Sleep-related movement disorders and disturbances of motor control

Panagiotis Bargiotas and Claudio L. Bassetti

Purpose of review

Review of the literature pertaining to clinical presentation, classification, epidemiology, pathophysiology, diagnosis, and treatment of sleep-related movement disorders and disturbances of motor control.

Recent findings

Sleep-related movement disorders and disturbances of motor control are typically characterized by positive motor symptoms and are often associated with sleep disturbances and consequent daytime symptoms (e.g. fatigue, sleepiness). They often represent the first or main manifestation of underlying disorders of the central nervous system, which require specific work-up and treatment. Diverse and often combined cause factors have been identified. Although recent data provide some evidence regarding abnormal activation and/or disinhibition of motor circuits during sleep, for the majority of these disorders the pathogenetic mechanisms remain speculative. The differential diagnosis is sometimes difficult and misdiagnoses are not infrequent. The diagnosis is based on clinical and video-polysomnographic findings. Treatment of sleep-related motor disturbances with few exceptions (e.g. restless legs/limbs syndrome) are based mainly on anecdotal reports or small series.

Summary

More state-of-the-art studies on the cause, pathophysiology, and treatment of sleep-related movement disorders and disturbances of motor control are needed.

Keywords

nocturnal (sleep-related) movement disorders and disturbances of motor control, nocturnal epilepsy, parasomnias, sleep

INTRODUCTION

Rapid eye movement (REM) and non-REM (NREM) sleep are associated with profound neurophysiological and neurochemical changes in the brain, which lead to changes in the control of motor functions [1]. This explains why movement disorders and disturbances of motor control sometimes appear only or preferentially during sleep. This review will discuss literature pertaining to clinical presentation, classification, epidemiology, pathophysiology, diagnosis, and treatment of sleep-related movement disorders/disturbances of motor control (SRMD/DMC).

DEFINITION/CLASSIFICATION

SRMD/DMC are typically characterized, with the exception of sleep paralysis, by involuntary, excessive motor activities (hyperkinesias), meaning positive motor symptoms. On rare occasions (e.g. in the context of REM sleep behavior disorder, RBD) voluntary movements may also be affected during

sleep differently than during the day [2]. Interestingly, changes in muscle tone are not a relevant feature of SRMD/DMC (as it is, conversely, during daytime), with the exception of RBD.

These disorders/disturbances are commonly age-dependent [1]. Epidemiological data of the most relevant disorder/disturbances are summarized in Table 1 [3–21].

No classification system is universally accepted. Table 2 presents a possible classification of motor phenomena during sleep in terms of causes, clinical features, and sleep stages in which they occur.

Department of Neurology, Sleep-Wake-Epilepsy Center (SWEZ), University Hospital (Inselspital) and University of Bern, Bern, Switzerland

Correspondence to Dr Panagiotis Bargiotas, Department of Neurology, Inselspital, Bern University Hospital, Freiburgstrasse 18, 3010 Bern, Switzerland. Tel: +41 0 31 632 30 66; fax: +41 0 31 632 96 79; e-mail: panagiotis.bargiotas@insel.ch

Curr Opin Neurol 2017, 30:405–415

DOI:10.1097/WCO.0000000000000466

KEY POINTS

- Sleep-related movement disorders/disturbances of motor control are frequent and may lead to significant sleep disturbances and consequent daytime symptoms.
- Sleep-related movement disorders/disturbances of motor control often represent the first or main manifestation of an underlying disorder of the central nervous system.
- For the majority of sleep-related movement disorders/disturbances of motor control, pathophysiological mechanism remain speculative.
- International diagnostic or therapeutic criteria have been developed only for a few disorders (e.g. RLS).

CLINICAL MANIFESTATION

The key-features of the main SRMD/DMC are presented in the following:

Hypnic jerks (sleep starts, predormital myoclonus)

Hypnic jerks are sudden, brief contractions of the body or limbs occurring spontaneously or after a stimulus, typically at sleep onset [22]. The contractions can be accompanied by a cry and hallucinations (flashes, feeling of falling) and dreams [23]. Hypnic jerks are described as a benign movement phenomenon [24]; however, they are frequent in neurological disorders such as parkinsonian syndromes resulting in a further cause of sleep disruption [25].

Table 1. Epidemiology of sleep-related movement disorders

Sleep-related movement disorders/disturbances of motor control	Frequency in the general population	Specific features
Sleep-related bruxism (SB) [3,4]	10–20%	Highest frequency in children (14–20%) The prevalence is not associated with sex and decreases with age Parental history of SB predicted SB severity
PLMS [5]	5–20%	Rare below the age of 30–40 years Common in sleep disordered breathing and narcolepsy Very common (70–90%) in RLS
Sleep paralysis [6]	5–15%	More common in narcolepsy (50%) and in psychiatric patients (30%)
Sleepwalking [7–11]	5–15%	Typically affects children (peak age 10–12 years) but it can persist or appear <i>de novo</i> in adulthood Lower frequency (1–5%) in adults The prevalence of childhood sleepwalking increases with the degree of parental history of sleepwalking. Among adult sleepwalkers 13% report an onset in adulthood
RLS [12,13]	1–10%	Rare in children More common in uremia, iron deficiency, pregnancy (10–30%) Variable prevalence in the general population among countries
Sleep terrors [7]	1–5%	Typical age-of-onset during the early childhood (peak of prevalence at 1.5 years). Lower frequency in adults Parental history of sleepwalking predicts the incidence of sleep terrors in children and the persistent nature of sleep terrors.
RBD [14,15]	1%	The prevalence of RBD is about 0.5–1% in the general population and 2–8% in the older adult population More common (25–50%) in synucleinopathies (e.g. M. Parkinson)
Sleep-related (nocturnal) eating disorders [16]	1%	The age of onset is typically in adulthood, women are more frequently affected than men. More common in sleepwalkers and in patients with RLS/PLMS
Rolandic epilepsy [17,18]	–	Typically starts around the age of 10 years It is thought to be among the more frequently occurring childhood epilepsy (14% of childhood epilepsies) However, definite epidemiological data are lacking Prevalence between 10–25% in children under 15 years
Panayiotopoulos syndrome [19]	–	Starts around the age of 5 Around 13% of children aged 3–6 years who have had one or more afebrile seizures
SHE [20]	–	Definite epidemiological data are lacking (around 2%)
Sleep related rhythmic movement disorders [21]	–	Typical onset in early infancy, rarely persisting in adulthood in patients with a positive family history, mental retardation or associated sleep disorders Familial forms have been described
Sleep related dissociative disorders	–	typically appearing in patients with known, underlying psychiatric disorders

PLMS, periodic limb movements in sleep; RBD, REM sleep behavior disorder; RLS, restless legs/limbs syndrome; SHE, sleep-related hypermotor epilepsy.

Diagnosis usually does not require any ancillary tests [26].

Sleep-related bruxism

Sleep-related bruxism consists of rhythmic tooth grinding or tapping. Typically occurs in light NREM and REM sleep. Diurnal jaw muscle discomfort is often reported, and masseter hypertrophy and alterations to the teeth and surrounding structures can be observed [27]. In a recent study, sleep-related bruxism was associated with pain and reduced sleep quality [3].

Primary and secondary forms (associated with sleep-disordered breathing, dystonia, or psychiatric disorders, e.g. anxiety, depression, and attention-deficit/hyperactivity disorder, ADHD [28]) are known.

Diagnosis is suspected clinically and can be confirmed by typical electromyography (EMG) artefacts during video-polysomnography [26].

Nocturnal groaning (catathrenia)

It consists of repetitive expiratory respiratory (groaning, moaning) monotonous sounds occurring in clusters (mainly but not exclusively in REM sleep) and lasting for up to 30–50 s [29]. A recent study reported that the occurrence of catathrenia is associated with anatomical abnormalities of the mouth [30]. An association with (other) parasomnias is known and differential diagnosis include snoring, stridor, sleep talking, and central sleep apnea [31,32]. The diagnosis is made clinically.

Facio-mandibular myoclonus

This refers to nocturnal myoclonic jerks involving the jaw and facial musculature which can lead to diurnal jaw discomfort and teeth alterations [33]. The diagnosis is based on specific EMG activity of oro-facial, masticatory and cervical muscles during polysomnography.

Excessive fragmentary myoclonus

Excessive fragmentary myoclonus (EFM) refers to the presence of excessive, nonrhythmic, and nonperiodic limb twitches with no or only minimal movement effect [34]. Although EFM is described as a benign movement phenomenon [24], recent studies suggest an association with Parkinson's disease [35] and with peripheral nerve pathologies [36[¶]]. The diagnosis is based on the presence of characteristic EMG pattern of at least 150 ms, in at least 20 min of NREM sleep, and a rate of five EMG EFM potentials per minute, during polysomnography.

Restless legs/limbs syndrome

The restless legs/limbs syndrome (RLS) is a common, today still underdiagnosed, sensorimotor disorder. The disease can appear in childhood, its course is usually chronic with rare periods of remission. The severity of symptoms varies largely but usually enhances with age. The following international RLS diagnostic criteria have been suggested [37]:

- (1) An urge to move, usually accompanied by unpleasant (or painful) sensations in the legs.
- (2) The symptoms begin or worsen during periods of rest or inactivity (sitting/lying).
- (3) The symptoms are partially or totally relieved by movement (walking/stretching).
- (4) The symptoms are worse or occur only in the evening or at night.

Diagnosis is usually made clinically. The sensory discomfort may involve the arms (30–50% of patients) or other body parts, as well [38]. Supportive criteria include the detection of periodic limb movements in sleep (PLMS) during polysomnography or leg actigraphy [37] (in 70–90% of RLS patients [39]), response to dopaminergic treatment and positive family history. In addition, supportive criteria may be helpful to differentiate RLS from RLS mimics [40].

The disorder is chronic and associated with multiple comorbidities. Patients often present with insomnia, psychiatric symptoms, and occasionally with daytime excessive sleepiness and fatigue [41]. Primary and secondary forms (associated with iron deficiency, pregnancy, uremia, spinal cord diseases, parkinsonian disorders, spinocerebellar atrophies) are known [13,42–45,46[¶]].

Periodic limb movements in sleep

PLMS are repetitive, periodic/pseudoperiodic, stereotyped movements, usually consisting of a toe extension with a dorsiflexion of the ankle and a flexion of the knee and hip. The contractions last 0.5–10 s, the episodes consist of at least four consecutive contractions/movements, the intervals between the episodes ranges from 5 to 90 s (on average 20–40 s).

PLMS may result to increased arousability during the night or daytime symptoms such as fatigue, suggesting the presence of a periodic limb movements disorder (PLMD) if no other sleep disorder is diagnosed. Recently, severe PLMS have been reported to be associated with abnormal sleep behaviors, mimicking dream enacting behavior [47[¶]] and with greater cognitive decline among nondemented individuals [48[¶]]. The detection of PLMS

is made by leg actigraphy and video-polysomnography.

Propriospinal myoclonus at sleep onset

Propriospinal myoclonus refers to repetitive axial jerks slowly spreading rostral and downward during the sleep–wake transition period and may lead to insomnia and falls out of the bed [49]. Differential diagnosis includes Hypnic jerks, RLS, PLMS, or epileptic myoclonus [50].

Sleep paralysis

Sleep paralysis (recently reviewed by Sharpless [51]) refers to short-lasting (typically <10 min [52]) episodes of inability and struggle to move and speak while in the process of falling asleep or awakening [53]. Its frequency can vary from a few life events to daily episodes. Stress, excessive sleepiness, irregular sleep–wake cycle and sleep deprivation, jet lag, and uncomfortable sleeping may increase its frequency [54–56]. Anxiety, hallucinations [sensed presence; sensation of a pressure on the chest with shortness of breath (incubus); and sensation of floating/flying (out-of-body experience)] and lucid dreaming [54] are often experienced. Primary (sporadic and familial) and secondary forms (associated with narcolepsy, other sleep or psychiatric disorders [57,58]) are known. Diagnosis is usually made clinically.

Sleep-related rhythmic movement disorders

These are stereotyped, repetitive movements such as head rolling/banging, body rolling/rocking, occurring commonly at sleep onset with a frequency of 0.5–2 Hz and duration of seconds up to several minutes. The diagnosis is suspected clinically and made by video-polysomnography.

Sleep terror (pavor nocturnus)

It consists of spells characterized by sudden onset of fear, loud screaming, and autonomic symptoms arising from deep NREM sleep and usually lasting only few minutes. Patients are difficult to be awakened and have no recall of the episodes [26]. Frequently co-occurs with other parasomnias (e.g. sleepwalking) and in combination may lead to a consequent significant daytime sleepiness [59]. The diagnosis is suspected clinically and made by video-polysomnography.

Sleepwalking (somnambulism)

Sleepwalking refers to an ambulatory activity arising (in most cases) from deep NREM sleep [26,60,61].

Patients wake up suddenly, look around with a confused stare, leave the bed, and deambulate. Movements can be repetitive and purposeless, on other occasions they appear complex and meaningful [62]. Sleepwalkers are difficult to be awakened and there is usually no recall of the episodes. Self-injuries [63,64] and rarely even homicidal, filicidal, and suicidal episodes are possible [65]. Sleepwalkers have a higher frequency of other parasomnias (sleep terrors, confusional arousals, enuresis, sleepwalking, bruxism), nocturnal eating (somnophagia), and abnormal sexual behavior during sleep [66]. More recently specific reward-related personality traits [67] have been associated with sleepwalking.

Typically, sleepwalking occurs once per night (first part) and lasts several minutes. The frequency can range from few episodes in a lifetime up to several (five to six) episodes per night [9]. Sleepwalking usually appears in the context of a sporadic or familial NREM parasomnia, less frequently also in the context of a more complex NREM/REM overlap parasomnia (e.g. in neurodegenerative diseases), of a nocturnal epilepsy (epileptic wandering), or of dissociative disorders [68–70].

Diagnosis is usually made clinically. Video-polysomnography may be needed for the correct interpretation of frequently reported nocturnal deambulatory activities.

Rapid eye movement sleep behavior disorder

REM sleep behavior disorder (RBD) is a disorder characterized by an abnormal behavior arising from REM sleep which is accompanied by (oft frightening) vivid dreaming [26,71]. The patients ‘act out their dreams’ exhibiting a variety of motor activities ranging from talking and twitching of the limbs extremities to screaming, kicking, punching, grabbing, and even complex violent and nonviolent behaviors [2]. Injuries are typical and may involve the patients or their bed partners. Autonomic activation is limited/absent. Typically, RBD occurs repeatedly in the second part of the night.

RBD appears more often in elderly males and commonly in the context of synucleinopathies [72,73] but also in narcolepsy [74]. Recently, motor complex behaviors, mimicking dream-enacting, have been reported in children with type 1 narcolepsy as well [75], further highlighting the relation between the two disorders.

The onset of RBD may precede the appearance of daytime manifestations (e.g. cognitive, motor symptoms) by up to decades [76]. Parkinsonian patients may exhibit during REM sleep/RBD in comparison to wakefulness a paradoxical improvement of speech, facial, and extremity motor control. RBD

in parkinsonian patients has been associated with psychiatric manifestations, hallucinations, cognitive decline, and sleepwalking [2,77].

Rarely, RBD occurs also in the context of a NREM/REM overlap parasomnia and its extreme form of a wake–sleep state breakdown (status dissociatus) [78–80].

International diagnostic criteria have been developed [81]. Diagnosis is confirmed by video-polysomnography documenting a loss of motor atonia, an increase of phasic muscle activity during REM sleep, and corresponding behaviors.

Sleep-related epilepsy with simple motor manifestations

The most common syndrome is that of rolandic epilepsy (also called benign epilepsy with centro-temporal spikes, BECTS), which is characterized by infrequent nocturnal (often at sleep onset or before awakening) simple, hemifacial motor seizures lasting 1–3 min. Postictal amnesia is not always present.

Panayiotopoulos syndrome refers to an idiopathic epileptic syndrome with (typically nocturnal) short episodes of disturbed autonomic functions (e.g. nausea, retching, vomiting) and occasionally with syncope-like epileptic seizures [82].

Others: typically temporal lobe epilepsy and occasionally sleep-related hypermotor epilepsy (SHE) with paroxysmal arousals, may manifest with nocturnal simple motor manifestations [33,83].

Sleep-related epilepsy with complex motor manifestations

SHE and hypermotor seizures of nonfrontal origin (e.g. insula, temporal, or occipital cortex) are characterized by a wide spectrum of stereotyped manifestations, mostly occurring during NREM sleep. The short-lasting (10–30 s) episodes present a sudden onset of motor behaviors including repetitive patterns (hand clenching, arm raising/lowering, knee bending), more complex patterns (hypermotor/hyperkinetic seizure) such as body rocking, kicking, pedaling, hitting, boxing, screaming, and episodic nocturnal/epileptic wandering, and occasionally asymmetric tonic posturing of the extremities (nocturnal paroxysmal dystonic seizures) [33,84–88]. The amnesia can be incomplete.

For the diagnosis of SHE, a video-polysomnography is needed. The features differentiating SHE from motor parasomnias and dissociative sleep related disorders are: several attacks per night at any time during the night, brief duration of the attacks, and stereotyped motor pattern [85,86].

Nocturnal sleep-related eating disorders

Nocturnal sleep-related eating disorders (NSRED) present with an evening hyperphagia and/or nocturnal awakening and ingestion of food. Awareness of the night eating is inconstant/absent. When awareness is present the term of night eating syndrome (NES) is preferred.

NSRED comorbidities include depression, insomnia, RLS, and sleepwalking [16,89]. Recent studies reported associations of nocturnal eating with anxiety and obsessive–compulsive symptoms [90,91].

Sleep-related dissociative disorders/psychogenic nonepileptic seizures

These disorders manifest typically after awakening from sleep with nocturnal episode of complex motor behaviors (e.g. vocalization, screaming, crying, deambulation/sleepwalking, violence, self-mutilation) and are common in patients with known, underlying psychiatric disorders [70,92]. Episodes may last for several minutes up to hours. Amnesia is typically present for the episodes. A coexistence of ‘true’ epileptic seizures and parasomnias in a single patient is possible and can complicate the diagnosis. In these cases video-polysomnography/telemetry is necessary.

ETIOLOGY

Several etiologic factors, not uncommonly in combination, of SRMD/DMC have been identified, suggesting for some of them an underlying continuous spectrum of disorder (for RLS reviewed by Trenkwalder *et al.* [93]).

Genetic factors are known to be relevant for RLS [reviewed recently by Winkelmann *et al.* [94]], sleepwalking (positive family history in 25–50%), bruxism, SHE (25% of cases), rolandic epilepsy [84,95,96], and for RBD [97,98] including the susceptibility of individuals with RBD to develop synucleinopathies [99,100] (although negative for APOE e4 allele [101]). More recent studies reported an association of PLMS [102] and of sleep paralysis [103] with specific single-nucleotide polymorphisms.

Neurodegeneration probable RBD is associated with neurodegenerative disease markers such as hyposmia and nonmotor symptoms [15] and over 50% of patients with idiopathic RBD develop within 5–10 years a neurodegenerative parkinsonian disorder (typically a synucleinopathy) [72,73,104,105,106,107].

Focal brain damage is mainly related to nocturnal epilepsy (e.g. in the context of cortical dysplasia [108]), RBD (limbic encephalitis, brainstem stroke,

demyelinating disorders [109,110]), and RLS (e.g. spinal cord demyelination, subcortical stroke).

Psychiatric factors/disorders is an underlying stress or psychiatric disorder is often found in (particularly adult) patients with sleepwalking, bruxism, sleep-related dissociative disorders, nocturnal eating disorder, and sleep paralysis [111–113]. A number of studies highlighted the association of RLS with psychiatric disturbances (recently reviewed by Mackie *et al.* [114]) mainly with depression and anxiety and with ADHD [115]. Interestingly, patients with RLS with augmentation are at high risk of developing impulsive behavior, suggesting that impulse control disorder and augmentation may share a common pathophysiology [116²²].

Drug effects: Benzodiazepine may trigger/exacerbate sleepwalking, occasionally also NSRED; antidepressants may exacerbate RBD, RLS,

sleepwalking, and bruxism; neuroleptics can exacerbate RLS and bruxism; dopaminergic drugs may trigger/aggravate NSRED and bruxism [117,118].

Others: Alcohol has been shown to exacerbate RBD, PLMS, bruxism, and sleepwalking [119]. A deficient iron transport/metabolism and content, recently highlighted also in quantitative magnetic susceptibility [46²³], may underlie RLS.

PATHOPHYSIOLOGY

Motor control requires afferent, integrating, and efferent systems involving the spinal cord, the brainstem, the cerebellum, the basal ganglia, and the cerebral cortex.

Nocturnal/sleep-related motor manifestations arise from a disruption of such mechanisms, often in form of the abnormal activation and/or disinhibition of motor circuits. However, the exact

FIGURE 1. The flowchart shows important steps and tools for the diagnostic work-up of sleep-related movement disorders and motor control disturbances. Detailed history: whenever possible including an interview with bed partners. International diagnostic criteria have been developed only for a few disorders including RLS and RBD [37,81]. Questionnaires: Few scales have been suggested to estimate the severity of some of these disorders (e.g. RLS and RBD), others to differentiate between epileptic and nonepileptic disorders [77,130,131]. Home video: it may be helpful as a first step before performing more sleep or telemetric investigations, especially if suspicion for nocturnal epilepsy or parasomnias. Video-polysomnography including multiple EEG and EMG derivations: In some situations (e.g. NREM parasomnias, nocturnal epilepsy) sleep deprivation may be used to trigger motor events. Neuroimaging: it should be considered especially in patients with suspicious nocturnal epilepsy, REM sleep behavior disorder (RBD) and adult-onset (frequent) NREM parasomnias. Based on specific diagnostic hypotheses specific diagnostic including EEG (e.g. epilepsy, adult-onset parasomnias), daytime neurophysiological studies (e.g. sleepwalking, RBD) genetic tests (e.g. epilepsy), detailed cognitive (e.g. RBD) and psychiatric (e.g. sleep related dissociative disorders, nocturnal eating disorder, sleep paralysis, RLS) assessments, should be considered.

contribution of afferent or efferent systems (and corresponding neurotransmitters) in these disorders remains speculative [60,120]. In sleepwalking, autosomal dominant SHE, and nocturnal (sleep-related) panic attacks a primary dysfunction of ascending, activating systems may prevail [60,121]. In addition, in RBD a primary dysfunction of descending, inhibitory systems is currently favored [120]. In most situations, a dysfunction in both systems is probably involved [77].

In sleep paralysis, emerging hypotheses regarding the pathophysiology of the commonly reported ‘bedroom intruder’ hallucinations implicate the mirror neurons system and interaction between several brain region, mainly the prefrontal cortex and the sensory feedback [122].

In RLS, increasing data support the notion of a complex network disorder, involving primarily the brain iron content reduction and then the dysfunction of dopaminergic pathways in nigrostriatal and mesolimbic networks [123].

Complex nocturnal/sleep-related motor manifestations/behaviors are thought to arise from the abnormal (nonepileptic/epileptic) activation of innate (genetically determined) or learned motor patterns (central pattern generators) that are essential for survival [124,125]. The term of ‘state dissociation’ has been used to describe the coexistence

of abnormally activated brain areas with others that exhibits features of normal sleep [126]. Such a dissociation has been documented in few patients with parasomnias and sleep-related epilepsy by means of neuroimaging and neurophysiological methods [121,127,128].

DIAGNOSTIC WORK UP

The etiological differential diagnosis between SRMD/DMC is sometimes difficult [84] and coexistence in a single patient (e.g. nocturnal seizures and parasomnias) is frequent [33,129]. In addition, the clinical phenomenology can be observed in the context of sleep disorders of different etiology.

International diagnostic criteria have been developed only for RLS and RBD [37,81]. Few scales have been suggested to estimate the severity of some of these disorders (e.g. RLS and RBD), or to differentiate between disorders [77,130,131].

A diagnostic work-up of SRMD/DMC is presented in Fig. 1.

TREATMENT

For most SRMD/DMC, only anecdotal reports or small series have been published on treatment. International treatment guidelines have been

Table 3. General principles of treatment for sleep related movement disorders

Nonpharmacological interventions	Pharmacological interventions	Other Interventions
Psychotherapy NREM parasomnias Bruxism Sleep paralysis Sleep-related dissociative disorders/psychogenic non epileptic seizures Stress reducing techniques NREM parasomnias Bruxism Sleep paralysis Sleep-related dissociative disorders/psychogenic non epileptic seizures Cognitive-behavioral treatment NREM parasomnias Bruxism Sleep paralysis Sleep-related dissociative disorders/psychogenic nonepileptic seizures Safe sleep environment Sleepwalking RBD Nocturnal epilepsy Oral appliances, splints Bruxism	Benzodiazepines (commonly clonazepam) NREM parasomnias RBD Sleep-related rhythmic movement disorders Excessive non-REM and REM myoclonus Intermittent RLS Nocturnal eating disorders Dopaminergic agents RLS PLMS RBD Nocturnal eating disorders Melatonin RBD Antiepileptic drugs (e.g. carbamazepine, gabapentin or topiramate) Nocturnal eating disorder RLS Nocturnal epilepsy Antidepressants Sleep paralysis Nocturnal panic attacks Sleep-related rhythmic movement disorders Opioids RLS	Avoidance of triggering/exacerbating situations Sleep deprivation NREM parasomnias Nocturnal epilepsy Sleep paralysis Fever NREM parasomnias Nocturnal epilepsy Sleep paralysis Drugs (see section etiology) Treatment of underlying disorder Surgical intervention Severe nocturnal frontal lobe epilepsy Parasomnias in the context of Parkinson’s disease (e.g. deep brain stimulation) Injections of botulinum toxin Sleep bruxism Positive Airway pressure therapy Sleep-disordered breathing

NREM, non-REM; PLMS, periodic limb movements in sleep; RBD, REM sleep behavior disorder; REM, rapid eye movement; RLS, restless legs/limbs syndrome.

published only for RLS [132]. General principles of treatment are presented in Table 3.

CONCLUSION

SRMD/DMC are frequent and may lead to significant sleep disturbances (e.g. sleep fragmentation/insomnia, sleep-associated injuries/violence) and consequent daytime symptoms (e.g. fatigue, sleepiness). In addition, they can represent the first or main manifestation of an underlying disorder of the central nervous system (e.g. epilepsy, neurodegenerative disease) which requires specific work-up and treatment. In most SRMD/DMC, the pathogenetic mechanisms remain speculative. Several etiologic factors have been identified, not uncommonly in combination however the differential diagnosis is sometimes difficult. International diagnostic or therapeutic criteria have been developed only for a few disorders (e.g. RLS). More state-of-the-art studies on the treatment of these disorders are needed.

Acknowledgements

None.

Financial support and sponsorship

P.B. received a grant from the University Hospital Bern, Inselspital (Insel-Grant), Bern, Switzerland.

C.B. has received honoraria for consultancy, lectures, and board memberships from the following institutions/companies: Jazz, Servier, UCB, Zambon. His research is currently supported by grants of the following institutions/companies: Swiss National Science Foundation (SNF), ResMed, Respirationics, Vifor Pharma, UCB Pharma, Schweizerische Herzstiftung, Tropos Stiftung, Parkinson Schweiz.

Conflicts of interest

The authors have no conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

1. Chokroverty S, Allen RP, Walters AS, *et al.* Sleep and movement disorders. Oxford University Press; 2013.
2. De Cock Cochen VC, Vidaihet M, Leu S, *et al.* Restoration of normal motor control in Parkinson's disease during REM sleep. *Brain* 2007; 130:450–456.
3. Khoury S, Carra MC, Huynh N, *et al.* Sleep bruxism-tooth grinding prevalence, characteristics and familial aggregation: a large cross-sectional survey and polysomnographic validation. *Sleep* 2016; 39:2049–2056.
4. Bader G, Lavigne G. Sleep bruxism; an overview of an oromandibular sleep movement disorder. REVIEW ARTICLE. *Sleep Med Rev* 2000; 4:27–43.
5. Ohayon MM, Roth T. Prevalence of restless legs syndrome and periodic limb movement disorder in the general population. *J Psychosom Res* 2002; 53:547–554.
6. Sharpless BA, Barber JP. Lifetime prevalence rates of sleep paralysis: a systematic review. *Sleep Med Rev* 2011; 15:311–315.
7. Petit D, Pennestri MH, Paquet J, *et al.* Childhood sleepwalking and sleep terrors: a longitudinal study of prevalence and familial aggregation. *JAMA Pediatr* 2015; 169:653–658.
8. Hublin C, Kaprio J, Partinen M, *et al.* Prevalence and genetics of sleepwalking: a population-based twin study. *Neurology* 1997; 48:177–181.
9. Kavey NB, Whyte J, Resor SR, *et al.* Somnambulism in adults. *Neurology* 1990; 40:749–752.
10. Hublin C, Kaprio J. Genetic aspects and genetic epidemiology of parasomnias. *Sleep Med Rev* 2003; 7:413–421.
11. Ohayon MM, Mahowald MW, Dauvilliers Y, *et al.* Prevalence and comorbidity of nocturnal wandering in the U.S. adult general population. *Neurology* 2012; 78:1583–1589.
12. Rothdach AJ, Trenkwalder C, Haberkost J, *et al.* Prevalence and risk factors of RLS in an elderly population: the MEMO study. Memory and Morbidity in Augsburg Elderly. *Neurology* 2000; 54:1064–1068.
13. Hübner A, Krafft A, Gadjest S, *et al.* Characteristics and determinants of restless legs syndrome in pregnancy: a prospective study. *Neurology* 2013; 80:738–742.
14. Kang SH, Yoon IY, Lee SD, *et al.* REM sleep behavior disorder in the Korean elderly population: prevalence and clinical characteristics. *Sleep* 2013; 36:1147–1152.
15. Mahlknecht P, Seppi K, Frauscher B, *et al.* Probable RBD and association with neurodegenerative disease markers: a population-based study. *Mov Disord* 2015; 30:1417–1421.
16. Auger RR. Sleep-related eating disorders. *Psychiatry* 2006; 3:64–70.
17. Cavazzuti GB. Epidemiology of different types of epilepsy in school age children of Modena, Italy. *Epilepsia* 1980; 21:57–62.
18. Sidenvall R, Forsgren L, Blomquist HK, *et al.* A community-based prospective incidence study of epileptic seizures in children. *Acta Paediatr* 1993; 82:60–65.
19. Panayiotopoulos CP. Vomiting as an ictal manifestation of epileptic seizures and syndromes. *J Neurol Neurosurg Psychiatry* 1988; 51:1448–1451.
20. Vignatelli L, Bisulli F, Giovannini G, *et al.* Prevalence of nocturnal frontal lobe epilepsy in the adult population of Bologna and Modena, Emilia-Romagna region, Italy. *Sleep* 2015; 38:479–485.
21. Mayer G, Tracik F, Wilde F. Rhythmic movement disorder revisited. *J Sleep Res* 2000; 9 (Suppl 1):127.
22. Oswald I. Sudden bodily jerks on falling asleep. *Brain* 1959; 82:92–103.
23. Frenette E, Guilleminault C. Nonepileptic paroxysmal sleep disorders. *Handb Clin Neurol* 2013; 112:857–860.
24. American Academy of Sleep Medicine. International classification of sleep disorders. 3rd ed. Darien, IL: American Academy of Sleep Medicine; 2014.
25. Chiaro G, Calandra-Buonaura G, Sambati L, *et al.* Hypnic jerks are an underestimated sleep motor phenomenon in patients with parkinsonism. A video-polysomnographic and neurophysiological study. *Sleep Med* 2016; 26:37–44.
26. American Academy of Sleep Medicine. International classification of sleep disorders. 3rd ed. Darien, IL: American Academy of Sleep Medicine; 2014.
27. Rugh JD, Harlan J. Nocturnal bruxism and temporomandibular disorders. *Adv Neurol* 1988; 49:329–341.
28. Kato T, Lavigne GL. Sleep bruxism: a sleep-related movement disorder. *Sleep Med Clin* 2010; 5:9–53.
29. De Roeck J, van Hoof E, Cluydts R. Sleep-related expiratory groaning: a case report. *Sleep* 1983; 12:237.
30. Hao Z, Xu L, Zhang J, *et al.* Anatomical characteristics of catathrenia (nocturnal groaning) in upper airway and orofacial structures. *Sleep Breath* 2016; 20:103–111.
31. Guilleminault C, Hagen CC, Khaja AM. Catathrenia is not expiratory snoring. *Sleep* 2008; 31:774–775.
32. Guilleminault C, Hagen CC, Khaja AM. Catathrenia: parasomnia or uncommon feature of sleep disordered breathing? *Sleep* 2008; 31:132–139.
33. Tinuper P, Provini F, Bisulli F, *et al.* Movement disorders in sleep: Guidelines differentiating epileptic from nonepileptic motor phenomena arising from sleep. *Sleep Med Rev* 2007; 11:255–267.
34. Broughton R, Tolentino MA, Krelina M. Excessive fragmentary myoclonus in NREM sleep: a report of 38 cases. *Electroencephalogr Clin Neurophysiol* 1985; 61:123–133.
35. Sobreira-Neto MA, Pena-Pereira MA, Sobreira ES, *et al.* Excessive fragmentary myoclonus in patients with Parkinson's disease: prevalence and clinico-polysomnographic profile. *Sleep Breath* 2015; 19:997–1002.
36. Raccagni C, Loscher WN, Stefani A, *et al.* Peripheral nerve function in patients with excessive fragmentary myoclonus during sleep. *Sleep Med* 2016; 22:61–64.

In this study authors assessed patients with excessive fragmentary myoclonus during sleep using electrophysiological examination and found frequent peripheral nerve dysfunction, including polyneuropathy, L5 nerve root lesions, and benign fasciculations. These data suggest that despite the benign character of EFM, further diagnostic is necessary to rule out important comorbidities.

37. Allen RP, Picchietti D, Hening WA, *et al.* Restless legs syndrome: diagnostic criteria, special considerations, and epidemiology. A report from the restless legs syndrome diagnosis and epidemiology workshop of the National Institute of Health. *Sleep Med* 2003; 4:101–119.

38. Bassetti C, Mauerhofer D, Mathis J, *et al.* Restless legs syndrome: a clinical study of 55 patients. *Eur Neurol* 2001; 45:67–74.
 39. Montplaisir J, Boucher S, Poirier G, *et al.* Clinical, polysomnographic and genetic characteristics of restless legs syndrome: a study of 133 patients diagnosed with standard criteria. *Mov Disord* 1997; 12:61–65.
 40. Baumann C, Marti I, Bassetti CL. Restless legs symptoms without periodic limb movements in sleep and without response to dopaminergic agents: a restless legs-like syndrome? *Eur J Neurol* 2007; 14:1369–1372.
 41. Kallweit U, Siccoli M, Poryazova R, *et al.* Excessive daytime sleepiness in idiopathic restless legs syndrome: characteristics and evolution under dopaminergic treatment. *Eur Neurol* 2009; 62:276–278.
 42. Collado-Seidel V, Kohnen R, Samtleben W, *et al.* Clinical and biochemical findings in uremic patients with and without restless legs syndrome. *Am J Kidney Dis* 1998; 31:324–328.
 43. Abele M, Burk K, Laccione F, *et al.* Restless legs syndrome in spinocerebellar ataxia types 1, 2, and 3. *J Neurol* 2001; 248:311–314.
 44. Allen RP, Earley CJ. The role of iron in restless legs syndrome. *Mov Disord* 2007; 22 (Suppl 18):S440–S448.
 45. Ondo WG, Vuong KD, Jankovic J. Exploring the relationship between Parkinson disease and restless legs syndrome. *Arch Neurol* 2002; 59:421–424.
 46. Li X, Allen RP, Earley CJ, *et al.* Brain iron deficiency in idiopathic restless legs syndrome measured by quantitative magnetic susceptibility at 7 tesla. *Sleep Med* 2016; 22:75–82.
- This is an MRI imaging study to assess differences in brain iron concentrations between RLS patients and healthy controls and their relation to severity of disease and periodic limb movement during sleep. Its novel contribution to the field is the use of quantitative magnetic susceptibility measurements to an in-vivo indicator of brain iron content.
47. Gaig C, Iranzo A, Pujo M, *et al.* Periodic limb movements during sleep ■ mimicking REM sleep behavior disorder. *Sleep* 2016. [Epub ahead of print]
- In this study, authors used polysomnographic data to describe for the first time a subtype of periodic limb movements causing dream enacting behavior in the absence of RBD diagnosis.
48. Leng Y, Blackwell T, Stone KL, *et al.* Periodic limb movements in sleep are ■ associated with greater cognitive decline in older men without dementia. *Sleep* 2016; 39:1807–1810.
- In this study authors showed a relationship between PLMS and cognition suggesting greater cognitive decline associated with higher PLMS frequency.
49. Vetrugno R, Provini E, Meletti S, *et al.* Propriospinal myoclonus at the sleep-wake transition: a new type of parasomnia. *Sleep* 2001; 24:835–843.
 50. Antelmi E, Provini F. Propriospinal myoclonus: the spectrum of clinical and neurophysiological phenotypes. *Sleep Med Rev* 2015; 22:54–63.
 51. Sharpless BA. A clinician's guide to recurrent isolated sleep paralysis. *Neuropsychiatr Dis Treat* 2016; 12:1761–1767.
 52. Passouant P, Billiard M. The evolution of narcolepsy with age. In: Guilleminault C, Dement WC, Passouant P, editors. *Narcolepsy*. New York: Spectrum publications Inc.; 1976. pp. 179–200.
 53. Dahlitz M, Parkes JD. Sleep paralysis. *Lancet* 1993; 341:406–407.
 54. Denis D, Poerio GL. Terror and bliss? Commonalities and distinctions between sleep paralysis, lucid dreaming, and their associations with waking life experiences. *J Sleep Res* 2017; 26:38–47.
 55. Liskova M, Janeckova D, Kluzova Kracmarova L, *et al.* The occurrence and predictive factors of sleep paralysis in university students. *Neuropsychiatr Dis Treat* 2016; 12:2957–2962.
 56. Sharpless BA, Grom JL. Isolated sleep paralysis: fear, prevention, and disruption. *Behav Sleep Med* 2016; 14:134–139.
 57. Mellman TA, Aigbogun N, Graves RE, *et al.* Sleep paralysis and trauma, psychiatric symptoms and disorders in an adult African American population attending primary medical care. *Depress Anxiety* 2008; 25:435–440.
 58. Cheyne JA. Sleep paralysis: state transition disruption and narcolepsy. In: Bassetti CME, editor. *Narcolepsy and Hypersomnia*. New York: Dekker; 2006. pp. 109–117.
 59. Carrillo-Solano M, Leu-Semenescu S, Golmard JL, *et al.* Sleepiness in sleepwalking and sleep terrors: a higher sleep pressure? *Sleep Med* 2016; 26:54–59.
 60. Zadra A, Desautels A, Petit D, *et al.* Somnambulism: clinical aspects and pathophysiological hypotheses. *Lancet Neurol* 2013; 12:285–294.
 61. Bassetti CL. Sleepwalking (Somnambulism). In: Laurs S, Tononi G, editors. *The neurology of consciousness: cognitive neuroscience and neuropathology*. London, UK: Academic Press; 2009.
 62. Schenck CH, Mahowald MW. A polysomnographically documented case of adult somnambulism with long-distance automobile driving and frequent nocturnal violence: parasomnia with continuing danger as a nonsane automatism. *Sleep* 1995; 18:765–772.
 63. Bušková J, Piško J, Pastorek L, *et al.* The course and character of sleepwalking in adulthood: a clinical and polysomnographic study. *Behav Sleep Med* 2015; 13:169–177.
 64. Lopez R, Jaussent I, Scholz S, *et al.* Functional impairment in adult sleepwalkers: a case-control study. *Sleep* 2013; 36:345–351.
 65. Siclari F, Khatami R, Urbanik F, *et al.* Violence in sleep. *Brain* 2010; 133:3494–3509.
 66. Andersen ML, Poyares D, Alves RSC, *et al.* Sexsomnia: abnormal sexual behavior during sleep. *Brain Res Rev* 2007; 56:271–282.
 67. Perogamvros L, Aberg K, Gex-Fabry M, *et al.* Increased reward-related behaviors during sleep and wakefulness in sleepwalking and idiopathic nightmares. *PLoS One* 2015; 10:e0134504.
 68. Pedley TA, Guilleminault C. Episodic nocturnal wanderings responsive to anticonvulsant drug therapy. *Ann Neurol* 1977; 2:30–35.
 69. Oberholzer M, Poryazova R, Bassetti CL. Sleepwalking in Parkinson's disease: a questionnaire-based survey. *J Neurol* 2011; 258:1261–1267.
 70. Schenck CH, Milner DM, Hurwitz TD, *et al.* Dissociative disorders presenting as somnambulism: polysomnographic, video and clinical documentation. *Dissociation* 1989; 2:194–204.
 71. Schenck CH, Bundlie SR, Patterson AL, *et al.* Rapid eye movement sleep behavior disorder. *JAMA* 1987; 257:1786–1789.
 72. Iranzo A, Molinuevo JL, Santamaria J, *et al.* Rapid-eye-movement sleep behaviour disorder as an early marker for a neurodegenerative disorder: a descriptive study. *Lancet Neurol* 2006; 5:572–577.
 73. Postuma RB, Gagnon JF, Vendette M, *et al.* Quantifying the risk of neurodegenerative disease in idiopathic REM sleep behavior disorder. *Neurology* 2009; 72:1296–1300.
 74. Knudsen S, Gammeltoft S, Jennum PJ. Rapid eye movement sleep behaviour disorder in patients with narcolepsy is associated with hypocretin-1 deficiency. *Brain* 2010; 133 (Pt 2):568–579.
 75. Antelmi E, Pizzi F, Vandi S, *et al.* The spectrum of REM sleep-related ■ episodes in children with type 1 narcolepsy. *Brain* 2017. [Epub ahead of print]
- This study explores for the first time the presence, especially in REM stage, of motor complex behavior mimicking dream-enacting behavior in children with type 1 narcolepsy, highlighting further the cross-talk between RBD and narcolepsy.
76. Iranzo A, Molinuevo JL, Santamaria J, *et al.* Rapid-eye movement sleep behaviour disorder as an early marker for a neurodegenerative disorder: a descriptive study. *Lancet Neurol* 2006; 5:572–577.
 77. Poryazova R, Oberholzer M, Baumann CR, *et al.* REM Sleep behavior disorder in Parkinson's disease: a questionnaire-based Survey. *J Clin Sleep Med* 2013; 9:55–59.
 78. Di Fabio N, Poryazova R, Oberholzer M, *et al.* Sleepwalking, REM sleep behaviour disorder and overlap parasomnia in patients with Parkinson's disease. *Eur Neurol* 2013; 70 (5–6):297–303.
 79. Mahowald MW, Schenck CH. Status dissociatus – a perspective on states of being. *Sleep* 1991; 14:69–79.
 80. Provini F, Vetrugno R, Pastorelli F, *et al.* Status dissociatus after surgery for tegmental ponto-mesencephalic cavernoma: a state-dependent disorder of motor control during sleep. *Mov Dis* 2004; 19:719.
 81. Schenck CH, Montplaisir JY, Frauscher B, *et al.* Rapid eye movement sleep behavior disorder: devising controlled active treatment studies for symptomatic and neuroprotective therapy—a consensus statement from the International Rapid Eye Movement Sleep Behavior Disorder Study Group. *Sleep Med* 2013; 14:795–806.
 82. Panayiotopoulos CP, Michale M, Sanders S, *et al.* Benign childhood focal epilepsies: assessment of established and newly recognized syndromes. *Brain* 2008; 131:2264–2286.
 83. Bernasconi A, Andermann F, Cendes F, *et al.* Nocturnal temporal lobe epilepsy. *Neurology* 1998; 50:1772–1777.
 84. Scheffer IE, Bhatia KP, Lopes-Cendes I, *et al.* Autosomal dominant nocturnal frontal lobe epilepsy misdiagnosed as sleep disorder. *Lancet* 1995; 343:515–517.
 85. Provini F, Plazzi G, Tinuper P, *et al.* Nocturnal frontal lobe epilepsy. A clinical and polygraphic overview of 100 consecutive patients. *Brain* 1995; 122:1017–1022.
 86. Plazzi G, Provini E, Tinuper P, *et al.* Nocturnal frontal lobe epilepsy: clinical, video-polysomnographic, and genetic data in 100 cases. *Neurology* 1998; 50:A67.
 87. Tinuper P, Provini E, Bisulli F, *et al.* Hyperkinetic manifestations in nocturnal frontal lobe epilepsy. Semeiological features and physiopathological hypothesis. *Neurol Sci* 2005; 26:210–214.
 88. Siclari F, Nobili L, Lo Russo G, *et al.* Stimulus-induced, sleep-bound, focal seizures: a case report. *Sleep* 2011; 34:1727–1730.
 89. Vetrugno R, Manconi M, Ferini-Strambi B, *et al.* Nocturnal eating: Sleep-related eating disorder or night eating syndrome? A videopolysomnographic study. *Sleep* 2006; 29:949–954.
 90. Marconi S, Scarlatti F, Rizzo G, *et al.* Is nocturnal eating in restless legs syndrome linked to a specific psychopathological profile? A pilot study. *J Neural Transm (Vienna)* 2015; 122:1563–1571.
 91. Vainai P, Ferri R, Anelli M, *et al.* New data on psychological traits and sleep profiles of patients affected by nocturnal eating. *Sleep Med* 2015; 16:746–753.
 92. Thacker K, Devinsky O, Perrine K, *et al.* Nonepileptic seizures during apparent sleep. *Ann Neurol* 1993; 33:414–418.
 93. Trenkwalder C, Allen R, Hogl B, *et al.* Restless legs syndrome associated ■ with major diseases: a systematic review and new concept. *Neurology* 2016; 86:1336–1343.
- This article is a thorough review of the RLS with focus on associated comorbidities/etiology.
94. Winkelmann J, Schormair B, Xiong L, *et al.* Genetics of restless legs syndrome. *Sleep Med* 2017; 31:18–22.
- This article is a thorough review of the RLS genetics.

95. Xiong L, Montplaisir J, Desautels A, *et al.* Family study of restless legs syndrome in Quebec, Canada: clinical characterization of 671 familial cases. *Arch Neurol* 2010; 67:617–622.
 96. Lemke JR, Lal D, Reinthaler EM, *et al.* Mutations in GRIN2A cause idiopathic focal epilepsy with rolandic spikes. *Nat Genet* 2013; 45:1067–1072.
 97. Dauvilliers Y, Postuma RB, Ferini-Strambi L, *et al.* Family history of idiopathic REM behavior disorder: a multicenter case-control study. *Neurology* 2013; 80:2233–2235.
 98. Gan-Or Z, Mirelman A, Postuma RB, *et al.* GBA mutations are associated with rapid eye movement sleep behavior disorder. *Ann Clin Transl Neurol* 2015; 2:941–945.
 99. Gan-Or Z, Girard SL, Noreau A, *et al.* Parkinson's disease genetic loci in rapid eye movement sleep behavior disorder. *J Mol Neurosci* 2015; 56:617–622.
 100. Toffoli M, Dreussi E, Cecchin E, *et al.* SNCA 3'UTR genetic variants in patients with Parkinson's disease and REM sleep behavior disorder. *Neurol Sci* 2017; doi: 10.1007/s10072-017-2945-2. [Epub ahead of print]
 101. Gan-Or Z, Montplaisir JY, Ross JP, *et al.* The dementia-associated APOE epsilon4 allele is not associated with rapid eye movement sleep behavior disorder. *Neurobiol Aging* 2017; 49:218.e13–218.e15.
 102. Winkelman JW, Blackwell T, Stone K, *et al.* Genetic associations of periodic limb movements of sleep in the elderly for the MROS sleep study. *Sleep Med* 2015; 16:1360–1365.
 103. Denis D, French CC, Rowe R, *et al.* A twin and molecular genetics study of sleep paralysis and associated factors. *J Sleep Res* 2015; 24:438–446.
 104. Chahine LM, Xie SX, Simuni T, *et al.* Longitudinal changes in cognition in early Parkinson's disease patients with REM sleep behavior disorder. *Parkinsonism Relat Disord* 2016; 27:102–106.
- This prospective cohort study explores associations between RBD and cognition in patients with Parkinson's disease. Authors show that newly drug-naïve Parkinson's disease patients with RBD have, already at baseline, more cognitive deficits and with disease progression higher rates of cognitive decline compared to Parkinson's disease patients without RBD. These findings highlight the important prognostic value of RBD in Parkinson's disease.
105. Mollenhauer B, Zimmermann J, Sixel-Doring F, *et al.* Monitoring of 30 marker ■ candidates in early Parkinson disease as progression markers. *Neurology* 2016; 87:168–177.
- This longitudinal 2-year follow-up study investigates the progression of several candidate biomarkers over the course of Parkinson's disease. RBD may be a useful marker to monitor progression of Parkinson's disease. Daytime sleepiness progressively worsens over the course of Parkinson's disease.
106. Sixel-Doring F, Zimmermann J, Wegener A, *et al.* The evolution of REM ■ sleep behavior disorder in early Parkinson disease. *Sleep* 2016; 39:1737–1742.
- This observational study confirms the progressive nature of RBD in early Parkinson's disease despite initiation of dopaminergic treatment. Compared to healthy controls, initially de novo Parkinson's disease patients show a significant increase in the frequency of polysomnographic confirmed RBD in the 2-years follow-up.
107. Iranzo A, Tllosa E, Gelpi E, *et al.* Neurodegenerative disease status and postmortem pathology in idiopathic rapid-eye-movement sleep behaviour disorder: an observational cohort study. *Lancet Neurol* 2013; 12:443–453.
 108. Nobili L, Francione S, Mai R, *et al.* Surgical treatment of drug-resistant nocturnal fronta lobe epilepsy. *Brain* 2007; 130:561–573.
 109. Mathis J, Hess CW, Bassetti C. Isolated mediotegmental lesion causing narcolepsy and rapid eye movement sleep behaviour disorder: a case evidencing a common pathway in narcolepsy and rapid eye movement sleep behaviour disorder. *J Neurol Neurosurg Psychiatry* 2007; 78:427–429.
 110. Iranzo A, Graus F, Clover L, *et al.* Rapid eye movement sleep behavior disorder and potassium channel antibody-associated limbic encephalitis. *Ann Neurol* 2006; 59:178–181.
 111. Ohayon MM, Zulley J, Guilleminault C, *et al.* Prevalence and pathologic associations of sleep paralysis in the general population. *Neurology* 1999; 52:1194–1200.
 112. Labelle MA, Desautels A, Montplaisir J, *et al.* Psychopathologic correlates of adult sleepwalking. *Sleep Med* 2013; 14:1348–1355.
 113. Ghanizadeh A. ADHD, bruxism and psychiatric disorders: does bruxism increase the chance of a comorbid psychiatric disorder in children with ADHD and their parents? *Sleep Breath* 2008; 12:375–380.
 114. Mackie S, Winkelman JW. Restless legs syndrome and psychiatric disorders. *Sleep Med Clin* 2015; 10:351–357.xv.
 115. Snitselaar MA, Smits MG, Spijker J. Prevalence of restless legs syndrome in adult ADHD and its subtypes. *Behav Sleep Med* 2016; 14:480–488.
 116. Heim B, Djamshidian A, Heidbreder A, *et al.* Augmentation and impulsive ■ behaviors in restless legs syndrome: coexistence or association? *Neurology* 2016; 87:36–40.
- In the present study, authors examined RLS patients for impulsive behavior. Patients with RLS with augmentation have an almost six-fold increased risk of exhibiting impulsive behavior, suggesting that augmentation and ICDs are related and may share a common pathophysiology.
117. Falisi G, Rastelli C, Panti F, *et al.* Psychotropic drugs and bruxism. *Expert Opin Drug Saf* 2014; 13:1319–1326.
 118. Uca AU, Uguz F, Kozak HH, *et al.* Antidepressant-induced sleep bruxism: prevalence, incidence, and related factors. *Clin Neuropharmacol* 2015; 38:227–230.
 119. Pressmann MR. Factors that predispose, prime and precipitate NREM parasomnias in adults: clinical and forensic implications. *Sleep Med Rev* 2007; 11:5–30.
 120. Boeve BF. Pathophysiology of REM sleep behavior disorder and relevance to neurodegenerative disease. *Brain* 2007; 130:2770–2788.
 121. Bassetti C, Vella S, Donati F, *et al.* SPECT during sleepwalking. *Lancet* 2000; 356:484–485.
 122. Jalal B, Ramachandran VS. Sleep paralysis, the ghostly bedroom intruder' and out-of-body experiences: the role of mirror neurons. *Front Hum Neurosci* 2017; 11:92.
 123. Rizzo G, Li X, Galantucci S, *et al.* Brain imaging and networks in restless legs syndrome. *Sleep Med* 2017; 31:39–48.
 124. Katz PS. Neurons, networks, and moto behavior. *Neuron* 1996; 16:245–253.
 125. Tassinari CA, Rubboli G, Gardella E, *et al.* Central pattern generators for a common semiology in fronto-limbic seizures and in parasomnias. A neurothologic approach. *Neurol Sci* 2005; 26:225–232.
 126. Mahowald MK, Schenck CH. Dissociated states of wakefulness and sleep. *Neurology* 1992; 42 (Suppl 6):44–52.
 127. Schindler K, Gast H, Bassetti C, *et al.* Hyperperfusion of anterior cingulate gyrus in a case of paroxysmal nocturnal dystonia. *Neurology* 2001; 57:917–920.
 128. Terzaghi M, Sartori I, Tassi L, *et al.* Evidence of dissociated arousal states during NREM parasomnia from an intracerebral neurophysiological study. *Sleep* 2009; 32:409–412.
 129. Tinuper P, Bisulli F, Provini F. The parasomnias: mechanisms and treatment. *Epilepsia* 2012; Suppl 7:12–19.
 130. The International Restless Legs Syndrome Study G. Validation of the International Restless Legs Syndrome Study Group rating scale for restless legs syndrome. *Sleep Med* 2003; 4:121–132.
 131. Manni R, Terzaghi M, Repetto A. The FLEP scale in diagnosis nocturnal frontal lobe from epilepsy, NREM and REM parasomnias: data from tertiary sleep and epilepsy unit. *Epilepsia* 2008; 49:1581–1585.
 132. Garcia-Borreguero D, Ferini-Strambi L, Kohnen R, *et al.* European guidelines on management of restless legs syndrome: report of a joint task force by the European Federation of Neurological Societies, the European Neurological Society and the European Sleep Research Society. *Eur J Neurol* 2012; 19:1385–1396.