Sleep Science and Practice

REVIEW Open Access

Rhythmic movements in sleep disorders and in epileptic seizures during sleep

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Abstract

Rhythmic movements during sleep may occur in the context of physiological sleep-related motor activity or be part of sleep-related movement disorders such as bruxism, periodic limb movement disorder, restless legs syndrome, and sleep-related rhythmic movement disorder. They may also characterize some frontal or temporal nocturnal seizures of sleep-related hypermotor epilepsy, or be considered as part of NREM parasomnias, especially sleepwalking, sexsomnia or sleep-related eating disorder, or REM-related behaviors such as REM behavior disorder or complex movements associated with pediatric narcolepsy type I.

In most cases rhythmic movements, especially when complex and long in duration, are related to different levels of arousal from sleep with recurrence of ancestral motor sequences promoted by central pattern generators in the brainstem, generally activated by the arousal process.

A differential diagnosis between physiological events and pathological movement disorders, and between the latter and sleep-related epilepsy is critical to provide optimal treatment and an accurate prognosis. Specific treatment options for the aforementioned disorders are beyond the scope of this article.

Keywords: Rhythmic movements, Sleep, Seizures, Parasomnias, Central pattern generators (CPGs)

Introduction

Simple and complex rhythmic movements during sleep can present as normal variants of physiological sleep movements [Sleep starts, Alternating Leg Movement Activity (ALMA), Hypnagogic Foot Tremor (HFT), Excessive Fragmentary Myoclonus (EFM)], as part of sleep-related movement disorders (SRMDs) [benign neonatal sleep myoclonus, bruxism, Periodic Limb Movement Disorder (PLMD), Restless Legs Syndrome (RLS), Sleep-related rhythmic movement disorder (SRRMD), and Propriospinal Myoclonus], as complex parasomnias such as sexsomnia (Andersen et al., 2007), as motor dyscontrol during REM sleep in children with type I narcolepsy (Antelmi et al., 2017), or as nocturnal hypermotor seizures of Sleep-related Hypermotor Epilepsy (SHE) (Tinuper et al., 2016) (See Table 1).

We aim to contextualize the different sleep-related motor events in relation to different physiopathogenetic mechanisms, while underscoring the novel unifying concept of the arousal process as their promotor.

Indeed, different theories may account for their origins and implications, most of which tend to favor a unitary physio-pathogenetic mechanism, crediting the arousal process as an important factor promoting a spectrum of behaviors that appear to be genetically embedded in our brain.

In light of this novel approach, we chose to group paroxysmal nocturnal epileptic events and sleep-related motor events (ICSD 3) to emphasize their clinical similarities rather than differences, possibly accounted for by common neurophysiologic mechanisms.

Central pattern generators (CPGs): the carillon theory

An Italian neurologist, C.A. Tassinari, claimed that genetically determined motor neural networks embedded in the spinal cord and brainstem of primates, first described by T.G. Brown in 1911 (Tassinari et al., 2012), can generate coordinated rhythmic movement patterns. These networks named Central Pattern Generators (CPGs) are able to promote a spectrum of species-

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Table 1 Different Rhythmic Movements in Sleep

Different Rhythmic Movements in Sleep			
	Wakefulness	NREM	REM
Normal Variants of Physiological Sleep Movements			
Sleep starts	✓		
HFT	✓	✓	
ALMA	✓	✓	✓
EFM		✓	
Sleep Related Movement Disorders (SRMDs)			
Benign neonatal myoclonus		✓	
Bruxism	✓	✓	✓
PLMS/PLMD		✓	✓
RLS	✓		
Leg cramps	✓	✓	
SRRMD	✓	✓	✓
PSM	✓		
DOA, Sexsomnia, SRED		✓	
REM sleep dyscontrol in type I Narcolepsy, RBD			✓
Nocturnal Hypermotor Seizures in SHE		✓	

Abbreviations: *HFT* Hypnagogic Foot Tremor; *ALMA* Alternating Leg Movement Activity; *EFM* Excessive Fragmentary Myoclonus; *PLMS* Periodic Limb Movements in Sleep; *PLMD* Periodic Limb Movement Disorder; *RLS* Restless Legs Syndrome; *SRRMD* Sleep Related Rhythmic Movement Disorder; *PSM* Propriospinal Myoclonus; *DOA* Disorders of Arousal; *SRED* Sleep Related Eating Disorder; *RBD* REM-sleep Behavior Disorder; *SHE* Sleep-related Hypermotor Epilepsy

specific self-sustained patterns of archetypical behaviors. The latter include locomotion, self-defense, feeding and reproductive behaviors critical for survival.

According to the "Triune" brain theory by MacLean (Reiner, 1990), they are the expression of the disinhibition of more primitive layers of the human brain, the limbic or paleopallium, and the midbrain or reptilian brain, which under certain conditions may escape the control of our most elaborate brain, the neopallium (see Fig. 1). An arousal is an event that triggers a spectrum of behaviors that look the same whether they are part of a seizure or of a parasomnia/sleep motor event.

This neuro-ethologic approach predicates that CPGs act as a sort of "carillon" (Tassinari et al., 2009), playing their stereotyped rhythmic motor melody, regardless of the stimulus prompting their activation.

Shared semiotic behaviors in different pathological conditions may, therefore, be seen upon arousal.

Different types of rhythmic behaviors such as teeth grinding and periodic leg movements may co-occur, whether in the context of temporal lobe seizures or as sleep bruxism (Meletti et al., 2004).

In both cases these behaviors occur during sleep, elicited by an arousal, and share burst frequency and automatic (HR) activation.

Other suitable examples include pedaling activity and pelvic thrusting described in the context of hypermotor frontal seizures (Tassinari et al., 2005), as well as RLS (Högl et al., 2007).

Copulatory violent behaviors during sleep including rhythmic pelvic thrusting may be part of sexsomnia episodes as well as temporal seizures (Voges et al., 2019).

Medial-frontal or even extra-frontal (Nobili et al., 2004) nocturnal seizures including leg flexion, pedaling, or crawling have been reported. Most of these patients also show periodic limb movements unrelated to EEG ictal abnormalities (Terzaghi et al., 2009).

Crawling behavior and sleepwalking automatisms are also part of disorders of arousal (DOA) in young children and adolescents.

The younger the brain, due to incomplete myelination, the more vulnerable the motor control, easily escaping the neopallium guidance; this is one of the reasons typical DOA are defined as genetically determined patterns of immature brains.

The role of cyclic alternating pattern (CAP)

CAP is defined as an alternation of transient activations (phase A) over a more stable background pattern (phase B) within periods of NREM sleep (Parrino et al., 2006).

The different A phases express the arousability-reactivity of the sleeping brain at different ages and sleep stages; A1 being prevalent in younger subjects during N3, whereas A2 and A3 express more sleep disruption in adults. Both nocturnal seizures in SHE, as well as SRMD and parasomnias, occur during the A phases, once again confirming the arousal mechanism as the prompting disinhibiting factor initiating the motor rhythm pattern in seizures as well as in normal sleep.

A comparison of sleep-related movement disorders and parasomnias with rhythmic features versus sleep-related hypermotor epilepsy (SHE) according to the ICSD-3 clinical criteria and the AASM scoring manual for sleep and associated events

Sleep-related movement disorders where the motor program is disimilar to epilepsy

Periodic limb movements in sleep (PLMS)

These are the most common sleep related movement disorder. PLMS in isolation are often picked up as an incidental finding on leg EMG or polysomnography, or the patient is brought by their spouse whose sleep is much more commonly disrupted by the repetitive kicking of the PLMS than the sleep of the patient. The other common scenario is in RLS when the PLMS may bleed into wakefulness in the form of myoclonic jerking of the legs while awake and lying or sitting. In this context, the patient usually appears hyperactive, irritable, emotionally

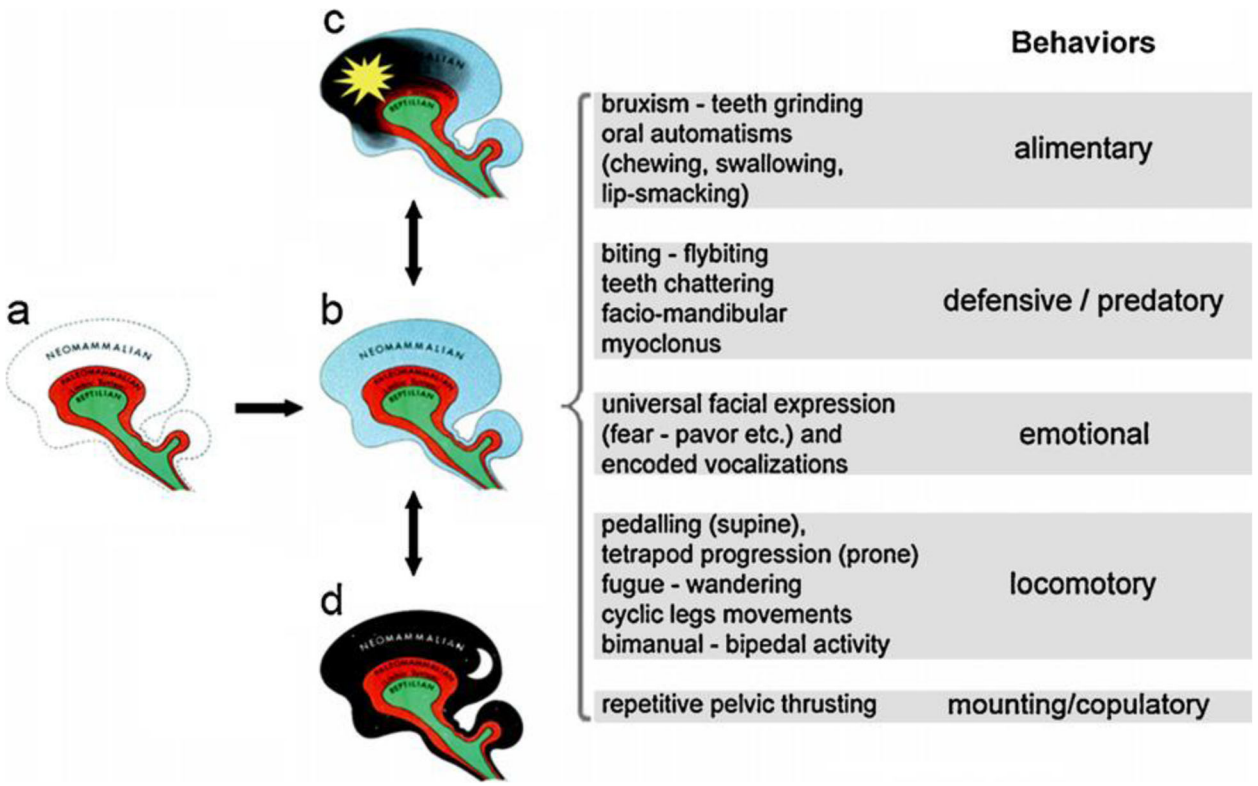

Fig. 1 The Triune Brain (MacLean, 1990). The immature paleomammalian neonatal brain **(a)** is unable to efficiently control motor output, due to incomplete myelination. Thus, fundamental (alimentary) and protective motor reflexes, like grasping and avoidance, are carried out by subcortical CPGs. **(b)** The mature neopallium controls and inhibits during conscious wakefulness most of these reflexes which may reemerge under special conditions, due to transient loss of neocortical control, as in **c** (during an epileptic seizure), or in **d** (during cerebral anoxia or in parasomnia/sleep-related motor events). *Modified with permission from Tassinari* et al., *2009*

distressed and presents a decreased attention span. In severe cases, these symptoms, especially in children, may progress to full blown ADHD.

PLMS are 0.5–10 s in duration and 5–90 s apart. At least 4 such movements in a row must be present for the individual movements to be classified as PLMS. These movements may intermittently continue for hours throughout the night, during both light NREM and REM sleep (Manconi et al., 2007), and may or may not be accompanied by EMG arousals. The movements themselves usually are dorsiflexons of the foot accompanied by flexions of the knee and thigh. They may be unilateral or bilateral (Iber et al., 2018). We did an early detailed analysis of PLMS and found that within a series, the movements are very stereotypic, but as the night progresses and body position changes so that the next series of movements may appear entirely different, e.g. the first series of movements might involve the right leg only, the second series might involve the left leg only, the third series both legs and the fourth series both legs and an arm. Dystonic leg movements or leg rolling were seen in some patients as part of a shift of the types of movements between series (unpublished data). The long duration of these movements and the long inter-movement intervals seen in PLMS suggest that comparison to sleep-related epilepsy is remote (see Table 2).

Sleep-related leg cramps

Sleep-related leg cramps consist of muscle cramps that last seconds to a few minutes and can occur during or around the time of sleep. They are often precipitated by vigorous exercise during the day and often consist of a plantar flexion of the foot. These features attest to the probably neuromuscular origin of the disorder and comparison to sleep-related epilepsy is thus remote (The American Academy of Sleep Medicine, 2014); additionally, most often, they do not display any rhythmicity.

Propriospinal myoclonus at sleep onset

In Propriospinal Myoclonus at sleep onset (PSM) body jerks are seen at sleep onset when the patient is lying down. The patients do not have RLS or PLMS. Rapid EMG has determined that the origin of this disorder lies in the propriospinal tracts of the spinal cord, thus the comparison to sleep-related epilepsy is remote (The American Academy of Sleep Medicine, 2014). There are

Table 2 Clinical features contributing to the differential diagnosis of paroxysmal movements during sleep

Clinical features contributing to the differential diagnosis of paroxysmal movements during sleep				
	Myoclonic Jerks	Stereotyped Movements	Sensory Symptoms	Behavioral Alterations
Normal Variants of Physiological Sleep Movements				
Sleep starts	✓		✓	
HFT		✓		
ALMA		✓		
Sleep Related Movement Disorders (SRMDs)				
Benign neonatal myoclonus	✓	✓		
Bruxism		✓		
PLMS/PLMD		✓		
RLS			✓	✓
Leg cramps			✓	
SRRMD		✓		✓
PSM	✓			
DOA, Sexsomnia, SRED			✓	✓
REM sleep dyscontrol in type I Narcolepsy, RBD				✓
Nocturnal Hypermotor Seizures in SHE	✓	✓		✓

Abbreviations: *HFT* Hypnagogic Foot Tremor; *ALMA* Alternating Leg Movement Activity; *PLMS* Periodic Limb Movements in Sleep; *PLMD* Periodic Limb Movement Disorder; *RLS* Restless Legs Syndrome; *SRRMD* Sleep Related Rhythmic Movement Disorder; *PSM* Propriospinal Myoclonus; *DOA* Disorders of Arousal; *SRED* Sleep Related Eating Disorder; *RBD* REM-sleep Behavior Disorder; *SHE* Sleep-related Hypermotor Epilepsy

no specific scoring rules for PSM on polysomnography except that the movements occur in wakefulness at sleep onset; they mostly do not include myoclonic rhythmic features.

Excessive fragmentary myoclonus

Excessive Fragmentary Myoclonus (EFM) is not a true movement disorder in that the movements resemble muscle fasciculations. In fact, they are arrhythmic muscle twitches that do not cause any visible large movements across joint spaces and are picked up as an incidental finding on leg EMG in NREM sleep during an overnight polysomnography. For this reason, they are described in this chapter only to fit the purpose of a possible differential diagnosis with other sleep-related movement disorders with rhythmic features. They resemble the phasic REM twitches that are seen in normal REM sleep or that appear in excess in REM Sleep Behavior Disorder (RBD). Most of the movements are 150 msec in duration. At least 5 such twitches/min must be seen for 20 min on the leg EMG during polysomnographic recording of NREM sleep to meet EFM criteria. The individual bursts of EFM tend not to cluster to the degree that phasic REM twitches do. Because no or only small muscle twitches are involved, with no large movements across a joint space, and because these movements resemble fasciculations which are known to be of neuromuscular origin, comparison with epilepsy is remote (Iber et al., 2018).

Sleep-related movement disorders where the motor program is similar to epilepsy

Hypnic jerks (sleep starts)

These are common benign movements seen just prior to falling asleep and usually consist of a single whole-body jerk right before sleep onset. Patients often experience the beginning of a sleep onset as frightening, with a sense of falling or a sense that an object is coming toward them. They often state that the awareness that they are falling asleep too fast frightens them and precipitates the body jerk. On rare occasions, the hypnic jerks can be multiple and interfere more profoundly with sleep onset, in which case the category of hypnic jerks crosses over from benign to disorder. Similar jerking movements have been described in epilepsy (Iber et al., 2018).

REM sleep behavior disorder

The same considerations apply to normal or excess phasic REM twitches in REM sleep behavior disorder (RBD). Either no movement is observed, or only small movements can be detected, such as minor twitching around the corner of the mouth or slight finger flexion and extension. To score phasic REM twitches in RBD, each 30-s epoch is divided into ten 3-s mini-epochs. Any mini epoch in which even a single phasic burst occurs is considered a positive epoch, and at least 5 of the mini-epochs must contain such twitches that are at least 4 times of the background EMG, for the entire 30-s epoch to be considered characteristic of RBD. The excess phasic twitches tend to occur in clusters of 0.1–5 s.

Typically, the chin EMG tone is low in normal REM sleep. In RBD, at least 50% of a 30-s epoch on a polysomnogram must have elevated chin tone for the epoch to be considered characteristic of RBD. 27% of all 30-s epochs in REM sleep must have either excess phasic activity of the chin or flexor digitorum superficialis EMG or, alternatively, elevated chin EMG for the entire overnight polysomnographic study to be considered characteristic of RBD. Again, because of the same considerations for EFM, the comparison to epilepsy is remote for this portion of the RBD spectrum (Iber et al., 2018).

It should be noted that when we speak of phasic REM twitches, we are referring to the minor movements that occur between episodes of dream enactment in RBD where large body movements appear to be voluntary but are outside the conscious awareness of the individual. Patients may enact running or fighting or participate in simulated swinging of a tennis racquet, golf club or baseball bat. Occasionally, rhythmic sequences including punching, kicking, and biting, may be described. Sleep related injury as a consequence is common in RBD. Here the comparison to epilepsy is extremely relevant as these types of movements can appear either in Disorders of Arousal (DOA, see below) or in partial seizures occurring during sleep of either the frontal or temporal lobe type. An overnight polysomnogram with full head EEG may help distinguish the EMG characteristics of RBD from inter-ictal spikes characteristic of epilepsy seen on the EEG. If the overnight polysomnography does capture any complex behaviors, the latter tend to repeat themselves in a stereotypic manner in sleep-related epilepsy, as opposed to the large variety of dream enactment behaviors seen in RBD, or non-stereotypic behaviors such as sleep walking, sleep terrors or confusional arousals seen in the umbrella category for these disorders known as DOA. A high percentage of patients with RBD will eventually develop parkinsonism or other neurodegenerative disorders of the alpha synuclein type (Iber et al., 2018) (The American Academy of Sleep Medicine, 2014).

Disorders of arousal (DOA)

In DOA, patients are caught between NREM sleep, usually deep N-3 sleep, and wakefulness. The mind is asleep, but the motor behavior of wakefulness is intact. Patients appear bewildered and often terrorized and may walk, talk scream and run around in a confused manner. As opposed to RBD, there is little or no dream recall and little or no recall of the events themselves; furthermore, in DOA the behaviors are not driven by dreams. There are no specific findings on polysomnography except that behaviors occur during NREM sleep or during the emergence from NREM sleep (Iber et al., 2018) (The American Academy of Sleep Medicine, 2014). Again, comparison to epilepsy is relevant in that similar behaviors may occur in sleep-related epilepsy or RBD. DOA usually occur in young children, whereas RBD is typically observed in older men.

Rhythmic movements within DOA are, however, rare and may be seen either as part of exploratory movements in abortive sleepwalking or, more frequently, as part of complex DOA, in particular, as Sleep Related Eating Disorder (SRED), where food intake may be preceded by orofacial myoclonus, rhythmic chewing (Vetrugno et al., 2006) and in sexsomnia, often accompanied by pelvic thrusting or rhythmic movements while attempting coitus. A sensory component is implicit in both SRED and sexsomnia and is strictly linked to the arousal initiating these behaviors.

Bruxism

Bruxism or tooth grinding may occur during sleep. Two types have been described. One is a phasic or grinding type where each grinding noise occurs every few seconds or so, and the bursts are 0.25–2 s in duration. At least 3 such individual grinds must be present to define an episode of bruxism. At least two audible episodes of bruxism on a night of polysomnographic study are necessary to make the diagnosis of bruxism. Individual grinds have to be twice the amplitude of the background EMG on an overnight polysomnography. The second kind is a clenching type where the tooth clenching lasts 2 s or more. Episodes have been described in all sleep stages, including REM (5%) (Tosun et al., 2003). Such phasic rhythmic episodes have been described in epilepsy as well (Iber et al., 2018).

Rhythmic movement disorder

Rhythmic Movement Disorder (RMD) is, by definition, the stereotypical sleep-related movement disorder. It occurs primarily in children where repetitive head banging (*jactatio capitis nocturna*), body rocking or other repetitive stereotypic movements occur prior to sleep onset and into light sleep. All of these may be considered as specific *pre dormitum* behaviors. Especially in older subjects, movements may be spread out and occur in any sleep stage, including REM (Mayer et al., 2007). The individual repetitive movements occur at a frequency of 0.5–2 Hz and at least 4 such movements in a row need to be present to make the diagnosis of RMD. Each movement has to be twice the background of EMG on polysomnography. A clinical consequence such as sleep-related injury needs to be present to make the diagnosis of RMD; however, this is often not the scenario, in which case the rhythmic movements are simply described. The semi-voluntary nature of these movements distinguishes them from epilepsy where similar

movements of a similar duration and frequency have also been described (Iber et al., 2018) (The American Academy of Sleep Medicine, 2014). A recent paper by Prihodova et al., (Prihodova et al., 2019) ascertains developmental disorders often comorbid with epilepsy as strong predisposing factors to SRMDs. The latter include ADHD, autism spectrum disorders, Tourette and Rett syndromes, most likely promoting alterations of the neural networks of CPGs responsible for rhythmic movements. Moreover, they describe rocking stereotypies as more typical of transitional NREM sleep, akin to some stereotypies of SHE, whereas rolling movements were most typically associated with REM sleep, when transient loss of the physiological muscle atonia would trigger the disinhibition of brainstem CPGs (Shah et al., 2018).

Hypnogogic foot tremor and alternating leg muscle activation

Hypnogogic Foot Tremor (HFT) and Alternating Leg Muscle Activation (ALMA) are two recently described disorders that are generally picked up as an incidental finding on polysomnography, as patients do not complain of symptoms related to them and no clinical consequences have been described to date. HFT consists of at least 4 bursts of 0.3–4 Hz activity in one leg of the polysomnographic EMG where the usual burst duration of each movement is 250 to 1000 milliseconds. The movements as described are tremors in a single foot, occurring in wake and light stages of NREM sleep (Wichniak et al., 2001).

ALMA, although not well described clinically, alternates between legs with similar frequency of 0.5–3 Hz and similar duration of 100–500 msec. They may occur during all stages of sleep (Chervin et al., 2003). It is suspected that these rhythmic types of behavior are variants of the same disorder and that the minor differences in frequency and duration of the movements may simply represent variance based upon the still small number of cases described in the literature. As with Bruxism and RMD, episodes of similar frequency and burst duration have been described in epilepsy (Iber et al., 2018).

Benign neonatal (infantile) sleep myoclonus

This disorder was originally called Benign Neonatal Sleep Myoclonus but was renamed Benign Infantile Myoclonus when it was realized that cases can extend beyond the neonatal period into infancy. The disorder consists of myoclonic jerks that occur only during sleep in any body part and disappear immediately upon awakening. They tend to occur in clusters. Rocking the infant during sleep can precipitate the movements. Although this disorder is in the differential diagnosis of epilepsy because of the similar appearance of the movements, the symptoms disappear within several weeks with no sequelae, attesting to the benign nature of the disorder (The American Academy of Sleep Medicine, 2014).

Sleep-related movement disorders in the context of nocturnal epilepsy disorders where the movement disorder is epileptic

Sleep-related Hypermotor epilepsy (SHE)

In 1981, Lugaresi and Cirignotta (Lugaresi & Cirignotta, 1981) first described paroxysmal events of different duration during nocturnal sleep characterized by dystonic posturing and violent often rhythmic movements, involving bimanual and cycling bipedal activity.

Later on, similar events were documented within neurosurgical pre-operative in-depth EEG recordings and ascertained as epileptic seizures of frontal origin (NFLE) (Williamson et al., 1985) (Waterman et al., 2012). These seizures mostly, if not exclusively, occur during sleep and comprise seizures of different etiology as part of symptomatic or idiopathic epilepsy. In particular, some familiar cases have been recognized as sharing an autosomal dominant inheritance (ADNFLE) linked to several gene mutations including either different subunits of the nicotinic receptor (nAChR) or, for the most severe forms, mutations of the sodium-activated potassium channel (KCNT1). Clinical and neurophysiological characterization of nocturnal seizures sharing hypermotor events led to some fundamental discoveries which have recently imposed a nomenclature change from NFLE to Sleep-related Hypermotor Epilepsy (SHE). The term was convened upon subsequent to a systematic literature review and a consensus conference gathering epileptologists, epidemiologists and sleep specialists from all over the world familiar with this topic.

The following evidence was discussed:

- Index seizures were pertinent to sleep rather than guided by a circadian framework.
- Motor aspects were the most prominent features of the described clinical events.
- In many cases, an extra-frontal origin had been documented for the alleged hypermotor sleep-related seizures.

In particular, hyperkinetic seizures have been described as originating from temporal lobe structures (Nobili et al., 2004) better characterized from insular (Ryvlin et al., 2006) and insular-opercular areas (Proserpio et al., 2011).

Clinical manifestations of seizures have different durations, as previously described within the context of NFLE (Provini et al., 1999), ranging from minor motor events to paroxysmal arousals to complex hypermotor

seizures or, rarely, long-lasting ambulatory wandering behavior (Montagna, 1992).

Behaviors are usually highly stereotyped within the same individual, often preceded by a microstructural event such as an arousal or CAP transient activation phases. Some seizures more than others display clear rhythmic components (see Fig. 2), including body rocking and rolling, bipedal cycling and kicking. A longer event duration and latency (> 5 s) from the first EEG evidence of seizure and the first hypermotor clinical manifestation characterized extra-frontal vs. frontal origin SHE (Gibbs et al., 2018).

Diagnostic criteria for SHE (Tinuper et al., 2016) distinguish different levels of evidence:

- witnessed (possible) SHE where only eye-witnessing but no objective documentation is provided;
- video-documented (clinical) SHE with typical video but no EEG recording provided;
- video-EEG (confirmed) SHE where full video-EEG corroborates the occurrence of hypermotor behaviors, with associated ictal or interictal typical EEG abnormalities.

The differential diagnosis with DOA in children or adults with similar clinical features relies on a different frequency and distribution pattern of the events, typically occurring sporadically and less stereotyped in the first part of the night (DOA,) versus more frequent, highly stereotyped, evenly-distributed events across the night (SHE) (Vignatelli et al., 2007).

From a neurophysiological standpoint, these criteria have been recently improved by establishing the occurrence of minor but especially full-blown events in N-3

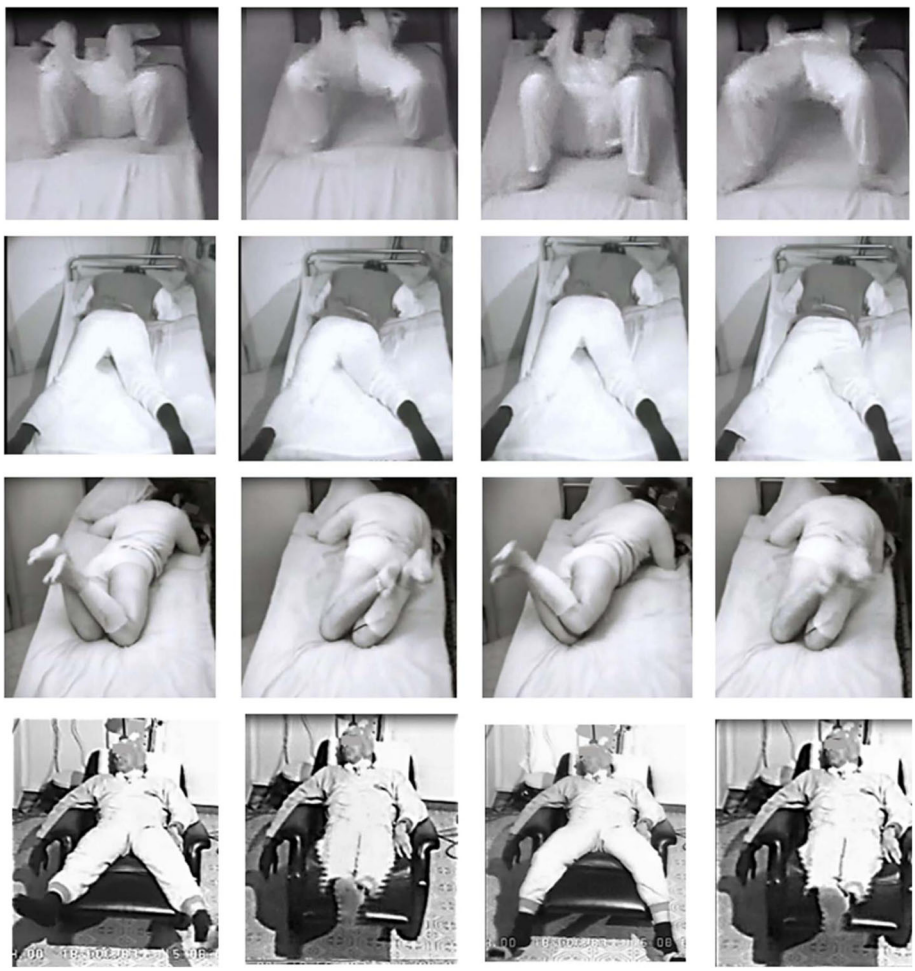

Fig. 2 Seizures fragments in four different patients with Sleep-related Hypermotor Epilepsy. The four photographic sequences show typical stereotyped rhythmic movements in SHE. From top to bottom: repetitive thrusting pelvic movements (in supine and prone position), rhythmic rocking of the pelvis with flexed legs and repetitive opening-closing of the legs (*Courtesy of Prof. Paolo Tinuper, IRCCS Istituto delle Scienze Neurologiche di Bologna, Italy)*

(DOA) versus N-2 (SHE) during nocturnal video-EEG recordings.

Conclusions

Rhythmic movements during sleep may occur as part of SRMDs and different parasomnias with a motor program similar or dissimilar to epilepsy, whereas SHE is the only epileptic movement disorder occurring during sleep that presents rhythmic features.

The description of the different nosological entities pertaining to these groups elucidates a possible common origin from arousal pathology and delineates relevant features that allow a differential diagnosis. The latter is important to address consequent treatment. In particular, recognition of an epileptic disorder warrants specific medications, whereas most DOA do not require any treatment. On the other hand, both REM-related parasomnias, whether or not in the context of narcolepsy, and SRMDs, may respond favorably to specialized treatment.

Abbreviations

ADHD: Attention Deficit Hyperactivity Disorder; ADNFLE: Autosomal Dominant Nocturnal Frontal Lobe Epilepsy; ALMA: Alternating Leg Movement Activity; CAP: Cyclic Alternating Pattern; CPGs: Central Pattern Generators; DOA: Disorders of Arousal; EEG: Electroencephalography; EFM: Excessive Fragmentary Myoclonus; EMG: Electromyography; HFT: Hypnagogic Foot Tremor; HR: Heart Rate; NFLE: Nocturnal Frontal Lobe Epilepsy; NREM: Non-rapid eye movement; PLMD: Periodic Limb Movement Disorder; PLMS: Periodic Limb Movements in Sleep; PSM: Propriospinal Myoclonus; RBD: REM Sleep Behavior Disorder; REM: Rapid Eye Movement; RLS : Restless Legs Syndrome; RMD: Rhythmic Movement Disorder; SHE: Sleep-related Hypermotor Epilepsy; SRED: Sleep Related Eating Disorder; SRMDs: Sleep-Related Movement Disorders; SRRMD: Sleep-Related Rhythmic Movement Disorder

Acknowledgements

The authors would like to thank Federica Provini for her invaluable guidance, advice and adept editing of this article.

Authors' contributions

RS conducted a literature search on: CPGs, CAP and SHE and wrote the respective sections, in addition to the introduction and conclusion. ASW wrote the sections pertaining to PLMS, sleep-related leg cramps, PSM, EFM, sleep starts, RBD, DOA, Bruxism, RMD, HFT, ALMA and Benign Neonatal Sleep Myoclonus. These sections were integrated by RS. Both authors read and approved the final manuscript.

Funding

No funding was obtained for this article.

Availability of data and materials

Not applicable.

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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Received: 25 July 2019 Accepted: 29 January 2020
Published online: 04 March 2020

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