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REVIEW ARTICLES

Dream enactment behavior: review for the clinician

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Dream enactment behavior commonly occurs on occasion in normal children and adults. Disruptive and frequent dream enactment behavior may come to the attention of the clinician either as the primary reason for consultation or as a prominent characteristic of a patient with other sleep disorders. Questioning patients with chronic neurologic and psychiatric disorders may also reveal previously unrecognized behavior. In the absence of sleep pathology, process of dream enactment likely begins with active, often emotionally charged dream content that may occasionally break through the normal REM sleep motor suppressive activity. Disrupted sleep resulting from many possible causes, such as circadian disruption, sleep apnea, or medications, may also disrupt at least temporarily the motor-suppressive activity in REM sleep, allowing dream enactment to occur. Finally, pathological neurological damage in the context of degenerative, autoimmune, and infectious neurological disorders may lead to chronic recurrent and severe dream enactment behavior. Evaluating the context, frequency, and severity of dream enactment behavior is guided first and foremost by a structured approach to the sleep history. Physical exam and selected testing support the clinical diagnosis. Understanding the context and the likely cause is essential to effective therapy.

Keywords: Dream enactment behavior, parasomnia, REM-sleep, REM sleep behavior disorder, Parkinson disease, neurologic disorders, psychiatric disorders, pathophysiology, evaluation, treatment

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BRIEF SUMMARY

Current Knowledge/Study Rationale: A comprehensive review for clinicians who care for patients is needed. We undertook a review of the current peer-reviewed publications and present the definition, normal nature, and sleep physiology and pathophysiology of dream enactment behavior. The frequencies and manifestations of different disease states are also presented. Current management is summarized.

Study Impact: Dream enactment behavior is common and can be normal. A review for clinicians is made to better inform health care professionals on its assessment and management.

INTRODUCTION

Although dream enactment behavior is classically associated with rapid eye movement (REM) sleep behavior disorder (RBD), it has, in fact, a broad spectrum of causes and severity. We review the overall mechanisms and causes of dream enactment behavior, in RBD and beyond.

REVIEW

Definitions of dream enactment behavior and related behaviors

The acting out of dreams, characterized by body movement, emotional expression, or audible verbalization of dream content has been termed dream enactment behavior (DEB) when the body's physical expression is followed by the individual recalling a dream. The phenomenon can range in intensity and frequency in the normal population. In most cases, DEB occurs during the transition from sleep to wake, which allows the individual to recall the event coincident with dream imagery immediately after it took place. This is unlike typical somnambulism or somniloquy, which generally occur during non-REM (NREM) sleep without any dream recall. Dream enactment behavior is the defining clinical feature of RBD.[1] Patients are reported by their bed partners to scream, speak, fall, or move their arms and legs to mimic their dreams.[2] Injuries and violence can occur to the patient and bed partner as the result of DEB. DEB is primarily suspected on the basis of a medical history although polysomnography adds important diagnostic information by assessing sleep fragmentation, potential NREM and REM parasomnia, and REM sleep without muscle atonia.[1–3]

From a clinician's point of view, unusual or violent physical movement in sleep may be directly or indirectly observed by the sleeper, bed partner or others sharing the sleeping environment, with normal efforts to comprehend the unusual activity. At times, these reports may be confused, with assumptions that the

sleeper "must have been dreaming" and other factors that may question the reliability of the dream recalled. Research oriented definitions may seek to restrict the definition for research participants to DEB associated only with REM sleep. Yet even individuals with typical common NREM sleep parasomnias of sleep terrors and somnambulism may often report dream recall when closely questioned[4,5] as well as more complex but well characterized parasomnias including dissociative disorders[6] and parasomnia overlap disorder[7] to give a few examples.

For the purposes of this review, we concentrated on REM sleep parasomnias, but allowed a wide review which included all parasomnias with potential dream recall. This is to allow the clinician faced with a patient with DEB and possible dream recall (according to the sleeper or the witness) and who may not have timely access to polysomnography, a more comprehensive and informative review.

Research definitions and questionnaires

Of the questionnaires designed to date, those for adults are generally more specific than the ones for pediatric uses. In children, most DEB studies have been assessed via questionnaires administered to parents. Although several questionnaires (eg, Pediatric Sleep Questionnaire, Children's Sleep Habit Questionnaire, Children's Sleep Behavior Scale, and Children's Sleep Hygiene Scale) have been used in studies in which DEBs in children were assessed, no questionnaire to our knowledge has been validated for assessing only DEBs during NREM or REM sleep.[1] Of all the scales listed, the Behavioral Evaluation of Disorders of Sleep Scale contains detailed possible DEB symptoms, state of awareness at the event, and other possible excluding criteria (ie, apnea).[8] With incorporation of additional questionnaires to help exclude other sleep disorders that may mimic DEB (eg, RLS, confusion arousal due to growing pains or cramp and epilepsy), the questionnaire can be good candidate to evaluate possible DEB symptoms in children. In adults, on the other hand, several questionnaires have been designed to screen DEB; most of these are designed primarily to screen for potential RBD (**Table 1**). The Mayo Sleep Questionnaire, the RBD Single Question Questionnaire (RBD-1Q), and the Innsbruck Sleep Behavior Inventory are among the short questionnaires, ideal for fast screening in general practice. Both the REM Sleep Behavior Disorder Questionnaire-Hong Kong (RBDQ-HK) and RBD-1Q have relatively good positive predictive value and negative predictive value.[9,10] With modification, RBDQ-HK can also be used to tract prospective changes in RBD symptom severity.

Alternative behaviors that may be confused with dream enactment behavior

When explicitly questioned during or right after the event, vague dream mentation has been reported primarily in patients with typical sleepwalking and sleep terrors. The usual content is less elaborate and more static with less detail and links with lived experience.[4] A differentiating characteristic has been the overwhelming physical manifestation of sleepwalking (robotic movement, leaving the bed, repetitive or banal behavior) or sleep terrors that dominate the events. With these episodes, dream mentation is occasionally described if the patient is questioned. However, dream imagery is not a major perceptual component after the experience (which is usually forgotten). Confusional arousals may also have associated dream imagery but are marked by their major motor and confusional components in transition to waking and occur often in the setting of sleep disruption or intoxication.[20]

Potential mechanisms of dream enactment behavior

Several potential mechanisms of DEB have been proposed and relate to the expected causes of DEB in specific individuals. This includes direct anatomical disruption of brainstem nuclei, pharmacologic disruption of REM-sleep motor function, overactive dream intensity such as a nightmare which overwhelms otherwise normal REM-sleep neurological function, and sleep disruption leading to transient mixed-sleep states. Anatomical disruption of brainstem nuclei are the focus of the neurologic lesions that may lead to DEB. For this review, the mechanisms are categorized by the sleep stages when the DEB episode occurs and its-related anatomical structure.

For NREM parasomnias, DEB mechanisms in NREM sleep are relatively complex and unclear. Several structures within the limbic systems have been hypothesized as a generator for the DEBs in non-REM sleep, this includes: amygdala and thalamus.[4,21,22] Future medical imaging studies will be needed to understand the mechanisms involved.

For REM parasomnias, the primary mechanism for DEB is believed to be caused by a direct disruption of brainstem nuclei that maintain the atonia of REM sleep. Although the exact mechanism triggers the transition from non-REM to REM sleep remains unclear, most studies agreed that the REM-on glutamatergic neurons residing in sublaterodorsal nucleus (SLD) is responsible for the loss of muscle tone.[23]

In parkinsonism, the RBD mechanism is believed to be initiated in the brainstem region (namely pons and medulla). In fact, in a recent review by Borghammer and Van Den Berge pointed out that by summarizing the imaging studies available to-date, regardless of one's phenoconversion status, those with RBD symptoms have more atrophy signs in the pontine region than those without.[24] This phenomenon is align with findings of DEBs inducing by lesions in the dorsal pontine (further described in the neurology section below).[25] In RBD, it would be expected that the putative "flip-flop switch"[23] may be caught in an incompletely coordinated state, releasing motor inhibition during intense dream activity. This would manifest as major body motor behavior during a dominant REM cognitive state, resulting in an episode of DEB. Although lesions in medulla regions, where the downstream GABA-/glycinergic neurons reside, could also restore the muscular activities during the REM sleep in animal models, the clinical evidence supporting this hypothesis remains limited due to its role in regulating cardiorespiratory functions.[26] In some cases, sleep-disordered breathing, which may cause the medullary homeostasis to be involved, has been found inducing "DEBs" in both children and adults.[27,28] A prescription of continuous positive airway pressure will help differentiate the diagnosis if the DEBs ceases to occur after the treatment.

Table 1—Published questionnaires for dream enactment behavior in the context of suspected REM sleep behavior disorder.

	Innsbruck REM Sleep Behavior Disorder Inventory	RBD Single Question Questionnaire	Mayo Sleep Questionnaire	REM Sleep Behavior Disorder Questionnaire-Hong Kong	REM Sleep Behavior Disorder Screening Questionnaire	
No. of items	5	1	1 + 5 Conditional questions	13	13	
Interviewee	Patients (with/without bed partner)	Patients, bed partner, caregiver	Bed partner	Patients and/or bed partner	Patients (with/without bed partner)	
Validation place	Austria	Canada	United States	Hong-Kong, Korea, Japan, China	China, United States, Italy, Korea, Japan, Germany, Turkey	
Cohort detail	Sleep disorder, neurodegenerative disease	Community, sleep disorder	Community, neurodegenerative disease	Sleep disorder, neurodegenerative disease, mental illness	Sleep disorder, PD	
Cut-off	25% Positive rate	1 (Positive)	1 (Positive)	19/100[9,11–13]	8[14]	5[15]
Polysomnogram	Yes	Yes	Yes	Yes	Yes	Yes
Sensitivity	91.4[16]	93.8[10]	96.6[17–19]	92.5	82.9	97.6
Specificity	85.7	87.5	84.7	89.3	82	45.9
Estimated PPV	6.1	7.05	6	8.01	4.5	1.8
Estimated NPV	93.9	94	94	99.9	95.6	98.2
Note	No. of positive symptoms ÷ no. of answered questions = 25%		Question 1 and the subquestions from the original questionnaire	Factor 2 may be used as an alternative; 2 apnea questions may be applied[13]	Remove item 10 crude SN%: 91 [85–95]; crude SP%: 77 [66–85][15]	

NPV = negative pressure ventilation, PD = Parkinson disease, PPV = positive pressure ventilation, REM = rapid eye movement, SN% = sensitivity, SP5 = specificity.

Another possible mechanism of DEBs during REM sleep involves the disruption of activities in the thalamic-hypothalamic-pathway. This is most commonly associated with narcolepsy-associated RBD. However more functional anatomy studies will be needed to assess its role in affecting the SLD neurons in DEBs. The more intense and frequent DEB observed in RBD and the degenerative neurological diseases may be explained better by the decreased motor inhibition resulting from permanent cell loss in the ventral medulla and the pontine tegmentum. The decreased meso-pontine glutaminergic neural activity that projects to the medulla and spinal cord results in reduced REM-sleep without atonia. The permanent state of reduced motor inhibition allows for frequent, nightly detectable RSWA and frequent, disturbing and potentially dangerous episodes of DEB.[3]

Another potential mechanism is the overwhelming of the normal neuromuscular inhibition being reversed by sufficiently perceptual, dramatic and emotionally intense dreams. Nielsen and Kuiken proposed in 2013 that an individual's propensity to resonate with the emotions and the actions of other characters during dreaming may depend upon neural networks that underlie within the basic social cognition.[29] Specifically, this includes the mentalizing network and the mirror neuron system—two anatomically distinct networks subserving social cognition. Mirror neurons are known to be important for understanding the actions of other people, but some studies have indicated that the mirror neuron system also helps us understand both the actions and intentions of other people with a sense of empathy.[30,31] Motor-affective resonance generated by the mirror neuron system may mediate both enactment of dream imagery during sleep and emotional empathy during wakefulness. Furthermore, the authors suggest that dream enactments reflect more basic and developmentally earlier resonances rather than explain perceived affect in others. Nielsen and Krüiken found that several types of mirror behaviors (ie, imitative resonance with others' movements and emotional expressions, resonance in imagination and dream imagery) during waking are correlated with DEB and involve the activation of a common neural network that mediates enactment of dream imagery in sleep and emotional empathy while awake.[29]

When highly emotional dream content occurs, there would be expected to be a greater activation of the centers of the amygdala. This relative overactivity may escalate to an intensity that escapes the normal REM-sleep de-activation.[32] This may partly explain the frequent finding of DEB in psychiatric patients.[33] This may be a primary explanation for the DEB seen in patients with posttraumatic stress disorder (PTSD) without concomitant brain injury. This, with the physical and environmental sleep disruption that occurs in pregnancy and the postpartum period, might explain the high prevalence of DEB in this otherwise normal population (its brief, benign and episodic nature may illustrate that both intense dream activity and sleep disruption are required). The characteristic hyperarousal

seen in PTSD may span both the waking and sleeping life of the affected individual. It has been proposed that the neurobiological basis of the sleep disturbances in PTSD stem from "overdrive" during sleep and nightmares originate in or are amplified by the amygdala and medial prefrontal cortex,[34] which is distinct from the degenerative neurobiological basis of RBD in the context of synucleopathies. This hyper-functioning of the amygdala and medial prefrontal cortex is also proposed as the neurobiological basis of trauma associated sleep disorder (TASD).[35] As such there is no classical "lesion" in TASD and PTSD, but disturbed functioning due to the original trauma, conditioning and memory consolidation. This suggests that the hyper-functioning neuropsychological basis of these disorders in conjunction with normal anatomy may be amenable to therapy.

Disrupted sleep has been long considered a trigger of sleepwalking in sleepers who are at risk with either sleep terrors or somnambulism. It may also provoke episodes of DEB when the sleeper is at risk of other parasomnias. This has been seen with environmental noise, an individual waking the sleeper, as well as co-incident sleep fragmentation by another sleep disorder such as obstructive sleep apnea. The explanation proposed is that a transient period of mixed sleep states coincide in the sleeper where activated motor systems typical of the waking state are coincident with a dream sleep state of consciousness. Chronic sleep fragmentation by severe obstructive sleep apnea can clinically mimic RBD.[36]

Medication and certain states of intoxication may variably affect the sleeper who may be anatomically at risk (such as presymptomatic Parkinson disease) and modify the balance of sleep and motor inhibition that normally occurs in REM sleep. This has been one of the proposed mechanisms of the etiology of DEB associated with SSRI and other antidepressant medications (see below).

Frequency and prevalence of dream enactment behavior

Information about the prevalence and characteristics of DEB in both healthy and clinical populations has relevance in the diagnosis of RBD and other parasomnias (**Table 2**). Identifying DEB may be an important clinical clue in recognizing psychiatric or neurological conditions. For example, it is well established that RBD is an early symptom of future onset Lewy body dementia, while full diagnosis of RBD is often preceded by many years of DEB.[37] It is also known that individuals with autism spectrum disorder or with major depressive disorder may experience DEB at least once a week (**Table 3**).

In healthy populations, no clear link has been shown between frequency of DEB and future RBD symptoms while frequency and prevalence of DEB among the elderly is unknown. The goal of the following sections is to clarify what is known about DEB in relation to sleep and psychiatric and neurological disorders.

Common situations where dream enactment behavior may be considered normal

Children may have occasional DEB, but no known prevalence estimates have been published. In early adulthood, the majority of university students report some form of occasional DEB.[37] These behaviors were often simple and not disruptive and could include brief somniloquy or sexual arousal. Compared to women without pregnancy, of whom 56% report DEB, pregnant women reported less DEB (40%), but postpartum women reported more DEB (63%).[38] More overt motor activity was reported in postpartum women (57%) compared to a quarter of both women without pregnancy and pregnant women. The more recent sleep studies in pregnant populations, however, did not report about DEB.[42–44] A study based on a Canadian epidemiology cohort, the Canadian Longitudinal Study of Aging,[45] has estimated that among older adults aged 45–85, DEB occurs in 11% of the population, of which a majority may be normal episodic events while a minority may be due to neurological desease.[40] A common health condition associated with DEB is fever.[46,47] In one of the early studies, febrile illness was found to be associated with an increase in events of night terror and sleepwalking in children.[47]

Psychological and psychiatric causes of recurrent dream enactment behavior

Episodes of DEB may be reported in distressing or emotionally charged situations, such as sleep deprivation or bereavement. They can also occur in the context of psychiatric and psychological disorders, of which the most common are depression and posttraumatic stress disorder (**Table 3**).[32,48] The close association between DEB and antidepressants suggests that the sleep disorder is secondary to antidepressant medication.[10,11] However, REM-related muscle atonia is often not fully restored after the withdrawal of antidepressants, suggesting that the medication may precipitate diagnosis rather than cause sleep disorder in susceptible individuals,[32] suggesting there may be an underlying neurodegenerative condition that is exposed by antidepressants.[3,49,50] There have been several case reports showing association between DEB and psychiatric disorders such as PTSD, anxiety, and depression; these studies describe REM motor abnormalities that do not meet specific criteria for RBD.[51,52]

The type and the frequency of DEBs present in normal populations may be different from DEBs in patients with neurological disease. In a study of 1140 undergraduate students, Nielsen et al had documented behaviors that resemble those seen routinely in RBD evaluations but was found less frequent than previously estimated.[37] Similar to patients with RBD, student participants reported behaviors such as speaking, laughing, motor activity, and sexual activity as well as negative emotional responses, such as crying, anger and fear. Participants reported manifesting these behaviors on an average of 6 times per year (unlike patients with RBD who can experience DEBs several times a night).[74] The authors interpret their findings as forms of dream enactment behaviors reflecting a predispositio to heightened attentional engagement with imagery processes.[37] They suggest that recurrent DEB may serve to regulate affect. In a review paper published in 2008, Cartwright suggested that DEB is a phenomenon in response to an overload of new challenges to a system, combined with a genetic deficit in sustaining sleep motor atonia; this model proposes that DEB is a mixed picture of psychological precipitating factors superimposed on a genetic vulnerability.[75]

Table 2—Prevalence rates of dream enactment behavior in normal populations.

Population	Age	Definition	Prevalence	Reference
University students	Mean age 20	Any motor activity associated with dream imagery	66%	37
Pregnant women	Mean age 31	Any motor activity associated with dream imagery	40%	38
Postpartum women	Mean age 30	Any motor activity associated with dream imagery	63%	38
Adult population	24 or older	Moving in dreams	5.9% in men, 4.1% in women	39
Older adults	45 to 85	Moving in dreams	10.9%	40
Adult farmers		Moving in dreams	7.9%	41

Nightmare disorder with DEB is often misdiagnosed as psychiatric disorder. Nightmare disorder involves recurrent nightmares producing awakenings usually in the second half of the night and difficulty returning to sleep.[76] Various primary sleep disorders may also contribute to symptoms of nightmares and dream enactment behaviors; various mental health issues may also contribute to nightmares (eg, PTSD, substance abuse, stress, anxiety, borderline personality disorder, and schizophrenia).[76] PTSD-associated nightmares have been the most studied and, in fact, nightmares are part of the diagnostic criteria symptom cluster of intrusive/re-experiencing of the traumatic event.[76–78] Eighty percent of patients with PTSD reported PTSD-associated nightmares.[76] Although they may be difficult to differentiate, both DEB and nightmares may result in adverse health effects, psychological disturbances, and disrupted sleep; therefore, treatment of the sleep disorder is often recommended.[79] Sleep-related dissociative disorder is an altered state of consciousness characterized by dissociative episodes that occur near sleep-wake transitions that arise from wakefulness. These episodes are commonly associated with a history of trauma and other psychiatric disorders (eg, PTSD, borderline personality disorder).[70–73] Episodes may include walking, self-mutilation, and driving, with subsequent amnesia for these events. Episodes may also involve violent re-enactments of previous traumas and can result in violence and injury.

Dream enactment behavior due to neurological conditions

Besides psychiatric conditions, DEB is mostly commonly reported in patients with neurological conditions. Since apnea, RLS, and sleep epilepsy may often be confused as DEB when assessment of DEB is by questionnaires, the following section prioritizes the prevalence of DEB based on studies with video-polysomnography–confirmed cases (**Table 4**). For rare diseases or genetic diseases, case series will be presented if available. A series of questionnaire-based studies are presented when video-polysomnography studies are absent.

Synucleinopathies and degenerative movement disorders

Both RBD and NREM sleep DEB may be common presentations in neurological disorders. RBD is often reported with synucleinopathies and dementia. Of all reported synucleinopathies, the pooled prevalence is highest among those with multiple system atrophy (88%),[98] followed by dementia with Lewy bodies (DLB) (76%)[129] and Parkinson disease (PD) (23.6%; range 4.3–69.4%) (**Table 4**).[130] From the largest video-polysomnography study in PD, it is estimated that close to half of the PD patients (46%) may experience dream enactment during REM sleep.[109] Approximately 25% of newly diagnosed PD patients have RBD.[110] There have also been case reports of DEB in juvenile PD.[131] Night-terrors and sleepwalking have also been reported in patients with PD.[111,132,133] Based on the existing results, the prevalence for sleepwalking (2–3.6%) and night terror may not be different from the estimated prevalence in adults.[134]

Other than synucleinopathy-related movement disorders, DEB occurs uncommonly patients with amyotrophic lateral sclerosis (ALS)[135]; in one sample, 2 of 41 ALS patients (4.9%) showed signs of DEB during REM sleep and another 2 had REM sleep without atonia.[80] Most studies of DEB on patients with movement disorders focused primarily on RBD. These include: tauopathies such as progressive supranuclear palsy,[88,89,99] Huntington chorea,[136] Creutzfeldt-Jakob disease,[137,138] and Guadeloupean atypical parkinsonism.[139] However, studies to date were all either case reports or case series; larger cohort studies will be needed to determine the prevalence rate.

Alzheimer disease (AD) is the most common form of dementia, followed by vascular dementia and DLB.[140] The relationship between AD and dream enactment behavior is relatively unclear; there have been case series on RBD but the association remains unclear since most studies were performed without video-polysomnography or a definite diagnosis of AD.[112,113,141] Of the current report, idiopathic RBD patients rarely phenoconvert into AD.[114] In the autopsy report by Schenck and his colleagues, 2 AD-RBD patients both showed DLB pathology, which may indicate that the AD-RBD patients may be a subtype of Lewy body disease.[142] The most definitive study comes from a neuropathological series of 172 patients with RBD associated with neurodegenerative disease. In this series, 98% of those with polysomnogram-confirmed RBD had Lewy body deposition in brain.[143] For this reason, in the DLB consensus criteria, any dementia patient with polysomnography-proven RBD by definition meets criteria for probable DLB.[144] DEB was also reported in patients with frontotemporal dementia.[145] To our knowledge, it is relatively rare and we were therefore unable to assess the prevalence.

Narcolepsy

Based on the largest video-polysomnography study to date, 27% of patients with narcolepsy type 1 also had RBD.[119] Up to 15% of patients with narcolepsy type 1 and RBD may also

Table 3—Psychological and psychiatric causes of dream enactment behavior.

Cause	DSM-5 Category	Associated Features	Frequency of DEB	Reported Correlates	References
Anxiety	Anxiety disorders	(This section does not refer to anticipatory nocturnal anxiety or "sleep phobia") • DEB may persist after treatment of daytime anxiety; • Antidepressants may trigger early clinical presentation of an RBD due to underlying neurodegeneration Note: *Experiencing DEBs does not indicate high daytime anxiety and depression scores.*	No information	• Taking SSRIs • Taking SNRIs • Taking tricyclic antidepressants • Depression • Insomnia • Signs of neurodegeneration	5,49,53
Autistic disorder, Asperger disorder, or pervasive developmental disorder not otherwise specified	Autism spectrum disorder	• Disorders of initiation and maintenance of sleep are almost universal • Clonazepam is effective in treating abnormal motor control in sleep • SSRIs may cause or contribute to lack of muscle atonia during REM sleep • Children who are not medicated may not experience REM sleep without atonia • 54% of children experience parasomnias • Younger children experience more parasomnias	May occur nightly	• Dysregulation of melatonin synthesis • Sensitization to environmental stimuli • Insomnia • OSA • Periodic limb movements • Bruxism • Delayed sleep phase • Anxiety • Depression • Epilepsy	54–59
Bereavement	Major depressive disorder and depressive episodes	• Occurs mostly within 5 years of loss; • Dream/reality confusion; • Dreams produce intensely real endings, which produce awakenings.	No information	• Anxiety dreams • Existential dreams • Nightmares • Experiencing a range of distressing emotions	60
Depression	Major depressive disorder and depressive episodes	• Antidepressant medication use is associated with more frequent DEBs • RBD-associated laughter is associated with depression • REMREEA was associated with BDI and HADS scores (mood symptoms) and use of antidepressants • Antidepressants may trigger early clinical presentation of an RBD due to underlying neurodegeneration • Failure to recall enacted dreams Note: *Experiencing DEBs is not indicative of high daytime anxiety and depression scores.*	At least once weekly	• Taking SSRIs • Taking SNRIs • Taking tricyclic antidepressants • Taking dopaminergic drugs • Insomnia • Anxiety • Signs of neurodegeneration	5,33,49, 61–65
Postpartum state	Not applicable	• 63% of new mothers report some form of DEB (including expressing emotions, motor activity and speaking) • Dream-associated motor activity increases postpartum • Dreams mostly involve infants • The propensity to enact nightmares is possibly due to an increase in their emotional intensity • REM sleep deprivation may produce more intense dreams when sleep recovery occurs	At least once in the 12 weeks postpartum	• Frequent sleep interruption • Fluctuation in hormones such as oxytocin, prolactin, and vasopressin • Insecure attachment • Global symptom severity • Postpartum somniloquy or somnambulism • Dream anxiety • Nightmares	38,60
Posttraumatic stress disorder	Trauma-associated sleep disorder (trauma- and stressor-related disorders)	• Nightmare theme specifically linked to the personally experienced traumatic event ("flashbacks") • Nightmares occur both in NREM and REM sleep • Excessive movements • Complex vocal and motor behaviors during sleep • Presents at a considerably younger age than is typical for RBD	1 Event every 4–6 weeks	• RBD • General REM sleep disturbances • Taking SSRIs • Reduction in locus coeruleus neuronal activity • History of TBI • Insomnia • Nightmares • Parasomnias	52,66–69

(continued on following page)

Table 3—Psychological and psychiatric causes of dream enactment behavior. (continued)

Cause	DSM-5 Category	Associated Features	Frequency of DEB	Reported Correlates	References
Sleep-related dissociative disorder	Dissociative disorders	• Symptom criteria include the diagnosis of a dissociative disorder based on DSM criteria, with dissociative episodes arising during the main sleep period • Vivid dreams, nightmares, dissociation and symptoms of schizotypy • Potentially harmful behaviors during episodes, including walking, self-mutilation, and driving, with subsequent amnesia for these events • Occur either during the transition from wake to sleep or during awakenings at night • 5.3% of individuals who experience sleep-related injury, were identified as having sleep related dissociative disorder	No information	• Abnormal nocturnal behaviors, such as dream enactments • Schizotypal Personality; Disorder • PTSD • Narcolepsy • Nightmare disorder	70–73

BDI = Beck Depression Inventory, DSM-5 = *Diagnostic and Statistical Manual of Mental Disorders*, 5th ed., BDS = Behavior Dimensions Scale, HADS = Hospital Anxiety and Depression Scale, OSA = obstructive sleep apnea, RBD = REM sleep behavior disorder, REMREEA = sREM-related EMG activity, SSRI = selective serotonin reuptake inhibitor, SNRI = serotonin-norepinephrine reuptake inhibitor, TBI = traumatic brain injury.

exhibit complex motor activities during NREM sleep, a characteristic which is not typical in the idiopathic RBD patients.[146] A prospective polysomnographic study over 2 consecutive nights found that 40% of children with narcolepsy type 1 exhibited simple gesturing during NREM sleep and 70% during REM sleep.[147] The prevalence of RBD in adults with narcolepsy type 2, relative to type 1, is believed to be less common, ranging from 0.3 to 15%.[120,121,148] However, the prevalence rates of both RBD and any movements during nocturnal sleep were lower in drug-naïve narcolepsy.[121] Other types of parasomnia, including nightmare disorder and night terror, during NREM sleep may also be common, as described in one of the earliest studies.[124] Nightmares have been found to occur in a third of patients with narcolepsy, 52% of the time reported in narcolepsy type 1 and 20% in type 2.[122]

Autoimmune disorders and infectious diseases

DEBs have not been reported in most of the common autoimmune disorders, other than multiple sclerosis (1.4%)[149,150] but have been found in some rare autoimmune diseases, most notably in narcolepsy. With advances in the characterization of autoimmune diseases, descriptions of DEB have been published in a few patients with anti-IgLON5,[151,152] or other paraneoplastic and autoimmune encephalitis. In one of the first case series, all anti-IgLON5 patients showed abnormal sleep behaviors,[152] but in a later case series of 20 participants the prevalence was much lower, 27%.[153] Prevalence of apnea symptoms greatly increases during the course of the disease, introducing a possible confounder of apnea-induced sleep disruption/dream-related movement.[154] Further investigation will be needed to clarify the impact of treating sleep apnea and persistent DEBs in these patients.

Complex DEB has also been reported in autoimmune and paraneoplastic cerebellar degeneration,[155] anti-Ma2 encephalitis,[156] voltage-gated potassium channel complex autoimmunity,[157] LIG1-related limbic encephalitis,[158,159] as well as anti-NMDA receptor encephalitis.[125] Among 19 participants with autoimmune encephalitis (mostly anti-LIG1 or NMDA receptor encephalitis) evaluated with video-polysomnography, only 3 showed REM sleep without atonia. One possible explanation for the absence of video-polysomnographic DEB may be the reduction of N3/N4 and REM sleep stages commonly seen in these patients.

In addition to autoimmune encephalitis, parasomnias and DEB have been described in some other more common autoimmune diseases. Graves' disease, an *HLA-DQA1*-related autoimmune hyperthyroidism, was associated with sleepwalking in 2 studies, where all symptoms were resolved after thyrostatic treatments.[160,161] Guillain-Barre syndrome is an autoimmune disorder targeting primarily the myelin sheath. Varied sleep disturbances have been reported, with reduced sleep time and increased sleep fragmentation commonly seen in 2 hospital-based studies,[162,163] with some patients displaying RBD-like movements as well as status dissociatis.

Cerebrovascular events and neoplastic lesions of the brain

Although less common than apnea, insomnia, and hypersomnia in poststroke patients,[164] parasomnia have been reported in patients with brainstem stroke[25,165–174] and ischemic events of the thalamus.[22] A questionnaire-based study revealed that 11% of patients with ischemic stroke also experienced possible RBD.[175] Multiple possible sleep disorders can occur during the acute phase of ischemic stroke and require hospitalization, including RBD confirmed by polysomnography.[176] Overall, the most commonly reported lesion area in DEB occurs in the midpons. This is also the most commonly reported region of tumor-associated DEB.[165,166,177,178] Therefore, it is likely that associations between stroke/tumors and DEB are predominantly driven by focal damage in brainstem nuclei that regulate REM sleep (**Table 5**).

Pediatric neurology, hereditary diseases, and genetic mutations

In children, DEBs occur more frequently during NREM sleep than in REM sleep. This can be due to relative preponderance of N3 sleep in childhood. The prevalence of self-reported

Table 4—Prevalence of dream enactment behavior in neurological diseases based on video-polysomnography.

Disease	Subform	Ethnicity	Types of DEB	Frequency of DEB	Note	References
Movement disorder						
Amyotrophic lateral sclerosis			RBD	0–2.44%		80,81
Creutzfeldt–Jakob disease			RBD	14.3%		82
Huntington disease			RBD	0–12%		83–85
Pallidopontonigral degeneration			RBD	0%		86
Progressive supranuclear palsy			RBD	0–35%		87–89
Synucleinopathy	Dementia with Lewy bodies	Mixed	RBD	40–83%		90–97
				26.9–44.6%	Phenoconversion rate from iRBD: 43.5% Based on the 2019 multiple center study	
	Multiple system atrophy	Mixed	RBD	69.2–100% 3.08–21.4%	Pooled prevalence and 95% CI at 88% [79%,97%] Phenoconversion rate from iRBD: 4.55%	65,90,96–104
	Parkinson disease	Mixed	RBD	26.9–46.0% 4.3–69.4%	From iRBD Cohort 52.0% from the 2019 multiple center study	90,96,97,99,100, 105–111
Spinocerebellar ataxia	Machado–Joseph disease (type 3)		RBD	1.19%*	From iRBD Cohort	97
Dementia						
Alzheimer disease		Mixed	RBD	3.57–7.82%	From iRBD Cohort Probable Alzheimer disease (may or may not have Lewy body)	96,97,112–114
Autoimmune disorders						
Narcolepsy		Mixed	RBD	1–38.4%	Mixed of both types of narcolepsy	99,115–122
					Some patients may be on medication during the study	
			Night terror	62.5%		123,124
Autoimmune encephalitis			RBD	31.6%		125
Genetic disorders						
Down syndrome			RBD	0%		126
			Sleeptalking	42.6%		
Wilson disease			RBD	0–14.3%		127,128

DEB = dream enactment behavior, RBD = REM sleep behavior disorder.

parasomnias gradually decreases with age.[184] To date, only a few video-polysomnography studies have examined DEB in children, most often focusing on NREM sleep dream enactment. In RBD, several case series have been reported (**Table 6**). The most notable association is narcolepsy (as described earlier). Psychological distress and anxiety are also common inducers of DEB in both NREM and REM sleep (as described in the previous section).[184,186,187]

Among 57 children with Tourette syndrome, the prevalence of sleepwalking was reported at 18%, as opposed to 3.5% of those with learning disabilities and 1.8% of those with seizure.[188] A later study found more sleepwalking in participants with comorbid Tourette syndrome and attention deficit hyperactivity disorder.[189] An unusually high prevalence of sleeptalking has been reported in several other studies.[190–192] A later video-polysomnography study found that during sleep both tics and regular movements remain common in patients with Tourette syndrome,[193] although not clearly associated with severity of daytime tics.[194,195] Possible DEB-like movements may be associated with the increase in hyperarousal in this

Table 5—Lesions in patients with history of ischemia/hemorrhagic stroke, or brain tumor, aneurysm.

Lesion Location and Near-by Region	Type of DEB	Number of Subjects Identified	References
Medulla*	RBD, possible sleepwalking, possible NREM behavior disorder	5	165,167,171,179
Pons*	RBD, possible sleepwalking, possible NREM behavior disorder	20	25,165–174,178,180
Cerebellum*	RBD, night terror, sleepwalking	4	165,179,181,182
Midbrain	RBD	1	165
Thalamus	Night terror, sleep behavior disorder	2	22,183

*Some patients had lesions affecting multiple regions due to the position. NREM = non–rapid eye movement, RBD = REM sleep behavior disorder.

Table 6—Reports of pediatric dream enactment behavior associated with specific conditions.

Classification	Diseases/Disorders	DEB Type	References
Developmental disorder	Smith-Magenis syndrome, Type 1 Chiari, Moebius syndrome	Nightmare disorder, RBD	57,179
Cancer	Pilocytic astrocytoma, midline cerebellar astrocytoma	Nightmare disorder, RBD	57
Movement disorder	Tourette, juvenile Parkinson disease	Nightmare disorder, RBD	57,131,138
Autoimmune disease	Narcolepsy	Nightmare disorder, RBD	57
Other genetic mutation	Type 1 neurofibromatosis	Nightmare disorder, RBD	57
Other health events	Pituitary cyst, TBI, hyperthyroidism	Nightmare disorder, RBD	57,160

*Some cases have more than one comorbid disorders. DEB = dream enactment behavior, RBD = REM sleep behavior disorder, TBI = traumatic brain injury.

population.[196–198] An increase in movements during the REM sleep and even RBD has been reported.[138,196]

Another frequently noted health event associated with sleepwalking/sleep terror in children is migraine (estimated prevalence based on questionnaires ranged between 22 and 71%).[199–201] One hypothesis that may explain this phenomenon is the abnormal changes in serotonin level during slow wave sleep.[200,202] However, not all questionnaire studies found similar results. Rather than sleepwalking and DEB during the NREM sleep, migraine was linked to narcolepsy and insomnia in one report.[203] Further studies will still be needed to assess this association.

Parasomnia, apnea, and sleep-epilepsy are common challenges for many parents of children with developmental disorders.[204,205] In a questionnaire report of 147 children with intellectual disabilities, it was estimated that 2% were possible sleepwalkers and 6–16% had other possible DEB manifestations.[206] Neurodevelopmental disorders in which DEB has been studied include Down syndrome, Angelman syndrome,[207] cri du chat, cerebral palsy, Rett syndrome,[208] aspartylglucosaminuria,[209] autistic spectrum disorder, and several other genetic disorders.

Several questionnaire-based studies had been performed to investigate sleep disturbances in children with Down syndrome.[210–213] In an Edinburgh study,[211] children with Down syndrome had on average more parasomnia symptoms than shown in the normalized data of the healthy children around the same age, which is similar to the findings from several other European studies[210,212,213] and a Korean study.[214] Of the suggested symptoms of DEB, sleeptalking (30–57%) is the most commonly shared phenomenon across the studies. Sleepwalking was generally uncommon except in the Korean study (42%). In a video-polysomnographic study, 43% of adults with Down syndrome sleeptalked during NREM sleep. None showed overt RBD, although 6% had REM sleep without atonia.[126]

Several studies have indicated a possible increase in DEB events in children with cerebral palsy.[215–218] In an Italian study, children with cerebral palsy scored higher in parasomnia indices (9% screening positive) than typically developing children. Furthermore, when comparing within the disease group, they found that those with active epilepsy had even higher score on the Sleep Disturbance Scale for Children than those without (odds ratio of 14.0 with 95% confidence interval of [1.26–157]).[217] Another study of 100 children with cerebral palsy found that two types of DEB (sleeptalking and moving in a nightmare) seem to increase with age, which is the opposite in typically developing children. This was, however, not confirmed in the later studies.[218,219]

Rett syndrome is caused primarily by a spontaneous mutation in the *MECP2* on the X chromosome. Sleep disorders, including sleep apnea, nocturnal seizure, and parasomnia, were found as relatively common among children with Rett syndrome, in most studies.[208,220] However, DEB prevalence and features have not been described in video-polysomnography case series. In the large questionnaire-based Australia Rett Syndrome Database study, night laughing occurred in up to 68%; sleeptalking, 32%; night screaming, 46%; night terror, 40%; and sleepwalking, 7%.[221] It is unlikely that the DEB is induced by apnea, which appears rarely as a cause of DEB in children.[57]

Chiari malformation is a genetic disorder affecting primarily the cerebellum, lower pons, and medulla. In a Brazilian cohort, 23 patients (22%) with Chiari malformation (type I, 19%, and type II, 24%) had dream enactment episodes during REM sleep.[222] DEB during NREM sleep among children with Chiari malformation had also been reported in several studies.[179] However, since sleep apnea is common in patients with this disorder, it is unclear if some DEB is actually apnea-induced movement.[223]

Sanfilippo syndrome is a neurodegenerative disease caused by mutations in *SGSH, NAGLU, HGSNAT,* and *GNS* genes. Previous questionnaire-based studies revealed that possible DEB-like symptoms occur in 18%–38% of Sanfilippo patients.[224,225] In a polysomnography study, 3/6 children revealed histories of possible DEB.[226] However, many children were taking benzodiazepines, and based on the available polysomnography results and medical history, it may be that the apparent DEB were actually the common posthyperarousal responses seen in children (note that 3 did not have slow wave sleep).

Possible DEB events were also reported in children with cri du chat disorder,[227] tuberous sclerosis complex,[228,229] neuronal ceroid lipofuscinosis,[230] Smith-Magenis syndrome[229] and fragile X syndrome.[231] Yet the descriptions suggest a differential diagnosis, including epilepsy, apnea, or circadian rhythm disorder; therefore, it remains unclear if there is any clear association of these syndromes with DEB.[232–236]

Finally, there have been a few reports of video polysomnography–confirmed DEB, mostly RBD, in adults with genetic disorders. These include autosomal dominant leukodystrophy,[237,238] Niemann-Pick disease type C,[239] fatal familial thalamic degeneration,[183] spinocerebellar ataxia (types 2, 3, 31),[240–243] Wilson disease (12.5%, n = 40).[127] The few cases described make it impossible to estimate the prevalence of DEB.

With regard to mechanism, several genes have been linked with DEB, including:

1. NREM sleep: *HLA-DQB1*,[244,245] *GLRA1*,[246] autosomal dominant trait of genetic loci at chromosome 20q12-q13.12,[247] and TFAP2B mutation[248]
2. REM sleep: Narcolepsy: HLA-DQB1,[249] Idiopathic: GBA,[250,251] PARK2,[252] PINK1, SCARB2,[253] MAPT,[253,254] TMEM175 p.M393T,[255] C9orf72[256,]

In the case of overt RBD, most genes found to date are associated with neurodegenerative synucleinopathy. On the other hand, genes associated with NREM parasomnias are more diverse. The one gene seemingly "shared" by the two types of DEB is *HLA-DQB1*. *DQB1**05:01 allele was associated with both sleepwalkers and sleep terror,[244,245,257] and *DQB1**06:02 allele[258] to narcolepsy type 1. Interestingly, the two alleles seem to have an opposite direction of association between sleepwalking and narcolepsy type 1. *DQB1**06:02 was slightly less common among sleepwalkers. However, the *DQB1**05:01 allele was associated with a lower risk of narcolepsy type 1 in the presence of DQB*1**06:02 allele.[146] Since *HLA-DQA1* and *HLA-DQB1* each encode the α and the β chains of the same surface antigen, one may consider cells expressing HLA-DQ to be potentially associated with DEB. Against this potential cause of the genetic propensity to DEB is the low expression of *HLA-DQ* in the brain.[259]

Dream enactment behavior due to medication and substances

Behaviors consistent with sleepwalking, confusional arousals, and DEB have been related to alcohol and substance use/withdrawal have all been described in numerous case reports. Dream enactment behavior associated with medication may be due to a reversible side effect of the medication; alternatively, the medication may "unmask" an inherent propensity to DEB. A compilation of the studies is in **Table 7**.

The most well-described relationship is with antidepressants, with specific reports for fluoxetine,[263,264] venlafaxine,[265] and paroxetine,[266] and evidence of a broad link between numerous types of antidepressants and REM sleep without atonia.[267,268] This may occur especially in younger participants with RBD.[51] In polysomnography studies, onset may be gradual; sertraline gradually increases REM sleep without atonia when measured over serial nights in patients with depression.[269] Although the serotonergic antidepressants dominate the literature, other antidepressants have been described. This includes clomipramine,[270–273] phenelzine,[274] selegiline,[275] amitriptyline, nortriptyline, imipramine, desipramine, protriptyline, trimipramine,[263] mirtazapine,[276] nortriptyline,[277] and imipramine.[277]

The hypnotic suvorexant[278] has also been reported to cause RBD in Parkinson disease. Opioids (oxycodone and morphine) have been associated with the onset of dream enactment behavior in patients with cancer.[279] Beta-blockers have also been reported to induce RBD[280]; since propranolol has been proposed for treating posttraumatic stress disorder, future studies will be needed to assess the possible adverse effect of this medication.[66,281]

The mechanism for the loss of REM sleep without atonia with antidepressants remains unconfirmed. The mechanisms may be distinct from those seen in degenerative neurologic disorders and may vary according to medication.[268] The ventral portion of the sublaterodorsal nucleus project to the spine and result in the motor inhibition that is most pronounced in REM sleep.[282,283] Serotonergic activity in the descending pathways progressively decreases from waking, to NREM sleep, to REM sleep in parallel with observed atonia. Cholinergic, glutaminergic, and glycinergic neurons actively inhibit the postsynaptic lower motor neurons in REM sleep; this includes both descending systems interacting with the brainstem and direct synapses between the serotonergic system and the spinal interneurons. It has been proposed that in humans with REM sleep without atonia the selective serotonin reuptake inhibitor and similar medications facilitate motor disinhibition through the serotonergic system and silencing of the subcoeruleus nucleus, while tricyclic antidepressant medications interrupt the atonia through the parallel anticholinergic release of the glutaminergic-glycinergic system.[268]

Several types of medications have been reported to induce parasomnias more typical of sleepwalking than true dream enactment. The cases of sleepwalking and confusional arousals

Table 7—Substance-induced or withdrawal symptoms related dream enactment behaviors.

Drug or Compound	Names of Drugs	Types of DEB	Accumulated Subjects
Drug-induced DEB			
Antiepileptic drug	Topiramate	Sleepwalking	2
Hypnotic	Choral hydrate	Sleepwalking	2
	Methaqualone	Sleepwalking	3
	Sodium oxybate	Sleepwalking	3
	Suvorexant	Sleepwalking	1
Z-Drug	Zaleplon	Sleepwalking	2
	Zolpidem	Sleepwalking	57
	Zopiclone	Sleepwalking	1
Aminoketone antidepressant	Bupropion	Sleepwalking	2
Monoamine oxidase inhibitor	Phenelzine	RBD	7
Selective serotonin reuptake inhibitor	Citalopram	RBD	1
	Fluoxetine	RBD, sleepwalking	7
	Paroxetine	RBD, sleepwalking, night terror	3
	Sertraline	RBD, Sleepwalking	1
	Venlafaxine	RBD	1
Serotonin-norepinephrine reuptake inhibitor	Mirtazapine	RBD, Sleepwalking	5
	Reboxetine	Sleepwalking	1
Serotonin receptor agonist	Tandospirone	RBD	1
Tricyclic antidepressant	Amitriptyline	Sleepwalking	1
	Clomipramine	RBD, possible night terror	11
	Imipramine	RBD	2
	Nortriptyline	RBD	1
Lithium		Sleepwalking	39
Antipsychotic and atypical antipsychotic medication	Chlorprothixene	Sleepwalking	1
	Olanzapine	Sleepwalking	3
	Perphenazine	Sleepwalking	2
	Quetiapine	Sleepwalking	8
	Thioridazine	Sleepwalking	2
	Ziprasidone	Sleepwalking	1
Beta-blockers	Bisoprolol	RBD	2
	Metoprolol	Sleepwalking	2
	Propranolol	RBD, sleepwalking	9
Stimulant	Methylphenidate	Sleepwalking	1
Dopamine agonist	Bromocriptine	Sleepwalking	1
	Lisuride	Sleepwalking	1
Leukotriene receptor antagonist	Montelukast	Sleepwalking	1
Antibiotics	Ciprofloxacin	Sleepwalking	1
Other	Alcohol	RBD	6
	Chocolate	RBD, sleepwalking	1
	Coffee	RBD, sleepwalking	1
DEB related to withdrawal symptoms			
	Barbiturates	RBD/RSWA	NA*
	Meprobamate	RBD/RSWA	NA*
	Nitrazepam	RBD/RSWA	1
	Pentazocine	RBD/RSWA	1
	Phenelzine	RBD/RSWA	NA*
Alcohol		RBD/RSWA	10

*Unable to determine numbers of subjects affected due to inability to achieve an original article or the original article was not written in English. Modified and combined based on the information from previous review articles[260,261] and publications from the international RBD study group.[262] DEB = dream enactment behavior, RBD = REM sleep behavior disorder, RSWA = REM sleep without atonia.

have most commonly related to "z-type" medications (cyclopyrrolones and imidazopyridine zolpidem) as well as lithium (**Table 7**).[260,261,284] Of the z-type hypnotics, zolpidem has the most case reports of sleepwalking. Lithium, by itself, had been linked with sleepwalking in 39 cases. Sleepwalking had also been linked with several antipsychotic medications, especially quetiapine. It is likely that the mechanism of this link is a global increase in sleep drive, resulting in only partial arousals to external stimuli that might otherwise wake the individual.

Both substance use and withdrawal may alter sleep and thereby induce dream enactment behavior. Identifying specific changes can be difficult due to the difference in the quantity, frequency, and total time of exposure. Heavy alcohol usage, along with tobacco, is a relatively consistent risk factor for idiopathic RBD across most studies.[40] On the other hand, for sleepwalking, in the ICSD-3, the link is sufficiently strong that alcohol intoxication is an exclusion criteria for diagnosing sleepwalking.[285] This makes it relatively difficult to perform a systematic study of relationships between sleepwalking and alcohol use; future work will be needed to establish a workable model for the influence of alcohol on parasomnia.[286]

To our knowledge, there is no known link between dream enactment behavior and isolated cannabis use. Cannabidiol usage have been reported to suppress RBD in a case series of patients with Parkinson disease.[287] This may be due to reduction in REM sleep (found in a small study).[288] However, the effect may be dosage-dependent like alcohol, as shown in previous studies.[289] Similarly, a few studies had also suggested that cannabis products may help reduce PTSD-related nightmares.[289] More studies will be needed to evaluate the usage of cannabis products for DEB since some studies have indicated high prevalence of "rebound" dreaming issues after the withdrawal.[290]

Sleep-related violence and dream enactment behavior

The movements made in sleep by most sleepers with parasomnias are usually brief and benign. Yet some movements may be violent such as thrashing, punching and kicking; with the presence of another person sleeping in proximity or intervening with the sleeper, choking and a headlock maneuver have been documented in detailed reviews of sleepwalking and of RBD.[94,291] Aggressive behavior with recall of threatening mental imagery that may be interpreted as a dream may also follow night terrors.[292] Once the sleeper is wandering out of bed, the final diagnosis is sleepwalking the great majority of time.[293] The disorders that may cause sleep-related violence and injury include a wide range of parasomnias, other sleep disorders and malingering.[294,295] The most common final diagnoses are sleep terrors or sleepwalking, RBD, obstructive sleep apnea, dissociative disorder, and sleep epilepsy.[294] Although this may be a behavior seen in the majority of patients with parasomnias attending sleep clinics.[50,297,298] Sleep-related violence appears to be occurring in approximately 1-2% of adults when the population outside of sleep clinics is systematically surveyed and is often associated with dream imagery.[299]

Alcohol, hypnotics and other medications, sleep deprivation, recent life stress, and provocation of arousal or a defense reaction during sleep have been factors cited in contributing to more violent acts. The basic management requires diagnosis, as well as education of the patient and bed-partner. Avoidance of sleeping in proximity to others, a safe bed environment including the purging of any possible weapons from the sleeping environment, reducing life stressors and avoiding sleep disruption all generally can be recommended. Bed partners and others who may encounter the agitated sleeper should avoid confrontation and physical contact. Judicious medications have been beneficial once a diagnosis is established.[64] A bed alarm has reduced injury in patients with recurrent violent episodes due to RBD.[300]

DISCUSSION

Limitations of this review

Several limitations should be noted in documenting links between DEB and other conditions/medications. First, clear documentation of DEB under video-polysomnography is mostly limited to RBD. In practice, polysomnography has been used primarily in diagnosis for sleep-related breathing disorders; any lack of clinical awareness of DEB and sleep-related movement disorders in general may limit prevalence estimates of DEB. Other possible limitations are confounders that mimic DEB (ie, DEB induced by arousal, lack of sleep, apnea, circadian disruption, and use of medication) that may have been counted as dream enactment in questionnaire-based studies.[301] This can be especially problematic primarily in case reports, as not all articles clearly specify the exclusion criteria when reporting DEB. Therefore, we suggest that all future DEB studies state whether they have excluded potential differential diagnoses. Another limitation was the nonuniform terms employed across the many fields that study DEB (eg, pulmonology, neurology, psychiatry). For this review, we defined our terms based on the referenced works although some research groups may have defined DEB differently where unspecified.

Management of recurrent dream enactment behavior

The most important clinical step in evaluating DEB is describing in detail the events, with attention to any past or potential harm that may have been done to the patient or any bed partner. The intensity and frequency are also important to evaluate with respect to distress and any disturbance the DEB may cause. The characteristics of the sleeping environment are very important when potential harm is considered, such as surfaces that may cause injury with major body movements like swinging arms or kicking legs. Falls may occur and should be documented; safety-oriented flooring is recommended if there remains a significant risk of falling. Blows to a bed partner need to be documented with a view to prevent future injuries.

DEB in otherwise normal participants are described as infrequent isolated episodes. These may include speech, twitches beyond a few fingers, kicks, and body jerks with little sustained or directed movement. Physical signs of

sexual arousal may also be noted, which are considered normal REM-sleep physiology.[302] If no potentially dangerous activity is described, no more intervention beyond education and reassurance is necessary. When injurious or potentially dangerous behavior is described, education to promote a safe sleeping environment is necessary to avoid future injury. This should be tailored to the individual and their environment, but include education of the bed partner, removal of dangerous objects from the sleeping area, and changes to the bedroom to avoid falls.[303]

Non-pharmacological therapy should be considered after a DEB diagnosis. Psychotherapy may be useful for addressing prior trauma, eliminating antidepressant medication or for treating ongoing mental illness.[304] In addition, psychotherapy may address the potential anxiety elicited by a sleep diagnosis itself.[304] However, psychotherapy is a lengthier form of treatment compared to pharmacological therapy and, therefore, potentially may limit both access to therapy and treatment adherence.

If associated nightmares contribute to DEB, then management of the nightmares or associated sleep disorder should be initiated.[305–307] If an associated neurological or sleep disorder is suspected or diagnosed, such as RBD, then treatment of the disorder should be initiated without undue delay and treatment response followed. As there are dedicated clinical guides and summaries for treatment of nightmares, PTSD and RBD, a brief summary follows.

Most high-level evidence in the treatment of nightmares comes from randomized trials of participants with PTSD[79,305,308–313] The psychological approach with the best evidence for an important treatment effect is imagery rehearsal therapy (IRT), which may be combined with formal cognitive behavioral therapy for insomnia (CBT-I). Variants of IRT have shown beneficial effect, such as exposure, relaxation, and rescripting therapy (ERRT). Often SSRI therapy is initiated for the anxiety or depressive symptoms but are often poorly tolerated and can exacerbate DEB. The only medication with consistent high-level controlled evidence of important effect on nightmare reduction in this population was nightly prazosin.[79,305,308] Considering immediate side effects and serious adverse effects of long-term use of prazosin, nonpharmacologic therapy is preferred when possible.[79,312] A short term adjunctive pharmacologic therapy with propranolol administered before each of 6 weekly brief memory reactivation sessions showed improved clinician and self-rated scores of PTSD, which both include troublesome dreams.[281]

In the case of confirmed or probable REM sleep behavior disorder every effort to diagnose an underlying cause should be made, and modifications to the sleeping environment should be prescribed with a view to preventing injuries and distress.[303] This considers the recurrent and at times injurious nature of the DEB seen in RBD that improves but rarely resolves with pharmacotherapy. The best evidence for medication remains from case series and small trials for nightly clonazepam or melatonin. Clonazepam nightly as primary pharmacotherapy is based upon more than 25 years of anecdotal observation documenting important changes before and after clonazepam in DEB occurrences according to published series.[314] For melatonin, a small trial of 8 participants with mixed causes of RBD found that clinician global ratings improved.[315] But two more recent placebo-controlled trials have demonstrated no significant improvement. In a trial of patients with iRBD, no significant global improvement was noted, without significant change in secondary outcomes on questionnaires.[316] Another randomized parallel group placebo-controlled trial of patients with Parkinson disease and RBD found no significant improvement in number of RBD events noted on participant diaries as well as the number of injuries.[317] A placebo-controlled 4-week trial of 0.5 mg of nightly clonazepam in 40 patients with RBD showed no significant differences in the primary outcome of the global rating scale as well as secondary outcomes.[318] An international consensus statement has been published to guide the design of future trials in this field.[319]

Future directions

Video-polysomnography provides a great complexity of information and will continue to be the gold standard for diagnosing sleep disorders. However, since video-polysomnography is costly and very time-consuming, more accessible tools are needed. Potential tools include portable polysomnography, smart wearable devices,[320] video analytics tools,[321] machine learning algorithms,[322,323] and questionnaires.[324–326] In the case of portable polysomnography and machine-learning algorithms, they each provide unique perks in speeding up the time needed for diagnosis. Of the available sleep questionnaires, those for adults have been better developed than those for preadolescents and children. Although wearable tools and questionnaires may potentially be more efficient triage procedures, more work will still be needed to document reliability. Wearable devices are more cost-efficient and accessible, but future research will have to address several of their limitations, such as low accuracy, poor standardization, and low within/between device reliability before they can even be considered as diagnostic or screening tools.[327]

ABBREVIATIONS

AD, Alzheimer disease
DEB, dream enactment behavior
NREM, non–rapid eye movement
PD, Parkinson disease
PTSD, posttraumatic stress disorder
RBD, REM sleep behavior disorder
REM, rapid eye movement

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