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Parasomnia in children and adults as a differential diagnosis to non-lesional focal epilepsy—English version

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Abstract

Parasomnia is defined as striking behavior during sleep. The waking up disorders of pavor nocturnus and sleepwalking occur out of NREM sleep and the event usually occurs in the first half of the night. Nightmares and REM sleep behavior disorders occur more frequently in the second half of the night with an increase in REM sleep. All parasomnias must be differentiated from sleep-related epileptic seizures. For this purpose, video EEG documentation of nocturnal behavioral disturbances is the gold standard.

Keywords
NREM parasomnia · REM parasomnia · Sleep related hypermotor epilepsy (SHE) · Arousal disorder · Pavor nocturnus

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Parasomnias are defined as abnormal or noticeable behaviors during sleep or at the threshold between wakefulness and sleep. All parasomnia disorders are listed in the International Classification of Sleep Disorders (ICDS-3). A distinction is made between non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep parasomnias, with NREM parasomnias including sleepwalking, confusional arousal, and night terrors, and REM parasomnias including REM sleep behavior disorder, nightmare disorder, and isolated sleep paralysis [1]. Parasomnias are expressions of unusual or pathological central nervous activation during sleep or during the sleep–wake transition. In some cases, complex behavioral abnormalities that are associated with activation of the autonomic nervous system occur, especially in non-REM parasomnia. At the same time, consciousness is limited or completely absent [2, 3]. Parasomnias are common, especially in childhood, and can be observed in various forms in approximately 30% of children [4, 5]. However, they can also occur in adulthood or persist into adulthood and are then perceived as particularly unpleasant. Approximately 5% of adults report suffering from nightmares; sleepwalking and pavor nocturnus are rare in adults (< 1%; [6, 7]). From a semiological perspective, parasomnias can be mistaken for other seizure-like disorders during sleep, especially nocturnal epileptic seizures [2, 3].

The basics of sleep

Sleep stages were first described by Rechtsschaffen and Kales in 1968 and simplified in 2007 by the American Academy of Sleep Medicine (AASM) while defining criteria [1]. A sleep cycle is defined as the progression through the different sleep stages. According to the AASM criteria of 2007, five different sleep stages including a waking state are defined from the age of 2–3 months (**Fig. 1**):

- Waking state
- N1 sleep stage—transition between waking and sleep (non-REM [NREM])
- N2 sleep stage—stable sleep (non-REM)

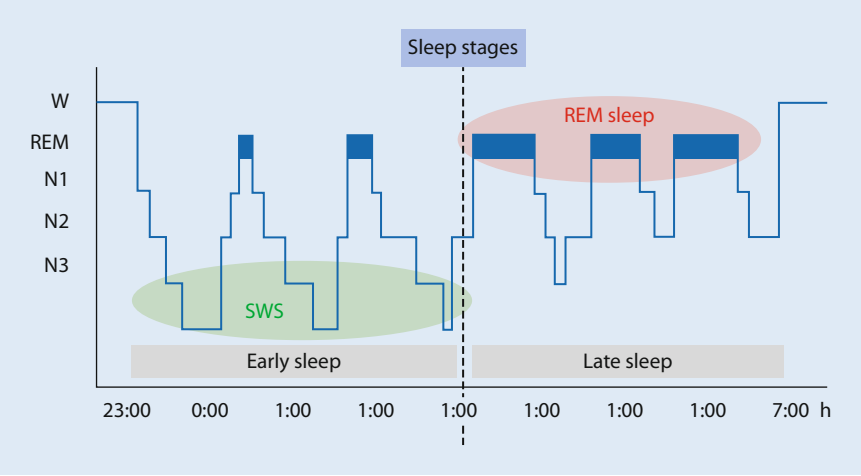

Fig. 1 ▲ Human sleep stages. *W* waking state, *REM* REM sleep, *N1–N3* superficial sleep, *SWS* slow-wave sleep. (Modified from [8])

- N3 sleep stage—deep sleep (non-REM)
- Rapid eye movement (REM) sleep, dream sleep

The sequence of the individual sleep stages results in the sleep profile ("hypnogram") within a sleep cycle, which a healthy sleeper goes through four to seven times per night, depending on sleep duration and interindividual variance. Superficial sleep (N1) occurs first when falling asleep, followed by N2 and N3 in rapid succession. Lastly, REM sleep can be observed, thereby completing a sleep cycle [2].

The sleep stages are distributed in a characteristic way over the sleep period and change with age. The sleep cycle in infants is very short (approximately 50–60 min); only from school age onward is the sleep architecture comparable to that of adults, with a cycle duration of approximately 90–110 min [2, 9]. The proportion of sleep stages N1 and N2 in the total sleep of a healthy, approximately 30-year-old sleeping person is approximately 55–60%. The share of deep sleep N3 is approximately 15–25%, while dream or REM sleep usually comprises 20–25% of the total sleep duration in adulthood. Over the course of a night, the proportion of deep sleep (N3) continuously decreases and the proportion of REM sleep continuously increases with the number of sleep cycles completed within a sleep period [2]. During physiological REM sleep, a healthy person experiences REM sleep atonia, which means that dreams cannot be physically acted out [10].

The first half of the night contains more deep-sleep components, thereby explaining the frequent occurrence of NREM parasomnias in this sleep phase, e.g., pavor nocturnus or sleepwalking, in the first hours after falling asleep. By contrast, more REM parasomnias are usually observed in the second half of the night, and nightmares therefore occur more frequently toward morning [2].

The proportion of individual sleep stages shows age-dependent variations. REM sleep decreases significantly over the entire lifespan. The share of REM phases in newborns is considerable at approximately 50%, going down to 24% from the age of 20 years and decreasing even further with age. Deep sleep (N3) peaks in young adults and, like dream sleep, declines steadily in favor of the other sleep stages with age [2].

Abbreviations

ADNFLE	Autosomal dominant nocturnal frontal lobe epilepsy
EEG	Electroencephalogram
NREM	Non-rapid eye movement
RBD	REM-sleep behavior disorder
REM	Rapid eye movement
SHE	Sleep-related hypermotor epilepsy

NREM and REM parasomnias

NREM parasomnias

Arousal disorders

Arousal disorders include confusional arousal, pavor nocturnus, and somnambulism. Arousal reactions usually occur from sleep stage N3, but can also occur from sleep stage N2 [1, 12, 13]. Full awakening is not always observed afterwards [14]. Common to all is a dissociated behavioral state of the brain. Here, a mapping of activated and deactivated brain areas and networks to clinical behavioral symptoms can be made.

Activation of the amygda temporo insular areas, disconnected from the prefrontal cortex, leads to emotional activation (e.g., fear), while deactivation of the hippocampal and frontal control cortex leads to amnesia for the event [15]. There is an abrupt arousal response (electroencephalography [EEG]: alpha and beta activity) in the motor cortex and limbic system of the cingulate gyrus with concurrent sustained NREM sleep stage N3 (EEG: delta activity) in the frontoparietal association cortex [16]. Pathophysiologically, there is dissociated activation of thalamocingulate tracts with further deactivation of the thalamocortical arousal system. The cause is suggested to be instability of NREM sleep or a possible mixture between NREM and REM sleep [17, 18]. Thus, there is a dissociation of wakefulness and sleep, which can also be referred to as "localized sleep." While cortical areas remain in deep sleep, other areas of the brain are active [19].

The prevalence of NREM parasomnias in childhood is 6–15% (confusional arousal 17%, sleep terror 6%, sleepwalking 15% one time/week, 1–6% one to four times/week). There is familial clustering, with a 10-fold increased risk of later sleepwalking in first-degree relatives [20, 21].

Arousal disorders typically begin with an abrupt startle (rubbing eyes, sitting up, looking around one's surroundings, etc.). Hypermotor withdrawal then begins, as well as a potentially agitated state with crying/sobbing during physical and verbal interaction with one's surroundings. Affected individuals often exhibit tachycardia, tachypnea, and sweating. Symptoms vary within the event and on recurrence.

For all forms, there is at least partial, usually complete, retrograde amnesia. All forms of arousal disorder must be differentiated from each other and distinguished from sleep-related nocturnal focal seizures [3, 15, 22].

Confusional arousal [12, 23]. This form of NREM parasomnia occurs in childhood with a peak between the ages of 2 and 10 years. Here, during the attacks, which often occur in the first half of the night, the children speak unintelligible words, mumble, or whine for up to 10 min (up to < 1 min) after partial awakening; only rarely does an attack last 10–30 min. The children do not leave their bed during this time, cannot be consoled by their relatives, and have retrograde amnesia for the event [15].

Pavor nocturnus [23]. *Synonym:* sleep terror, night terrors, night fear, abrupt awakening from nighttime sleep, nightmare, incubus, massive autonomic arousal.

Approximately 1–2 h after falling asleep, an abrupt awakening from nighttime NREM sleep occurs with a pronounced fear reaction (involving a loud, terrified scream, undirected fear up to a panic reaction). The child does not recall a dream experience in this case [2]. They do not perceive their surroundings, do not recognize their own parents, and reject them. Their eyes are open, their facial expression conveys fear, anger, and confusion, and they cannot be woken. The fear reaction usually causes flushing of the face and sweating, and tachycardia or tachypnea is possible. The duration of the event is reported to be 5–15 min. The child typically falls asleep again abruptly and suddenly and cannot remember the episode the next morning (amnesia).

The peak of occurrence has previously been described to be between 4 and 7 years [7]. Although previous literature states that approximately 17% of children are affected at this age, the analysis of children aged 1.5 years by Petit et al. showed a higher incidence rate of 34.4% of all children, particularly at this early age [9]. By contrast, the phenomenon is usually observed in adults only in < 1% of cases (maximum 2.3–2.6%; [2, 3]). For adults, even if the occurrence is rare, they are subject to a high level of distress due to the fear that such an attack may also occur during overnight stays away from home [7]. In 30–50% of cases, sleepwalking may occur after the event, and the two disorders are etiologically very closely related [7]. The sharp decrease in the frequency of pavor nocturnus attacks from the age of 4 years onward suggests that brain maturation processes play a role in their development. However, the exact mechanism is not yet clear. According to previous scientific work, one can assume a predisposition–stress model. Recent evidence suggests a close association with somnambulism in particular, supporting the theory of a common genetic basis [11].

Diagnostic criteria of pavor nocturnus according to AASM/ICSD-3 2014 [1].

- Episodes of sudden awakening from sleep, usually preceded by a loud sob or a loud cry. Awakening is accompanied by a strong vegetative response, and the affected person exhibits behavior indicative of a marked state of anxiety.
- At least one of the following other symptoms occurs:
  - Difficulty awakening the affected person
  - Confusion after successful awakening
  - Complete or partial amnesia regarding the episode
  - Dangerous or potentially dangerous behavior during the event
- The disorder cannot be better explained by another sleep disorder, internal or neurological disease, mental illness, or medication or substance abuse.

Somnambulism. *Synonym*: sleepwalking, seemingly purposeful automatisms, nonepileptic amnesia.

This form of arousal disorder occurs mainly in primary school age with an age peak from the fourth to the seventh year of life; the phenomenon is observed much less frequently after puberty. According to a recent study by Petit et al., approximately 13.4% of all children aged 10 years exhibit this phenomenon [9]. About 30% of all children have sleepwalked at least once in their lives, 3–6% more than once, while 30–50% of patients with pavor nocturnus later present with sleepwalking [9]. With a history of sleepwalking in one parent, the prevalence increases from 22.5 to 47.5% for children with one affected parent, and to 61.4% in the case of two affected parents [9]. The prevalence of sleepwalking is estimated to be < 1% (maximum 4%) for adults [2, 3, 7], with the male sex predominating from adolescence [2]. The level of suffering experienced by the small number of adults is high, as they perceive uncontrollable nocturnal activities to be unpleasant [7, 24]. According to recent findings, there is a close association with pavor nocturnus in particular, supporting the theory of a common genetic basis [11].

The activity mostly occurs in the middle of the night and/or toward the end of the night [7, 23]. For the duration of typically seconds to a few minutes, the patient stands up quietly and wanders about, exhibiting automated actions (e.g., getting dressed, walking to the living room, opening windows, among other activities). Their eyes are open during this process, but the individuals are not in full possession of their mental faculties, nor can they remember what happened when they return to bed afterward and go back to sleep. Their responsiveness to external stimuli, e.g., being spoken to, is reduced [7].

Diagnostic criteria of sleepwalking according to the AASM/ICSD-3 2014 [1]. The changes or behaviors occur during sleep.

- The persistence of sleep or the state of unrestricted consciousness or judgment during ambulation or behavior is documented by at least one of the following factors:
  - Difficulty awakening the affected person
  - Confusion after successful awakening
  - Complete or partial amnesia regarding the episode
  - Performing automated behavior at an inappropriate time
  - Inappropriate or nonsensical behavior
  - Dangerous or potentially dangerous behavior
- The disorder cannot be better explained by another sleep disorder, internal or neurological disease, men-

tal illness, or medication or substance abuse.

REM parasomnias

All forms of REM parasomnias have in common that they occur predominantly in the second half of the night and toward the earlier morning hours during REM sleep. This form of parasomnias must be distinguished from NREM parasomnias on the one hand, and from epileptic nocturnal (sleep-related) focal seizures on the other [3, 24].

Nightmares

Synonym: anxiety dreams, nightmare, anxiety dream attack, REM nightmare.

Nightmares are a universal phenomenon; almost everyone remembers their nightmares, with the lifetime prevalence being almost 100% [3]; 5% of children and 2–8% of adults regularly have a nightmare once a week [2, 3]; 50% of children and adults report experiencing occasional nightmares. Females are more commonly affected than males. The frequency decreases with age in cross-sectional studies; therefore, young adults are the most likely to report nightmares [2, 7]. They are most commonly observed between the ages of 3 and 10 years, with peak occurrence between 6 and 10 years of age [7]. Again, this is thought to be due to a predisposition–stress model [2, 7]. REM nightmares are negatively toned dreams that lead to awakening [7]. They are associated with signs of intense fear, anxiety, or a sense of impending danger, but there is no panic [2]. The typical content in children and adolescents is 50% persecution, 20% own death or injury, 15% death or injury of others, 10% falling into a bottomless pit [7]. Typically, the person wakes up, children cry and call for parents, are immediately aware of their parents and want to be comforted. The person usually continues to sleep after a delay. Nightmares are very well remembered, as patients wake up from the strongly emotional dream [2, 7].

Diagnostic criteria of nightmare according to the AASM/ICSD-3 2014 [1].

- Recurrent episodes of awakening from sleep recalling disturbing dream content, usually associated with fear and anxiety, but also with anger, sadness, disgust, or other unpleasant emotions.
- Complete awakening with only minor confusion or disorientation, dream recall is immediate and clear.
- At least one of the following associated factors are present:
  - Delayed return to sleep after the event
  - Occurrence of episodes in the second half of the night

REM sleep behavior disorder (RBD)

Synonym: abnormal dream behavior, acting out dreams, motor REM parasomnia, REM sleep parasomnia, REM sleep without atonia, oneirism.

The term "REM sleep behavior disorder" (RBD) refers to motor activity due to suspension of the muscle atonia common to REM sleep. Typically, the outgoing nerve signals from the motor cortex in the brainstem are strongly inhibited to prevent movement along with the dream images. Due to disinhibition, complex pathological motor activity occurs in the context of REM dreams. The almost always aggressive dream content is excessively acted out with motor activity during REM sleep, e.g., leg movements occur as in cycling or playing soccer [2, 10, 25].

Approximately 80–90% of those affected are over 60 years of age, 90% of whom are male [3, 7]. There is a low overall prevalence of 0.38%, going up to 0.5% in the older population [2]. It is strongly associated with neurodegenerative diseases, such as Parkinson's disease. Therefore, it is thought to be a particular and early symptom complex of a neurodegenerative disease characterized by decline in the area of the brainstem that inhibits muscle tone [7]. The frequency of occurrence is reported to be several times per week. The episodes occur in the last third of the night, i.e., the second half of the night [3].

Diagnostic criteria of REM sleep behavior disorder according to ICSD-3 2014 [1].

A. The patient suffers from aggressive or self-injurious behaviors during sleep.
B. Limb or body movements are linked to experiences in a dream.
C. At least one of the following features occurs:
   - Injurious or potentially injurious sleep-related behavior.
   - Dreams appear to be acted out.
   - The behaviors during sleep disrupt sleep continuity.
D. Polysomnographic studies reveal at least one of the following electrophysiological features during REM sleep:
   - Pronounced increase in chin EMG tone
   - Marked phasic chin or limb EMG activity, independent of tonic chin EMG activity and one or more of the following clinical features during REM sleep:
     - Pronounced limb or body movements
     - Complex, dangerous, or aggressive behaviors
     - Absence of epilepsy-like activity associated with the events
E. Symptoms are not associated with a mental disorder, but may be associated with neurological disorders.
F. Other sleep disorders (e.g., pavor nocturnus or sleepwalking) may occur but are not the cause of the behavior.

Criteria B and C represent minimum criteria.

Differential diagnosis of non-lesional focal epilepsy

Nocturnal behavior problems affect approximately 30% of children and 4% of all adults [6, 26]. The occurrence of nocturnal seizures (7–30% of all seizures) is common, especially in focal epilepsy; therefore, the differentiation of sleep-related epileptic seizures from parasomnias is significant in clinical practice [27]. From a patient history perspective, nocturnal seizure-like events are often difficult to classify since detailed descriptions of these events are often not available and the patient's description of the events is rarely usable. Accurate diagnosis is essential to avoid treatment failure. A detailed history and clinical neurological examination should be supplemented by a sleep log and video

Table 1 Differentiation of sleep-related epileptic seizures from parasomnias

	Autosomal dominant nocturnal frontal lobe epilepsy (ADNFLE)	Sleep-related epileptic seizures in specific focal epilepsy of childhood	NREM parasomnias	REM parasomnias
Age of manifestation	Age-independent	Age-dependent	Age-dependent, especially childhood	Age-dependent
Family history	< 40%	Familial occurrence	69–90%	Negative
Time of occurrence during the night	Anytime, not assigned to a specific time	Especially after falling asleep and when waking up in NREM sleep	Especially first third of the night (NREM sleep)	Last third of the night (REM sleep)
Semiology	Stereotype, same sequence, signs of lateralization	Characteristic seizures for each type of epilepsy	Variable	Variable
Amnesia	In most cases	Rare	In most cases	Dream content is mostly remembered
Duration	Seconds to 1–2 min	Minutes	1–30 Min	Short
Reorientation	Rapid to immediate reorientation	Rapid	Delayed	Rapid reorientation
Trigger	Sleep deprivation	Sleep deprivation	Sleep deprivation, noise, stress	Tiring day

REM rapid eye movement, *NREM* non-REM

recordings of the event. To clarify nocturnal phenomena suggestive of seizures, performing electroencephalography (EEG) alone while awake is inconclusive. Video recording by relatives is helpful for further differentiation; the gold standard for diagnosis is video-EEG recording of typical attacks [6, 28, 29], as these are most likely to be obtained during long-term video-EEG monitoring. In patients with persistent unclear behavioral abnormalities during sleep, close collaboration between epileptology and sleep medicine is essential.

As a general rule, parasomnias can be distinguished from epileptic seizures in that the events are observed at different times. Whereas NREM parasomnias of infancy tend to occur in the first half of the night, REM behavioral disorders are predominantly recorded in the second half as REM sleep increases [14]. The frequency of occurrence of nocturnal epileptic seizures is significantly higher (between one and ten epileptic seizures per night vs. between one and two parasomnias per night), and motor patterns in parasomnia are variable, whereas in epileptic seizures, by contrast, they are stereotypical [6, 29, 30]. Another helpful method of differentiation based on patient history is by indicating the duration of the event; epileptic frontal lobe seizures are short (less than 1 min), while parasomnias last longer (sometimes several minutes). Reorientation in parasomnia, e.g., after waking the affected person, is slower and often characterized by a phase of confusion and perplexity, whereas reorientation in nocturnal epileptic seizures is typically very rapid to immediate, especially if they are of frontal origin ([14]; ◘ **Table 1**).

Sleep-related hypermotor epilepsy (SHE) has been chosen as an umbrella term for sleep-related nocturnal epileptic seizures of genetic and structural etiology [30]. Indications for the presence of autosomal dominant nocturnal frontal lobe epilepsy (ADNFLE) in adulthood include a family history and seizure semiology typical of this form ([4, 28, 31–34]; ◘ **Infobox 1**). The first monogenetically inherited epilepsy identified in 1995 was ADNFLE resulting from a mutation in nicotinic acetylcholine receptor subunit A4 (CHRNA4). Since then, other mutations of the acetylcholine receptor (CHRNA2, CHRNB2), a mutation in an Na-gated potassium channel gene (*KCNT1*), and mutations of the gene *DEPDC5*, which encodes a repressor of the mTOR pathway, have also been described as causes of familial frontal lobe epilepsy [6]. The semiology of lesional frontal lobe seizures depends primarily on the zone of seizure origin in the frontal lobe. The highest diagnostic value is attributed to the first clinical signs of a seizure (typical signs of lateralization of a focal seizure; [35]). Typically, the seizures here often occur as clusters from sleep [6]. Using a diagnostic algorithm of nocturnal events, Derry et al. (2009) were able to correctly identify 94% of 120 nocturnal events. Crucial for this was the exact indication of whether the patient subsequently wakes up completely, as well as the occurrence of a versive posture and assessment of the patient's reorientation phase or postictal state [14]. Furthermore, the work of Derry et al. (2009) elaborated characteristics that allow the presence of parasomnia to be strongly or moderately distinguished, or those that do not contribute to the distinction. Again, markedly long duration, lack of post-episode awakening, and highly variable semiology were particularly indicative of parasomnias ([14]; ◘ **Infobox 2**).

Epileptic seizures in sleep-related epileptic seizures in specific focal epilepsies of childhood have a highly characteristic seizure semiology, which must be ascertained from the patient history (benign epilepsy with centrotemporal spikes [BECTS], benign epilepsy with occipital paroxysms [Panayiotopoulos or Gastaut type], Landau–Kleffner syndrome, and epilepsy with continuous sharp waves during slow-wave sleep [CSWS/ESES]). Accounting for 75–80% of the sleep cycle, NREM sleep is principally associated with a proconvulsive effect; by contrast, REM sleep, accounting for only 20–25% of the sleep cycle, is associated with an anticonvulsive effect. Seizures in nonlesional focal epilepsy in childhood occur preferentially briefly in NREM sleep after falling asleep

Infobox 1

Typical clinical features of ADNFLE (according to [27, 28])

- Cluster of nocturnal motor seizures
- Stereotypical seizure symptoms
- Arousal from sleep to dramatic, bizarre hyperkinetic phenomena
- Tonic/dystonic movement patterns
- Awareness often preserved
- Auras possible (anxiety, vertigo, non-specific, feeling of falling, etc.)
- Seizure clusters in all sleep stages
- Mostly from NREM sleep (stage 2)
- Duration 5 s to 5 min
- Rare seizures during the day
- Rapidly falling asleep after the seizure
- Clarity of consciousness during seizure
- Fear of falling asleep again

and before awakening in the morning [6, 36, 37]. In addition to a characteristic history, epilepsy-typical potentials on EEG or the transition of a suspected seizure into a bilateral tonic–clonic seizure is ultimately conclusive for the presence of epilepsy (■ **Table 1**).

Practical conclusion

- **NREM parasomnia and sleep-related seizures in nonlesional epilepsy can result in complex behavior during sleep. Differentiation can be challenging.**
- **Helpful questions to clarify nocturnal events:**
  When does the event occur?
  How often per night/per week?
  How does it start and end, and how long does it last?
  Is the person awake/wakeable?
  Does the person have a recollection of the event?
  Other symptoms (sweating, tachycardia, tachypnea, tremor, increased muscle tone)?
  Do other family members exhibit similar behavior?
- **Only a video-EEG analysis of the attacks makes it possible to differentiate between sleep-related epileptic seizures and NREM parasomnias.**
- **Video-EEG analysis is the gold standard for differential diagnosis and should always be performed when a reliable differentiation between parasomnias and sleep-related seizures is not possible on the basis of medical history.**
- **In patients with persistent unexplained behavioral abnormalities during sleep, close collaboration between epileptology and sleep medicine is essential.**

Infobox 2

Clinical phenomena of parasomnia (according to [13])

Clinical phenomena that make parasomnia ***highly likely*** *:*
Yawning, scratching, and abnormal nasal rubbing, rolling around in bed, internal or external triggers (noises, coughing, snoring), variable clinical symptomatology, physical and verbal interactions, emotional behavior, unclear ending, no full awakening after event with persistently altered behavior, duration > 2 min, discrepancy between severity and duration of personally experienced and recorded event.

Clinical phenomena that make parasomnia ***moderately likely*** *:*
Tremor/shaking, myoclonic twitching, coughing, senseless behavior, fumbling, manipulation of nearby objects, lack of stereotypy, failure to record an event on the first night of monitoring, recording fewer events (fewer than three).

Clinical phenomena that make parasomnia ***unlikely*** *:*
Brief episodes, sitting, standing, or walking around, preceded by "normal" awakening, brief episodes of arousal (up to 10 s), anxious emotional behavior.

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Declarations

Conflict of interest. E. Haberlandt declares that there is no conflict of interest.

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References

1. American Academy of Sleep Medicine (2014) International classification of sleep disorders, revised: diagnostic and coding manual, 3 edn. American Academy of Sleep Medicine, Westchester
2. Steinberg R, Weeß H-G, Landwher R (2010) Kapitel 3.5. In: Schlafmedizin – Grundlagen und Praxis, 2 edn. Uni-Med Science, pp 103–119
3. Happe S, Walther BW (2009) Kapitel 5. In: Schlafmedizin in der Praxis. Ecomed Medizin, pp 2017–2279
4. Tinuper P, Provini F, Bisulli F, Lugaresi E (2005) Hyperkinetic manifestations in nocturnal frontal lobe epilepsy. Semeiological features and physiopathological hypothesis. Neurol Sci 26(Suppl 3):s210–s214. https://doi.org/10.1007/s10072-005-0489-3
5. Kahn A, Van de Merckt C, Van de Rebuffat E et al (1989) Sleep problems in healthy preadolescents. Pediatrics 84(3):542–546
6. Heidbreder A, Möddel G (2016) Non-REM-Parasomnie oder Frontallappenepilepsie – Warum sollte man genau hinschauen? Klin Neurophysiol 47:1–8. https://doi.org/10.1055/s-0042-110083
7. Stuck BA, Maurer JT, Schredl M, Weeß H-G (2009) Kapitel 7. In: Praxis der Schlafmedizin. Springer, Berlin, Heidelberg, pp 201–220
8. Ackermann S, Rasch B (2014) Differential effects of non-REM and REM sleep on memory consolidation? Curr Neurol Neurosci Rep 14(2):430. https://doi.org/10.1007/s11910-013-0430-8
9. Stores G (1996) Practitioner review: assessment and treatment of sleep disorders in children and adolescents. J Child Psychol Psychiatry 37(8):907–925. https://doi.org/10.1111/j.1469-7610.1996.tb01489.x
10. Högl B, Stefani A (2017) REM sleep behavior disorder (RBD): Update on diagnosis and treatment. Somnologie (Berl) 21(Suppl 1):1–8. https://doi.org/10.1007/s11818-016-0048-6
11. Petit D, Pennestri MH, Paquet J, Desautels A, Zadra A, Vitaro F, Tremblay RE, Boivin M, Montplaisir J (2015) Childhood sleepwalking and sleep terrors: a longitudinal study of prevalence and familial aggregation. JAMA Pediatr 169(7):653–658. https://doi.org/10.1001/jamapediatrics.2015.127
12. Derry CP, Duncan JS, Berkovic SF (2006) Paroxysmal motor disorders of sleep: the clinical spectrum and differentiation from epilepsy. Epilepsia 47(11):1775–1791. https://doi.org/10.1111/j.1528-1167.2006.00631.x
13. Derry CP, Davey M, Johns M et al (2006) Distinguishing sleep disorders from seizures: diagnosing bumps in the night. Arch Neurol 63(5):705–709. https://doi.org/10.1001/archneur.63.5.705 (published correction appears in Arch Neurol. 2006 Jul;63(7):1037)
14. Derry CP, Harvey AS, Walker MC, Duncan JS, Berkovic SF (2009) NREM arousal parasomnias and their distinction from nocturnal frontal lobe epilepsy: a video EEG analysis. Sleep 32(12):1637–1644. https://doi.org/10.1093/sleep/32.12.1637
15. Castelnovo A, Lopez R, Proserpio P, Nobili L, Dauvilliers Y (2018) NREM sleep parasomnias as disorders of sleep-state dissociation. Nat Rev Neurol 14(8):470–481. https://doi.org/10.1038/s41582-018-0030-y
16. Terzaghi M, Sartori I, Tassi L et al (2009) Evidence of dissociated arousal states during NREM parasomnia from an intracerebral neurophysiological study. Sleep 32(3):409–412. https://doi.org/10.1093/sleep/32.3.409
17. Guilleminault C, Lee JH, Chan A, Lopes MC, Huang YS, da Rosa A (2005) Non-REM-sleep instability in recurrent sleepwalking in prepubertal children. Sleep Med 6(6):515–521. https://doi.org/10.1016/j.sleep.2005.03.003
18. Mahowald MW, Schenck CH (2005) Non-rapid eye movement sleep parasomnias. Neurol Clin 23(4):1077–1106. https://doi.org/10.1016/j.ncl.2005.06.001

19. Bandarabadi M, Vassali A, Tafti M (2020) Sleep as a default state of cortical and subcortical networks. Curr Opin Physiol 15:60–67
20. Bloomfield ER, Shatkin JP (2009) Parasomnias and movement disorders in children and adolescents. Child Adolesc Psychiatr Clin N Am 18(4):947–965. https://doi.org/10.1016/j.chc.2009.04.010
21. Lecendreux M, Bassetti C, Dauvilliers Y, Mayer G, Neidhart E, Tafti M (2003) HLA and genetic susceptibility to sleepwalking. Mol Psychiatry 8(1):114–117. https://doi.org/10.1038/sj.mp.4001203
22. Castelnovo A, Turner K, Rossi A et al (2021) Behavioural and emotional profiles of children and adolescents with disorders of arousal. J Sleep Res 30(1):e13188. https://doi.org/10.1111/jsr.13188
23. Kotagal S (2008) Parasomnias of childhood. Curr Opin Pediatr 20(6):659–665. https://doi.org/10.1097/MOP.0b013e328316bd9d
24. Zadra A, Desautels A, Petit D, Montplaisir J (2013) Somnambulism: clinical aspects and pathophysiological hypotheses. Lancet Neurol 12(3):285–294. https://doi.org/10.1016/S1474-4422(12)70322-8
25. Postuma RB, Gagnon JF, Montplaisir J (2013) Rapid eye movement sleep behavior disorder as a biomarker for neurodegeneration: the past 10 years. Sleep Med 14(8):763–767. https://doi.org/10.1016/j.sleep.2012.09.001
26. Bjorvatn B, Grønli J, Pallesen S (2010) Prevalence of different parasomnias in the general population. Sleep Med 11(10):1031–1034. https://doi.org/10.1016/j.sleep.2010.07.011
27. Young GB, Blume WT, Wells GA, Mertens WC, Eder S (1985) Differential aspects of sleep epilepsy. Can J Neurol Sci 12(4):317–320. https://doi.org/10.1017/s0317167100035411
28. Kurahashi H, Hirose S (2002) Autosomal dominant nocturnal frontal lobe epilepsy. In: Adam MP, Everman DB, Mirzaa GM et al (eds) GeneReviews®. University of Washington, Seattle
29. Vendrame M, Kothare SV (2011) Epileptic and nonepileptic paroxysmal events out of sleep in children. J Clin Neurophysiol 28(2):111–119. https://doi.org/10.1097/WNP.0b013e3182120fdc
30. Tinuper P, Provini F, Bisulli F et al (2007) Movement disorders in sleep: guidelines for differentiating epileptic from non-epileptic motor phenomena arising from sleep. Sleep Med Rev 11(4):255–267. https://doi.org/10.1016/j.smrv.2007.01.001
31. Steinlein OK, Mulley JC, Propping P et al (1995) A missense mutation in the neuronal nicotinic acetylcholine receptor alpha 4 subunit is associated with autosomal dominant nocturnal frontal lobe epilepsy. Nat Genet 11(2):201–203. https://doi.org/10.1038/ng1095-201
32. Heron SE, Smith KR, Bahlo M et al (2012) Missense mutations in the sodium-gated potassium channel gene KCNT1 cause severe autosomal dominant nocturnal frontal lobe epilepsy. nat Genet 44(11):1188–1190. https://doi.org/10.1038/ng.2440
33. Møller RS, Heron SE, Larsen LH et al (2015) Mutations in KCNT1 cause a spectrum of focal epilepsies. Epilepsia 56(9):e114–e120. https://doi.org/10.1111/epi.13071
34. Picard F, Makrythanasis P, Navarro V et al (2014) DEPDC5 mutations in families presenting as autosomal dominant nocturnal frontal lobe epilepsy. Neurology 82(23):2101–2106. https://doi.org/10.1212/WNL.0000000000000488
35. Kellinghaus C, Lüders HO (2004) Frontal lobe epilepsy. Epileptic Disord 6(4):223–239
36. Shouse MN (1987) Thalamocortical mechanisms of state-dependent seizures during amygdala kindling and systemic penicillin epilepsy in cats. Brain Res 425(1):198–203. https://doi.org/10.1016/0006-8993(87)90501-4
37. Steriade M, Contreras D, Amzica F (1994) Synchronized sleep oscillations and their paroxysmal developments. Trends Neurosci 17(5):199–208. https://doi.org/10.1016/0166-2236(94)90105-8