicated on the electroencephalogram.[48] [62]

Condition	Differentiating signs / symptoms	Differentiating tests
anti-IgLON5 disease	• A rare autoimmune condition presenting with gait disturbances, dysarthria, dysphagia, sleep apnoea, daytime sleepiness, and various parasomnias.[63]	• Antibodies to IgLON5, as well as HLA DRB1*10:01 and HLA DQB1*05:01.[64]

Criteria

Diagnostic and statistical manual of mental disorders, 5th ed., text revision (DSM-5-TR) criteria for parasomnias[1]

Criteria for parasomnias are as follows:

- Non-rapid eye movement (NREM) sleep arousal disorders
  - Recurrent episodes of incomplete awakening from sleep, usually occurring during the first third of the major sleep episode, accompanied by either one of the following:
    - Sleepwalking: repeated episodes of rising from bed during sleep and walking about. While sleepwalking, the individual has a blank, staring face; is relatively unresponsive to the efforts of others to communicate with him or her; and can be awakened only with great difficulty. When diagnosing it is necessary to specify if sleepwalking is accompanied by:
      - Sleep-related eating
      - Sleep-related sexual behaviour (sexsomnia)
    - Sleep terrors: recurrent episodes of abrupt terror arousals from sleep, usually beginning with a panicky scream. There is intense fear and signs of autonomic arousal, such as mydriasis, tachycardia, rapid breathing, and sweating, during each episode. There is relative unresponsiveness to efforts of others to comfort the individual during the episodes.
  - No or little (e.g., only a single visual scene) dream imagery is recalled
  - Amnesia for the episodes is present
  - The episodes cause clinically significant distress or impairment in social, occupational, or other important areas of functioning
  - The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication)
  - Co-existing mental disorders and medical conditions do not explain the episodes of sleepwalking or sleep terrors
- Nightmare disorder
  - Repeated occurrences of extended, extremely dysphoric, and well-remembered dreams that usually involve efforts to avoid threats to survival, security, or physical integrity and that generally occur during the second half of the major sleep episode.
  - On awakening from the dysphoric dreams, the individual rapidly becomes oriented and alert.

- The sleep disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- The nightmare symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication).
- Co-existing mental disorders and medical conditions do not adequately explain the predominant complaint of dysphoric dreams.
- When classifying, specify:
    - If occurs during sleep onset
    - If with mental disorder or medical condition
    - If with another sleep disorder
    - Acute (duration of period of nightmares ≤1 month), subacute (>1 month, <6 months), persistent (≥6 months)
    - Severity (based on frequency): severity can be rated by the frequency (e.g., mild [<1 episode per week], moderate [≥1 episode per week, but less than nightly], severe [nightly])

- Rapid eye movement sleep behaviour disorder (RBD)
    - There are recurrent episodes of vocalisations or complex motor behaviours that occur during rapid eye movement (REM) sleep.
    - Upon awakening from these episodes, the individual is completely awake, alert, and not confused or disoriented.
    - These behaviours arise during REM sleep and therefore usually occur more than 90 minutes after sleep onset, are more frequent during the later portions of the sleep period, and uncommonly occur during daytime naps.
    - Either of the following:
        - REM sleep without atonia on polysomnographic recording
        - A history suggestive of REM sleep behaviour disorder and an established synucleinopathy diagnosis (e.g., Parkinson's disease, multiple system atrophy)
    - The behaviours cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (which may include injury to self or the bed partner).
    - The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.
    - Co-existing mental disorders and medical conditions do not explain the episodes.

International classification of sleep disorders, 3rd edition, text revision (ICSD-3-TR)[2]

The ICSD-3-TR defines parasomnias as sleep-related occurrences that represent undesirable physical or cognitive experiences occurring out of sleep, or during the transition from sleep to the awake state, or from the awake state to sleep. It provides a classification of parasomnias and contains diagnostic criteria and severity criteria for each type of parasomnia.

The ICSD-3 lists unique parasomnias as follows:

- Non-rapid eye movement (NREM)-related parasomnias
    - Disorders of arousal (from NREM sleep)

- Confusional arousals
- Sleepwalking
- Sleep terrors

- Sleep-related eating disorder

- Rapid eye movement (REM)-related parasomnias
  - Rapid eye movement sleep behaviour disorder (RBD)
  - Recurrent isolated sleep paralysis
  - Nightmare disorder
- Other parasomnias
  - Exploding head syndrome
  - Sleep-related hallucinations
  - Sleep-related urological dysfunction (enuresis)
  - Parasomnia due to a medical disorder
  - Parasomnia due to a medication or substance
  - Parasomnia, unspecified

Approach

Management depends on the type of parasomnia and may include reassurance, sleep environment modification, cognitive therapy, and pharmacotherapy. Active treatment is necessary if the parasomnia may result in injury to patient or bed partner and if there is significant sleep disruption.

Non-rapid eye movement (NREM) parasomnias

During an episode of confusional arousal, sleepwalking, or sleep terror, no restraint or interference should be instituted, but environment modifications may be necessary to ensure patient safety. Reversal of risk factors or precipitating factors is also important. Patients must be advised about sleep deprivation, asked to stop medications that may precipitate sleepwalking such as zolpidem, and reassured. Occasionally, scheduled awakenings just before the anticipated time of the event may eliminate the episode.[5] [65] [66]

In addition, psychotherapy (especially hypnosis and relaxation) may be used with confusional arousal, sleepwalking, or sleep terrors.[65] [67]

There is a lack of drug treatment randomised controlled trials (RCTs). Drug treatment choices should be based on the frequency and severity of events.[5] [66] Anticonvulsant therapy with topiramate has been shown to be effective in some patients with sleep-related eating disorder and is a first-line choice if treatment is required.[5] [68] [69] Topiramate treatment duration is variable (may be long term) and depends on the response, adverse effects, and physician/patient choice.

Refractory NREM parasomnias

In rare cases, confusional arousals, sleepwalking, and sleep terrors may become severe or chronic enough to interfere significantly with sleep health. In such cases benzodiazepines are tried.[65] If benzodiazepines are not effective, selective serotonin-reuptake inhibitors (SSRIs), or even tricyclic antidepressants are occasionally used. However, such drugs should only be used in exceptional cases. [65] [70] Given the potential for adverse effects, these drugs should be used only by sleep consultants after exclusion of reversible precipitating factors for confusional arousals.[71]

Dopamine agonists are indicated in cases of sleep-related eating disorder if topiramate therapy fails.[72] [73] Pramipexole or ropinirole therapy has been reported to cause sudden sleep in patients even while they are engaged in activities during the day. Patients must be warned about the potential for this adverse effect, especially with regard to driving and operating heavy machinery. Treatment should be withdrawn gradually, as abrupt withdrawal may result in acute worsening of symptoms.

Benzodiazepines and psychotherapy have not been shown to be beneficial for sleep-related eating disorder.[68]

Rapid eye movement (REM) parasomnias

The diagnostic criteria for distinguishing REM parasomnias varies and can overlap. REM parasomnias include rapid eye movement sleep behaviour disorder (RBD), recurrent sleep paralysis, and narcolepsy. RBD can be further subdivided into isolated, secondary due to a medical condition, or drug-induced.[1] [2] [37]

Rapid eye movement sleep behaviour disorder (RBD) management

Ensuring a safe sleeping environment

It is critically important to help patients maintain a safe sleeping environment to prevent potentially injurious nocturnal behaviours. Patients and bed partners or household members should be advised to alter the environment to minimise risk in case of injuries; falling or jumping out of bed. Furniture, mirrors, and staircases may need to be moved or padded. Bedside weapons or objects that could inflict injury should be removed. Patients may need to sleep separately from the bed partner or put a pillow barrier between them.[37] A bed alarm can be considered.[74]

RBD pharmacological management

Along with sleep environment modification, pharmacological therapy is often instituted in RBD. However, there is not a robust evidence base for symptomatic treatment. RCTs are lacking, and treatment relies on case series evidence.[37] [67][74] Melatonin and clonazepam are recommended as first-line pharmacological options.[23] [37] [74] [75][76] [77] [78]

Melatonin has a favorable adverse-effect profile, especially in patients with a risk of falls, comorbid sleep apnoea, underlying liver disease, and those receiving polypharmacy with other medications.[37] [67] [74] [77]

The benzodiazepine clonazepam is highly effective.[37] [67] [74] [75] Patients receiving clonazepam should be cautioned about the potential for daytime drowsiness.[37] The addition of alcohol or other central nervous system depressant drugs to benzodiazepines increases the risk of drowsiness and sedation, and patients must be cautioned about this potential for interaction. Withdrawal of benzodiazepine treatment is associated with recurrence of symptoms.

Other RBD pharmacological options

Pramipexole (a dopamine agonist) has shown benefits in patients with isolated RBD based on several observational studies.[37] [74] [79] [80] [81] It was most effective in patients with periodic limb movements, suggesting that the effect of pramipexole may be through improving ancillary motor activity. Adverse effects may include orthostatic hypotension, daytime sleepiness, nausea, confusion, and impulse control disorder.[37]

Transdermal rivastigmine (a cholinesterase inhibitor) can be used for isolated and secondary RBD based on a randomised controlled clinical trial showing reduced dream enactment behaviours in patients with mild cognitive impairment and in patients with Parkinson's disease.[37] [82] [83] Adverse effects include skin irritation, nausea, headache, and bradycardia.[37]

Drug-induced REM sleep behaviour disorder

For patients with drug-induced REM sleep behaviour disorder, typically due to SSRIs or serotonin-noradrenaline reuptake inhibitors, discontinuing the offending drug may be helpful, if possible. However, risks of stopping the drug, such as in worsening the underlying mood disorder, needs to be weighed against the potential benefits, particularly given that it may take months to see RBD symptom improvement.[37]

Recurrent isolated sleep paralysis

Office evaluation and reassurance alone may be appropriate as first-line treatment.[5] Since sleep paralysis most commonly occurs in the supine position, strategies to prevent sleeping in the supine position may help.[25] Cognitive behavioural therapy can be tried (e.g., sleep hygiene, relaxation

techniques during episodes, coping strategies for frightening hallucinations, disputation of catastrophic thoughts, imaginary rehearsal of successful resolution of episodes).[25]

Escitalopram (an SSRI) has shown benefit in a small number of case reports.[84] In persistent cases, other SSRIs and tricyclic antidepressants can be considered since they have shown benefit in sleep paralysis due to narcolepsy. However, recurrent isolated sleep paralysis drug treatment options lack good quality evidence since there are no RCTs.[25]

Nightmares

Nightmares that recur enough to interfere with sleep health and patient well-being are managed with relaxation and desensitisation.[18] Nightmare-focused cognitive behavioural therapy (exposure and imagery rehearsal therapy) resulted in better treatment outcomes than indirect methods such as relaxation and recording of nightmares.[18][66] [85]

The alpha-blocker prazosin, when used in nightmares associated with post-traumatic stress disorder, has shown beneficial efficacy in several studies in civilian and military settings. Although one large clinical trial failed to show improvement, subsequent meta-analyses incorporating it still show net benefit.[18] [86]

Other pharmacological treatment options rely on data from anecdotal case reports, case series, and small open-label trials (e.g., atypical antipsychotics, clonidine, cyproheptadine, fluvoxamine, gabapentin, nabilone, phenelzine, topiramate, trazodone, tricyclic antidepressants), and they are not routinely recommended.[18] [87]

Refractory rapid eye movement sleep behaviour disorder (RBD)

RBD may be resistant to first-line treatment options and/or secondary due to a medical condition (e.g., Parkinson's disease, dementia with Lewy bodies, narcolepsy).

Some alternative pharmacological treatments may be tried (e.g., memantine, safinamide, sodium oxybate) but there is a lack of robust clinical evidence, and they are not routinely recommended.[37] [88] [89] [90]

Other parasomnias

Sleep-related hallucinations and exploding head syndrome

During an office evaluation, reassurance alone may be all that is required for sleep-related hallucinations and exploding head syndrome.[5] Sleep-related hallucinations management involves eliminating or reducing known triggers (e.g., sleep deprivation, tobacco smoking, drugs [e.g., adrenergic antagonists, sedative hypnotics, antidepressants]).[5]

For exploding head syndrome, it is important to treat underlying comorbid sleep disorders that may be triggering the parasomnia. Tricyclic antidepressants such as amitriptyline or clomipramine may also be beneficial.[5] [91]

Enuresis

The treatment of sleep enuresis requires diagnosis (by careful history and examination) and management of potential secondary causes. These include obstructive sleep apnoea, genitourinary and renal problems, seizures, diabetes mellitus, certain medications (some antipsychotics, valproate, and SSRIs), hyperthyroidism, and psychological factors such as history of sexual abuse.[5] [17] When secondary

causes have been excluded, the treatments of enuresis fall under 3 major categories: behavioural therapy, alarm therapy (used primarily in children), and pharmacotherapy.[5]

Behavioural therapy is supported by clinical and anecdotal data, but no randomised trials are available to validate its efficacy. Nevertheless, it has very few drawbacks and should be attempted as first line. It includes a strict bowel regimen to avoid hard stools or constipation, as this may cause or worsen enuresis; increasing fluid intake during the morning and early afternoon hours, and limiting it during the evening and night time; encouraging the patient to avoid holding urine and to void at least once every 2 hours; and biofeedback to help relax the pelvic floor muscles.[5] [17]

Alarm therapy has only been studied in children and has been shown efficacious in multiple trials. It involves using a moisture-sensitive alarm that goes off and awakens the child at the moment of bedwetting.[5] [17]

Pharmacotherapy: the drugs that have randomised trial support for efficacy include anticholinergics (e.g., oxybutynin, tolterodine), desmopressin, tricyclic antidepressants (e.g., imipramine), and verapamil. Rarely, medications can be used in combination.[5] [17]

Treatment aims for other parasomnias

In parasomnia unspecified, treatment is focused on the underlying psychiatric condition. If parasomnia is related to drug and substance abuse or underlying medical problems, treatment options will be focused on these.[5]

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Ongoing (summary)

non-rapid eye movement (NREM) parasomnia		
confusional arousals or sleep terrors	**1st**	**safety modifications and correction of predisposing factors**
	adjunct	**psychotherapy**
	2nd	**benzodiazepine**
	3rd	**selective serotonin-reuptake inhibitor or tricyclic antidepressant**
sleepwalking	**1st**	**safety modifications and correction of predisposing factors**
	adjunct	**psychotherapy**
	2nd	**benzodiazepine**
	3rd	**selective serotonin-reuptake inhibitor or tricyclic antidepressant**
sleep-related eating disorder	**1st**	**topiramate**
	2nd	**dopamine agonist**
rapid eye movement (REM) parasomnia		
rapid eye movement sleep behaviour disorder (RBD)	**1st**	**sleep environment modification**
	adjunct	**melatonin or clonazepam**
	2nd	**pramipexole or rivastigmine**
drug-induced rapid eye movement sleep behaviour disorder	**1st**	**discontinue causative drug**
recurrent isolated sleep paralysis	**1st**	**reassurance and positional therapy**
	2nd	**cognitive behavioural therapy**
	3rd	**selective serotonin-reuptake inhibitor or tricyclic antidepressants**
nightmares	**1st**	**cognitive behavioural therapy**
	2nd	**prazosin**

Ongoing (summary)

other parasomnias		
exploding head syndrome, sleep-related hallucinations	1st	reassurance and treatment of underlying sleep disorder
	adjunct	tricyclic antidepressant
enuresis	1st	behavioural therapy
	2nd	alarm therapy
	3rd	pharmacotherapy

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Ongoing

non-rapid eye movement (NREM) parasomnia

confusional arousals or sleep terrors

1st **safety modifications and correction of predisposing factors**

» Confusional arousals or night terrors generally do not require pharmacological therapy.

» Simple reassurance that the condition is not harmful and is likely to resolve over time is indicated.

» Simple measures such as removing objects that may cause harm to the patient from the sleeping areas and avoiding precipitants such as sleep deprivation are warranted, especially if episodes are frequent.

» Physical restraint during a confusional arousal or night terror is not indicated.

adjunct **psychotherapy**

Treatment recommended for SOME patients in selected patient group

» If safety modifications and correction of predisposing factors fail to work, psychotherapy (cognitive behavioural therapy or hypnosis) is added.[65] [67]

2nd **benzodiazepine**

Primary options

» clonazepam: 0.5 to 2 mg orally once daily at bedtime

OR

» temazepam: 15-30 mg orally once daily at bedtime

OR

» diazepam: 5-10 mg orally once daily at bedtime

OR

MANAGEMENT

Ongoing

» oxazepam: 10-20 mg orally once daily at bedtime

» If confusional arousals or night terrors persist despite observation, benzodiazepines are used.[65] Benzodiazepines are not approved for use in confusional arousals or night terrors and are used only exceptionally for these parasomnias. Such use should be supervised by a qualified sleep consultant, preferably one with special expertise in the management of parasomnias.

» Pharmacological therapy should only be instituted in chronic and severe cases that cause significant disruption in the patient's sleep or injury to the patient or bed partner.

» Patients receiving benzodiazepines should be cautioned about the potential for daytime drowsiness. The addition of alcohol or other central nervous system depressant drugs to benzodiazepines increases the risk of drowsiness and sedation, and patients must be cautioned about this potential for interaction. Withdrawal of benzodiazepine treatment is associated with recurrence of symptoms.

» Clonazepam, temazepam, oxazepam, or diazepam may be used for confusional arousals or night terrors.

» Patients should be seen in close follow-up soon after treatment is started and assessed for efficacy, adverse effects, need to change dose or stop treatment, and change to a different treatment. Monitor regularly and reassess after 4 weeks.

» Doses should be started low and increased gradually according to response.

3rd **selective serotonin-reuptake inhibitor or tricyclic antidepressant**

Primary options

» fluoxetine: 20-60 mg orally once daily

OR

» citalopram: 20-60 mg orally once daily

OR

» paroxetine: 20-50 mg orally (immediate-release) once daily

Ongoing

OR

» sertraline: 25-200 mg orally once daily

Secondary options

» clomipramine: 25-100 mg orally once daily at bedtime

OR

» imipramine: 10-50 mg orally once daily at bedtime

» If benzodiazepines are not effective, selective serotonin-reuptake inhibitors (SSRIs) (e.g., fluoxetine, citalopram, paroxetine, sertraline), or even tricyclic antidepressants (e.g., clomipramine, imipramine), are occasionally used.

» SSRIs and tricyclic antidepressants are not approved for use in confusional arousals or night terrors and are used only exceptionally for this parasomnia. Such use should be supervised by a qualified sleep consultant, preferably one with special expertise in the management of parasomnias.[65] [70]

» Antidepressants increase the risk of suicide in children, adolescents, and young adults according to studies with such medicines in depressed patients. Patients must be warned of this risk, as well as the other adverse effects of antidepressants.

» Patients should be seen in close follow-up soon after treatment starts and assessed for efficacy, adverse effects, need to change dose or stop treatment, and change to a different treatment and monitored regularly.

» Doses should be started low and increased gradually according to response.

sleepwalking — **1st** — **safety modifications and correction of predisposing factors**

» Avoiding or correcting predisposing factors and triggers (e.g., sleep deprivation, school or home stress, environmental noise), and making safety modifications of the sleep environment to avoid injury (e.g., locks on doors and windows) are first-line therapies.

» Pharmacological therapy should not be instituted unless these measures have failed and the sleepwalking is chronic and/or severe

MANAGEMENT

Ongoing

enough to cause significant disruption in the patient's sleep.

adjunct **psychotherapy**

Treatment recommended for SOME patients in selected patient group

» If safety modifications and correction of predisposing factors fail to work, psychotherapy (cognitive behavioural therapy or hypnosis) is added.[65]

2nd **benzodiazepine**

Primary options

» clonazepam: 0.5 to 2 mg orally once daily at bedtime

Secondary options

» diazepam: 5-10 mg orally once daily at bedtime

» Benzodiazepines are not approved for use in sleepwalking and should be used only exceptionally for this parasomnia. Consequently, such use should be supervised by a qualified sleep consultant, preferably one with special expertise in the management of parasomnias.

» Benzodiazepines should only be used in severe cases of sleepwalking that do not respond to simple measures (i.e., correction of predisposing factors, safety modifications, and psychotherapy), or if the sleepwalking is placing the patient or bed partner in harm's way. Clonazepam and diazepam are the benzodiazepines of choice for this indication; however, clonazepam is usually tried first.

» Patients receiving benzodiazepines should be cautioned about the potential for daytime drowsiness. The addition of alcohol or other central nervous system depressant drugs to benzodiazepines increases the risk of drowsiness and sedation, and patients must be cautioned about this potential for interaction. Withdrawal of benzodiazepine treatment is associated with recurrence of symptoms.

» Patients are seen in close follow-up soon after treatment starts and assessed for efficacy, adverse effects, need to change dose or stop treatment, and change to a different treatment. Monitor regularly and reassess after 4 weeks.

» Doses should be started low and increased gradually according to response.

Ongoing

3rd **selective serotonin-reuptake inhibitor or tricyclic antidepressant**

Primary options

» sertraline: 25-200 mg orally once daily

Secondary options

» clomipramine: 25-100 mg orally once daily at bedtime

OR

» imipramine: 10-50 mg orally once daily at bedtime

» Antidepressants are not approved for use in sleepwalking and are only used exceptionally for this parasomnia. Consequently, such use should be supervised by a qualified sleep consultant, preferably one with special expertise in the management of parasomnias.

» Tricyclic antidepressants are only used in severe cases of sleepwalking that do not respond to simple measures (i.e., correction of predisposing factors and safety modifications) or use of benzodiazepines.

» The selective serotonin-reuptake inhibitor sertraline may be considered as an alternative to tricyclic antidepressants for the management of sleepwalking if tricyclic antidepressants are not tolerated, or as third-line treatment if other options prove to be ineffective.[70]

» Monitor regularly and reassess after 4 weeks.

» Doses should be started low and increased gradually according to response.

sleep-related eating disorder

1st **topiramate**

Primary options

» topiramate: 25-150 mg orally once daily at bedtime

» Anticonvulsant therapy with topiramate has been shown to be effective in some patients with sleep-related eating disorder.[5] [68] [69]

» Duration of treatment is variable and depends on the response to treatment, adverse effects, and physician/patient choice. The treatment may need to be long term in patients with this parasomnia.

Ongoing

» Doses should be started low and increased gradually according to response.

2nd **dopamine agonist**

Primary options

» pramipexole: 0.125 to 1.5 mg orally once daily at bedtime

OR

» ropinirole: 0.5 to 8 mg orally (immediate-release) once daily at bedtime

» Dopamine agonists are indicated if topiramate therapy fails.[73] [72]

» Pramipexole and ropinirole therapy have been reported to cause sudden sleep in patients even while they are engaged in activities during the day. Patients must be warned about the potential for this adverse effect, especially with regard to driving and operating heavy machinery.

» Patients should also be warned that hypotension, especially orthostatic hypotension, may occur. Some patients have described visual hallucinations.

» Duration of treatment with pramipexole or ropinirole is variable and depends on the response to treatment, adverse effects, and physician/patient choice. The treatment may need to be long term. Treatment should be withdrawn gradually, as abrupt withdrawal may result in acute worsening of symptoms.

» Doses should be started low and increased gradually according to response.

rapid eye movement (REM) parasomnia

rapid eye movement sleep behaviour disorder (RBD)

1st **sleep environment modification**

» Safety precautions to prevent injury to the patient and the bed partner should be instituted in conjunction with pharmacotherapy. Furniture, mirrors, and other objects that could harm the patient if he or she left the bed should be removed. The bedroom door should be closed to avoid danger from staircases or wandering around the house.[37][67] A bed alarm can be considered.[74]

adjunct **melatonin or clonazepam**

Ongoing

Treatment recommended for SOME patients in selected patient group

Primary options

» melatonin: 3-12 mg orally (immediate-release) once daily at bedtime

OR

» clonazepam: 0.5 to 2 mg orally once daily at bedtime

» Melatonin (immediate-release) and clonazepam are recommended as first-line pharmacological options.[23] [37] [74] [75] [76] [77][78]

» Melatonin has a favorable adverse-effect profile especially in patients with a risk of falls, comorbid sleep apnoea, underlying liver disease, and those receiving polypharmacy with other medications.[37] [67] [77]

» Clonazepam is highly effective.[37] [67] [74] [75] No other benzodiazepines are reported to have the level of efficacy that clonazepam has in this disorder. Patients receiving clonazepam should be cautioned about the potential for daytime drowsiness.[37] The addition of alcohol or other central nervous system depressant drugs to benzodiazepines increases the risk of drowsiness and sedation, and patients must be cautioned about this potential for interaction. Withdrawal of benzodiazepine treatment is associated with recurrence of symptoms.

» Doses should be started low and increased gradually according to response.

2nd **pramipexole or rivastigmine**

Primary options

» pramipexole: 0.125 to 1.5 mg orally once daily at bedtime

OR

» rivastigmine transdermal: 4.6 mg/24 hour to 13.3 mg/24 hour patch once daily

» If refractory to melatonin or clonazepam, other medications such as pramipexole and rivastigmine may be beneficial.[37]

» Pramipexole (a dopamine agonist) has shown benefits in patients with isolated RBD based on several observational studies.[37] [74] [79]

Ongoing

[80] [81] It was most effective in patients with periodic limb movements, suggesting that the effect of pramipexole may be through improving ancillary motor activity. Adverse effects may include orthostatic hypotension, daytime sleepiness, nausea, confusion, and impulse control disorder.[37]

» Transdermal rivastigmine (a cholinesterase inhibitor) can be used for isolated and secondary RBD based on a randomised controlled clinical trial showing reduced dream enactment behaviours in patients with mild cognitive impairment and in patients with Parkinson's disease.[37] [82] [83] Adverse effects include skin irritation, nausea, headache, and bradycardia.[37] Because transdermal rivastigmine may cause bradycardia, an electrocardiogram may be considered before starting treatment.

» Safety precautions to prevent injury to the patient and the bed partner should be instituted in conjunction with pharmacotherapy.

» Doses should be started low and increased gradually according to response.

drug-induced rapid eye movement sleep behaviour disorder

1st **discontinue causative drug**

» For patients with drug-induced REM sleep behaviour disorder, typically due to selective serotonin-reuptake inhibitors or serotonin-noradrenaline reuptake inhibitors, discontinuing the offending drug may be helpful, if possible. However, risks of stopping the drug, such as in worsening the underlying mood disorder, needs to be weighed against the potential benefits, particularly given that it may take months to see RBD symptom improvement.[37]

recurrent isolated sleep paralysis

1st **reassurance and positional therapy**

» Patients should be reassured that isolated sleep paralysis is very common and has no untoward consequences.[5] Positional therapy to promote sleep in non-supine positions can be considered because it usually occurs during arousals from supine sleep.[25]

2nd **cognitive behavioural therapy**

» Cognitive behavioural therapy can be tried (e.g., sleep hygiene, relaxation techniques during episodes, coping strategies for frightening hallucinations, disputation of catastrophic thoughts, imaginary rehearsal of successful resolution of episodes).[25]

Ongoing

3rd **selective serotonin-reuptake inhibitor or tricyclic antidepressants**

Primary options

» escitalopram: 10-20 mg orally once daily

Secondary options

» fluoxetine: 20-60 mg orally once daily

OR

» citalopram: 20-60 mg orally once daily

OR

» paroxetine: 20-50 mg orally (immediate-release) once daily

OR

» sertraline: 25-200 mg orally once daily

Tertiary options

» clomipramine: 25-100 mg orally once daily at bedtime

OR

» imipramine: 10-50 mg orally once daily at bedtime

» In recurrent isolated sleep paralysis, the selective serotonin-reuptake inhibitor (SSRI) escitalopram has shown benefit in a small number of case reports.[84]

» In persistent cases, other SSRIs (e.g., fluoxetine, citalopram, paroxetine, sertraline) and tricyclic antidepressants (e.g., clomipramine, imipramine) can be considered since they have shown benefit in sleep paralysis due to narcolepsy. However, recurrent isolated sleep paralysis drug treatment options lack good quality evidence since there are no RCTs.[25]

» Doses should be started low and increased gradually according to response.

nightmares 1st **cognitive behavioural therapy**

» Cognitive behavioural therapy includes systemic desensitisation and relaxation techniques.

MANAGEMENT

Ongoing

» Nightmare-focused cognitive behavioural therapy (imagery rehearsal therapy and exposure, relaxation, and rescripting therapy) resulted in better treatment outcomes than indirect methods such as relaxation and recording of nightmares.[18] [66] [85]

» Nightmares that recur enough to interfere with sleep health and patient well-being are managed with these techniques.[18]

2nd **prazosin**

Primary options

» prazosin: 1 mg orally once daily at bedtime initially, increase gradually according to response, maximum 15 mg/day

» The alpha-blocker prazosin, when used in nightmares associated with post-traumatic stress disorder, has shown beneficial efficacy in several studies in civilian and military settings. Although one large clinical trial failed to show improvement, subsequent meta-analyses incorporating it still show net benefit.[18] [86]

other parasomnias

exploding head syndrome, sleep-related hallucinations

1st **reassurance and treatment of underlying sleep disorder**

» A group of other parasomnias can arise from both REM and NREM sleep (or wakefulness) and typically have a characteristic pathognomonic symptom (loud bang or flash of light, vivid hallucinations).

» Patients with exploding head syndrome or sleep-related hallucinations should be reassured that this parasomnia is not associated with any serious consequences.[5]

» Sleep-related hallucinations management involves eliminating or reducing known triggers (e.g., sleep deprivation, tobacco smoking, drugs [e.g., adrenergic antagonists, sedative hypnotics, antidepressants]).[5]

» Treatment of an underlying comorbid sleep disorder, such as sleep apnoea, may also improve these parasomnias.[5] [91]

adjunct **tricyclic antidepressant**

Treatment recommended for SOME patients in selected patient group

Primary options

Ongoing

» clomipramine: 25-100 mg orally once daily at bedtime

OR

» amitriptyline: 10-150 mg orally once daily at bedtime

» Tricyclic antidepressants (e.g., amitriptyline, clomipramine) may also be beneficial in patients with exploding head syndrome.[5] [91]

» Doses should be started low and increased gradually according to response.

enuresis

1st **behavioural therapy**

» Behavioural therapy is supported by clinical and anecdotal data, but no randomised trials are available to validate its efficacy. Nevertheless, it has very few drawbacks and should be attempted as first line. It includes a strict bowel regimen to avoid hard stools or constipation, as this may cause or worsen enuresis; increasing fluid intake during the morning and early afternoon hours, and limiting it during the evening and night time; encouraging the patient to avoid holding urine and to void at least once every 2 hours; and biofeedback to help relax the pelvic floor muscles.[5] [17]

2nd **alarm therapy**

» Has only been studied in children, and has been shown efficacious in multiple trials. It involves using a moisture-sensitive alarm that goes off and awakens the child at the moment of bedwetting.[5] [17]

3rd **pharmacotherapy**

Primary options

» desmopressin: 0.2 to 0.6 mg orally 1 hour before bedtime

OR

» oxybutynin: 5 mg orally once daily at bedtime

OR

» tolterodine: 4 mg (extended-release) orally once daily at bedtime

OR

MANAGEMENT

Ongoing

» imipramine: 25-75 mg orally once daily at bedtime

OR

» verapamil: 80 mg orally once daily at bedtime

» The drugs that have randomised trial support for efficacy include anticholinergics (e.g., oxybutynin, tolterodine), desmopressin, tricyclic antidepressants (e.g., imipramine), and verapamil. Rarely, medications can be used in combination.[5] [17]

Emerging

Melatonergic agents

A case report suggests that the melatonergic agents agomelatine and extended-release melatonin can aid in relieving sleep-related eating disorder.[92] Extended-release melatonin has been studied for the treatment of rapid eye movement sleep behaviour disorder (RBD). While findings are conflicting, a 'chronobiotic' protocol of low-dose extended-release melatonin given at the same time nightly improved RBD symptoms even several months after treatment discontinuation.[93]

Primary prevention

Because sleep deprivation is a risk factor for some parasomnias, adequate sleep is a primary prevention strategy that would be expected to impact on their incidence.

Good sleep hygiene is recommended. Sleep hygiene advice typically includes the following suggestions:

- Going to bed around the same time each day
- Avoiding daytime naps
- Limiting caffeine, nicotine, and alcohol 4 to 6 hours before bedtime
- Avoiding work-related issues before bed

If 20 minutes after bedtime the patient is still awake, it is suggested that they get out of bed and find something to do until they feel sleepy again, and then return to bed.

Secondary prevention

Prevention of sleep-related injury is an important consideration. During an episode of confusional arousal, sleepwalking, or sleep terror, no restraint or interference should be instituted. Environment modifications may be necessary to ensure patient safety and prevent injury. In rapid eye movement sleep behaviour disorder, patients and bed partners or household members should be advised to alter the environment to minimise risk in case of falls or jumping out of bed.

Patient discussions

Patient education includes explaining how predisposing factors (such as sleep deprivation and stress) may precipitate parasomnias and that these should be avoided or corrected if possible.

Patients receiving benzodiazepines are cautioned about the potential for daytime drowsiness. They are advised not to drive, operate heavy machinery, or engage in activities that entail risk when they are somnolent. The addition of alcohol or other central nervous system depressant drugs to benzodiazepines increases the risk of drowsiness and sedation, and patients must be cautioned about this potential for interaction. They should also be warned about the harmful effects of prolonged use.

Patients should be advised that topiramate may be associated with somnolence, dizziness, and in some cases anorexia and a decrease in weight. Memory problems have also been described with topiramate in some patients.

Pramipexole and ropinirole therapy have been reported to cause sudden sleep in patients even while they are engaged in activities during the day. Patients must be warned about the potential for this adverse

effect, especially with regard to driving and operating heavy machinery. They should also be warned that hypotension, especially orthostatic hypotension, can occur with these therapies.

Given the high lifetime risk of neurodegenerative disease in patients with isolated RBD, disclosure of this risk should be offered in a patient-centred manner, considering the values and desires of the patient and the risks and benefits of such disclosure. In general, disclosure of risk allows the patient to plan for their future and consider participation in research. However, disclosure may result in anxiety for a diagnosis that may not occur, particularly when there is currently no proven disease modifying or preventative strategy. Yet, withholding disclosure may negatively impact the provider-patient relationship if the patient learns of the risk on their own, such as on the internet.[37] [95]

FOLLOW UP

Monitoring

Monitoring

Patients with violent parasomnias are followed up closely to make sure that the treatment administered is effective and well tolerated.

- Rapid eye movement sleep disorder patients are evaluated 1 month after initiation of treatment and then followed every 3-6 months to ensure continued efficacy of clonazepam and compliance.
- Patients taking antidepressant medicine to treat parasomnias should be closely monitored for evidence of suicidal ideation, especially younger patients on such medicines.
- Because fibrotic complications such as pleural and peritoneal fibrosis have been observed very rarely with dopamine agonist therapy (particularly the ergot dopamine agonists such as bromocriptine, pergolide, and cabergoline, rather than the non-ergot dopamine agonists such as pramipexole and ropinirole), patients should be monitored for symptoms related to such pathology.

Complications

Complications	Timeframe	Likelihood
sleep-related injury to patient or bed partner (rapid eye movement sleep behaviour disorder [RBD])	**variable**	**high**
RBD is often associated with injury to the patient and/or bed partner due to very vigorous activity associated with the parasomnia. RBD must be treated expeditiously to avoid this complication.		
sleep-related injury and violence (confusional arousals)	**variable**	**medium**
The adult form of confusional arousal is associated with violent behaviour in some cases, especially if one intervenes during the parasomnia. Patients with this form of confusional arousal need treatment and close follow-up. Bed partners and other household members need to be educated about the need to avoid intervention during the parasomnia and to maintain a safe environment for the patient.		
poor daytime performance	**variable**	**medium**
Parasomnias disrupt sleep for the patient, as well as the sleep of the bed partner and/or other household members.		

Prognosis

Non-rapid eye movement (NREM) sleep parasomnias

Patient prognosis is very good for NREM parasomnias (confusional arousals, sleepwalking, and sleep terrors), which are more common in childhood. The parasomnia decreases in severity as the child grows older. It usually resolves by adolescence. Remission rates are reported as between 50% and 65%.[1] However, the adult variant form of confusional arousals may persist without remission and is associated with violent complications, including homicide.[2]

Sleep-related eating disorder is chronic and unremitting, but controllable.

Rapid eye movement (REM) sleep parasomnias

- Rapid eye movement sleep behaviour disorder (RBD) responds very well to treatment with melatonin and/or clonazepam in most cases.
- Isolated RBD is associated with a high rate (up to 93%) of development of neurodegenerative disorders such as Parkinson's disease and other synucleinopathies.[35] Median time from diagnosis of RBD to diagnosis of neurodegenerative disease is 8 years. Neurodegenerative pathology can be seen in post-mortem cases even if a clinical neurodegenerative diagnosis was not made.[94]
- The prognosis for nightmare disorder is variable, with underlying psychopathology being a poor prognostic factor.
- There has been little research into the prognosis of people with recurrent-isolated sleep paralysis.

Other parasomnias

- Exploding head syndrome usually resolves spontaneously.
- Hallucinations related to sleep are very variable but tend to get better with time.
- Enuresis is usually self-limiting in most cases with children and in adults who follow the recommended treatment.

Diagnostic guidelines

International

Video-polysomnography procedures for diagnosis of rapid eye movement sleep behavior disorder (RBD) and the identification of its prodromal stages (https://academic.oup.com/sleep/article/45/3/zsab257/6409886)

Published by: International RBD Study Group **Last published:** 2021

North America

International classification of sleep disorders, 3rd edition, text revision (ICSD-3-TR) (https://aasm.org/clinical-resources/international-classification-sleep-disorders)

Published by: American Academy of Sleep Medicine **Last published:** 2023

The AASM manual for the scoring of sleep and associated events (https://aasm.org/clinical-resources/scoring-manual)

Published by: American Academy of Sleep Medicine **Last published:** 2023

Recommended protocols for the multiple sleep latency test and maintenance of wakefulness test (https://aasm.org/clinical-resources/practice-standards/practice-guidelines)

Published by: American Academy of Sleep Medicine **Last published:** 2021

Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea (https://aasm.org/clinical-resources/practice-standards/practice-guidelines)

Published by: American Academy of Sleep Medicine **Last published:** 2017

Treatment guidelines

United Kingdom

BAP consensus statement on evidence-based treatment of insomnia, parasomnias and circadian rhythm disorders: an update (http://www.bap.org.uk/docsbycategory.php?docCatID=2)

Published by: British Association for Psychopharmacology **Last published:** 2019

Consensus statement on evidence-based treatment of insomnia, parasomnias and circadian rhythm disorders: an update (https://www.bap.org.uk/docdetails.php?docID=31)

Published by: British Association for Psychopharmacology **Last published:** 2019

North America

Clinical practice guideline for the management of rapid eye movement sleep behavior disorder (https://aasm.org/clinical-resources/practice-standards/practice-guidelines)

Published by: American Academy of Sleep Medicine **Last published:** 2023

Position paper for the treatment of nightmare disorder in adults (https://aasm.org/clinical-resources/practice-standards/practice-guidelines)

Published by: American Academy of Sleep Medicine **Last published:** 2018

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This approach is in line with the guidance of the International Bureau of Weights and Measures Service.

Figure 1 – BMJ Best Practice Numeral Style

5-digit numerals: 10,000

4-digit numerals: 1000

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// Acknowledgements:

Dr Roneil Malkani would like to gratefully acknowledge Dr Hrayr Attarian and Dr Pierre Giglio, previous contributors to this topic.
DISCLOSURES: PG declares that he has no competing interests. HA has carried out consultancy work for Harmony Bioscience, and received royalties from Springer. HA is also an author of a number of references cited in this topic.

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