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Things that go bump in the night: diagnosing sleep-related movement disorders without a sleep laboratory

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ABSTRACT It is common for general physicians to experience diagnostic doubt and trepidation whenever faced with patients who exhibit abnormal nocturnal behaviours or excessive movements at night. There is also a perception that expensive and often poorly available overnight tests are usually required for diagnostic precision. In fact, the majority of conditions, whether they be parasomnias or, more rarely, nocturnal seizures, can be reliably diagnosed from a directed history, if available. Although the evidence base for treating parasomnias and sleep-related movement disorders remains minimal, accurate recognition is important for a variety of reasons.
This review covers the diagnostic features of the full range of parasomnias and movement disorders that might present to a multidisciplinary adult sleep clinic. Throughout, it will be argued that the recognition of key or salient features obtained from a good history is the most important diagnostic tool. Indeed, when diagnostic doubt remains after a thorough sleep history, it is relatively rare for detailed tests to add much in the way of useful information.

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1. PARASOMNIAS ARISING FROM NON-RAPID EYE MOVEMENT (REM) SLEEP

Non-REM sleep parasomnias are very common in childhood and are usually benign, rarely coming to medical attention. They typically arise from the first period of deepest non-REM (slow wave) sleep, around an hour after sleep onset. Over time, a spectrum of behaviours may be seen, even in the same subject, varying from brief confused arousals to frank sleepwalking. Agitated events may be seen, especially in younger age groups, leading to disturbing 'night terrors'. Despite apparent severe distress and intense autonomic activation, the child rarely recalls the nocturnal events. Because they are thought to reflect an incomplete transition from deep sleep to wakefulness, non-REM parasomnias are often termed 'arousal disorders'.

Most non-REM sleep parasomnias improve through adolescence but may persist on a regular basis in at least 1% of adults. An enormous range of behaviours may be reported, some of which are complex and potentially hazardous. In adulthood, subjects appear to be more prone to exhibiting activities which may be considered 'goal-seeking' in nature, including eating or cooking. Males, in particular, while in a state of parasomnia, may engage in intimate relations with bed partners or display violence. Subsequent recall is invariably absent or severely diminished although some report dream-like or hallucinatory images, particularly if the parasomnia is associated with significant agitation. Common themes include spiders or other unpleasant visual imagery. Often, there is simply a sense of 'impending doom' causing the subject to rush from the bedroom, often vocalising and disturbing partners. It is usually possible to communicate at a limited level with subjects in this state although they typically display a degree of confusion and appear only partially awake.

Typically, an adult with a significant non-REM sleep parasomnia will have events from deep slow wave sleep within an hour or two of sleep onset. Events rarely occur more than once a night but may cluster with several symptomatic nights in a week. Often, there are more prolonged spells free of disturbances. The behaviours across nights might be similar but are not precisely stereotyped. Triggers or precipitants are often identified and include internal factors that tend to deepen non-REM sleep (such as sleep deprivation) or external factors (such as an uncomfortable or noisy environment) which may partially arouse the deeply sleeping subject. Although controlled evidence is limited, short-acting hypnotics such as zolpidem may induce sleepwalking or similar behaviours in predisposed individuals. Similarly, alcohol in some may appear to trigger parasomnias, potentially through secondary factors such as sleep deprivation, increased snoring or a full bladder. By contrast, in clinical

practice, some subjects report that significant alcohol intake may reduce their chances of exhibiting nocturnal disturbances.It is usually possible to distinguish non-REM sleep parasomnias from nocturnal seizures on clinical grounds (Table 1).

2. REM SLEEP BEHAVIOUR DISORDER

Vivid or narrative dreams are most closely associated with the REM sleep stage. In normal REM sleep, only the eyes and diaphragm should move. Indeed, descending inhibitory impulses from the brainstem actively inhibit voluntary motor neurons. A subject in REM sleep is therefore completely floppy' and should be areflexic if examined with a tendon hammer.

In REM sleep behaviour disorder (RBD) this mechanism fails, causing subjects to literally 'act out' their internal dreams. This parasomnia affects middle-aged or elderly men in particular and is strongly associated with parkinsonism even though the more obvious motor features may develop many years later. This observation has led to great interest in RBD as a potential window for looking at early Parkinson's disease and potentially exploring future effective neuroprotective treatments. Recent evidence suggests that the presence of RBD may eventually predict a more complex form of parkinsonism with dementia and psychosis as early features.

The main characteristics of RBD are outlined in Table 2 Typical behaviours include lashing out, kicking or punching often with vocalisation. The subject will generally recall the dream content if awoken during the event. Since REM sleep is usually concentrated towards the end of the night, RBD episodes tend also to be more pronounced at this time.

The reasons why RBD affects predominantly males or why the dream behaviours are so aggressive or agitated are unclear. Increasingly, however, more benign behaviours such as laughing or singing are being described. Although diagnosis is often very clear from history alone, overnight

TABLE 1 Non-REM sleep parasomnias may mimic frontal lobe epilepsy but, if detailed information on the nature and pattern of events is available, distinction is usually possible without recourse to prolonged overnight polysomnography.

Clinical feature	Non-REM sleep parasomnias (non-RP)	Nocturnal complex partial seizures (often frontal lobe origin, frontal lobe epilepsy [FLE])	Comment
Age of onset	Childhood in vast majority	Variable	Childhood non-RPs will persist into adulthood in around 25% of subjects, often with temporary resolution during adolescence
Positive family history	Present in at least 80%	Present in less than 40%	Specific mutations for non-RP have not been identified A rare form of autosomal dominant FLE is caused by nicotinic receptor mutations
Timing	Usually within 90 minutes of sleep onset	No pattern	Non-RPs arise from deep (slow wave) non-REM sleep FLE is associated with all types of non-REM sleep and very rarely from REM sleep
Events per week	Typically 0–3	>7	Multiple events per night are much commoner in seizure disorders
Identifiable triggers	May be reported (sleep deprivation, factors fragmenting sleep such as severe snoring)	Usually none	
Mental state after event	Confusion and poor recall	Return to full awareness usually quick	Partial awareness and responsivity fairly common during non RP, less common in FLE
Leaving bedroom	Can be reported	Not usually seen	
Behavioural complexity	Extremely variable	Usually not complex and invariably stereotyped	Complex acts such as phone texting and driving are not incompatible with non-RP activity

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TABLE 2 The main characteristics of RBD

Clinical features of REM sleep behaviour disorder (RBD)	Comment
Typical patient is late middle-aged male	Probably more common in women than realised but less troublesome to bed partners
Lashing out, punching, kicking are typical actions, often with vocalisation	The subject will often appear to be acting out an aggressive or unpleasant dream with eyes closed Subject may fall out of bed but ambulation not seen 'Victims' of any violence are bystanders
Events recur through the night	Episodes may be more prolonged towards the end of the night when REM sleep is more dominant
Return to full awareness after event generally quick	Associated dream recall common
Subtle signs of parkinsonism may be present	Mild tremor/hypokinesia and symptoms of reduced olfaction should be sought
Injuries not uncommon	Bed partners usually bear the brunt of violent behaviours
Spontaneous resolution rare	Long-term treatments may be indicated
Antidepressant therapy may worsen episodes	Venlafaxine and mirtazapine appear to be particularly prone to exacerbating RBD
Can be seen in younger patients	Usually in combination with non-REM sleep parasomnias (an 'overlap' parasomnia) or in the context of narcolepsy

polysomnography is advocated by many to confirm the presence of abnormal muscle tone during REM sleep periods. The investigation will also help to rule out agitated or confused arousals from severe snoring or frank apnoeas which may mimic RBD in subjects with obstructive sleep-related breathing disorder.

It may also be observed in up to 30% of narcoleptic subjects although it is rarely a clinical problem in this group. Occasionally, it can also accompany non-REM parasomnias such as sleepwalking in younger subjects when the term 'overlap parasomnia' may be used. Most antidepressant drugs worsen or even induce RBD and should be discontinued if possible.

3. PERIODIC LIMB MOVEMENTS OF SLEEP AND RESTLESS LEGS SYNDROME

Restless legs syndrome (RLS) is best conceived as a sensorimotor disorder, defined wholly by subjective symptoms and characterised predominantly by a strong urge to move the legs, particularly in the late evening. There is nearly always associated limb discomfort that can be difficult both to describe and localise. A common report is that of an unpleasant 'crawling' sensation under the skin around the shins. Symptomatic but temporary relief is obtained by moving or rubbing the affected limb(s). The adverse consequences on both sleep onset and maintenance are often severe and may lead to the potentially sterile debate of whether RLS is more a movement or a sleep disorder. In any event, RLS is common and affects around 3% of Caucasian populations when screened by personal interview using validated criteria. Given the relatively non-specific nature of these symptom-based diagnostic criteria, self-completed questionnaires may lack diagnostic precision and have led to overestimates of RLS prevalence in the past. Furthermore, there is a clearly an extremely wide spectrum of RLS symptom severity and frequency with approximately 20% of symptomatic subjects reporting clinically significant symptoms on more than two evenings each week, potentially justifying attempts at drug therapy. In severe or atypical cases, leg restlessness and discomfort may extend to other body parts, particularly the arms.

It affects females preferentially and there is an association with increasing age up until the eighth decade. However, RLS also affects children and, as such, is often unrecognised in younger populations. A positive family history is frequently obtained, especially if symptom onset occurs before 40 years of age.

Many people with RLS will also be aware of involuntary limb movements that may occur at regular intervals during the wakeful state, usually when drowsy or at the point of sleep onset. However, these periodic limb movements (PLMs) are most frequently observed or noticed by bed partners during non-REM sleep in the first third of the night and affect the vast majority of RLS subjects. Most authorities consider the clinical phenomena of RLS and PLMs to share both a common underlying neurobiology and potential for adverse health consequences. Phenotypically, PLMs usually resemble a slowed version of a withdrawal reflex in a lower limb, starting with great toe and then foot dorsiflexion. The slow flexor jerk may then extend to involve the knee and hip over 0.5–1 seconds, potentially then leading to a more generalised movement or arousal from sleep which may mimic simple restlessness. The duration of movements associated with a PLM may last up to ten seconds as recognised by recently modified scoring criteria. They can also occur in REM sleep but are usually of shorter duration and less frequent.

During sleep PLMs can certainly affect the sleep quality of bed partners but may well not be recognised or recalled by the subjects themselves. When monitored, transient heart rate rises often coincide with PLMs, indicating minor associated autonomic arousals in the absence of definite electroencephalogram (EEG) changes. In general, up to a third of PLMs are linked to micro-arousals, defined as a return to alpha or theta EEG activity for 3–15 seconds. This remains a controversial area, however, largely due to issues around reliably scoring and defining these minor EEG changes.

PLMs during sleep may also be observed in up to 30% of normal elderly populations but are usually at a low level and have no likely clinical significance. Indeed, a number of studies have failed to demonstrate a clear relation between the frequency of PLMs during the night and reports of poor sleep quality or daytime somnolence, even when the PLMs are reported to have caused micro-arousals.

Rarely, significant and potentially arousing PLMs are seen during sleep recordings of subjects with unrefreshing sleep in the absence of RLS, reflecting periodic limb movement disorder (PLMD). In such subjects, especially if there is no bed partner, PLMs may not have been suspected from history alone even though sleep continuity is adversely affected and the deeper stages of non-REM sleep poorly maintained. As a result, PLMD is important to recognise and treat as it may rarely cause significant daytime somnolence. Furthermore, troublesome adult sleepwalking may sometimes be triggered by sleep disorders such as PLMD when subjects are partially aroused from deep slow wave non-REM sleep by their excessive limb movements.

4. PHENOMENA OCCURRING AT SLEEP-WAKE TRANSITIONS

Sleep starts (hypnic jerks) and related phenomena

The vast majority of people will recognise or recall infrequent mildly unsettling episodes of abrupt generalised 'jolts' occurring just at the point of sleep onset. The sensation is often likened to briefly 'falling through space'. Phenotypically, these myoclonic jerks are not stereotyped and may produce either flexion or extension movements of trunk and limbs. The jerks superficially resemble startle reflexes although, unlike these, asymmetry of limb movements is commonly observed. Generally, these wake-sleep transition phenomena are infrequent and more likely to occur in a sleep deprived young population, potentially aggravated by caffeine excess. If events are captured during sleep investigations, brief autonomic and EEG arousals usually follow the jerks but sleep onset occurs normally thereafter and there are no adverse clinical consequences.

Rarely, sleep starts may be frequent or dramatic enough to cause significant insomnia, especially if they recur through the night at each sleep-wake transition. Occasionally, myoclonic seizure activity may be mistakenly suspected. Anxiety secondary to the sleep starts may also fuel the subject's inability to fall asleep. Furthermore, a minority of subjects experience prominent auditory or even visual phenomena, occasionally in the absence of any motor phenomena or myoclonic movements. Sensory descriptions of 'bangs', 'shocks' or even 'explosions', usually in the head, are typical. Understandably, these can produce concern and require reassurances that they are not due to epilepsy or cerebral vascular pathology.

At a simplistic level, sleep starts probably reflect a temporary fault in the usually smooth transition from wake to sleep such that part of the brain fails to 'fall asleep' and an alerting brainstem mechanism is subsequently activated when this mismatch is detected. An interesting and somewhat bizarre likely related phenomenon, the 'blip syndrome', has been described in which subjects suddenly feel as though only part of their brain has lost consciousness when drowsy. This brief disconcerting phenomenon probably rarely comes to medical attention but may also reflect a partial failure of smooth transition from wake to light sleep.

Propriospinal myoclonus at sleep onset

The term 'propriospinal myoclonus' (PSM) was introduced in 1991 to describe non-rhythmic axial jerks causing symmetric flexion of the neck, trunk and lower limbs in three index cases. Detailed electrophysiology implied that the movements were caused by electrical discharges arising from a spinal 'pattern' generator with spread both rostrally and caudally via relatively slow (<5 m/s) pathways, presumed, from animal evidence, to be in the long propriospinal tracts. Subsequent reports of the phenomenon have emphasised the important roles of body position and behavioural state. In particular, PSM was observed to be much commoner when recumbent and extremely drowsy, at the point of sleep onset. Indeed, when the movements are confined to the wake-sleep transition, it can be considered a parasomnia, adversely affecting sleep onset and altering subsequent sleep structure. In this situation, it is commonly observed that mental activity, voluntary movements of the limbs and, indeed, sleep itself can all suppress the myoclonic movements. Rarely, as seen in other forms of myoclonus, PSM appears to be stimulus sensitive, triggered variously by sudden noises or external tactile stimulation to either the back or abdomen.

A variety of spinal cord pathologies including trauma have been reported to trigger or precipitate PSM, often with considerable delay in symptom onset after the presumed primary pathology. Although the majority of

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cases have normal conventional magnetic resonance (MR) spinal cord imaging, one recent review suggests that detailed diffusion tensor imaging may reveal subtle disorganisation of white matter tracts in the majority of cases.

Intensified hypnic jerks or sleep starts may mimic PSM, especially when they result in significant sleep-onset insomnia. However, they usually involve isolated body segments and do not primarily affect the abdominal musculature. Focal abnormal involuntary movements of the abdominal wall, variously described as 'moving umbilicus syndrome', 'belly dancer's dyskinesia', or 'diaphragmatic flutter', appear as different phenomena to PSM. In these, the affected muscles demonstrate irregular or writhing contractions at high rates of 30–90 per minute. Periodic limb movements (PLMs), although fairly frequently seen in relaxed wakefulness, invariably start in the distal musculature and, in stark contrast to PSM, predominate during non-REM sleep. However, when PLMs are associated with restless legs syndrome they may co-exist with PSM.

The surprising demonstration that the characteristic electromyogram (EMG) pattern of muscle activation ascribed to PSM can be identically observed in healthy volunteer subjects asked to mimic the axial myoclonic movements has led some to question the organicity of PSM in otherwise typical cases. The disappearance of PSM with mental distraction and sleep onset might also suggest a psychological aetiology.

Rhythmical movement disorder of sleep

Sleep-related rhythmic movement disorder (RMD) is a relatively poorly researched phenomenon that is most commonly seen at the wake-sleep transition although can occur in drowsy wakefulness or, surprisingly, given its nature, during any stage of sleep including REM sleep. Occasionally it can be seen in association with partial arousals from sleep. It is characterised by a variety of sustained stereotypic movements involving the major muscle groups. The cadence of movements is strikingly regular at a frequency of 0.5–2 Hz. Typical movements include head rolling or banging, axial rocking, and even rhythmical striking of the head with the hands. Some subjects exhibit more than one type of RMD, dependent on body position during sleep, with vigorous head rolling most commonly seen in the prone position. Synchronous humming or moaning is not uncommon although subsequent recollection of such activity is usually absent. It is relatively rare for RMD to cause major concern to the subjects themselves and, certainly, in adults, it is the bed partner whose sleep is predominantly disturbed, especially since the movements can continue for periods of 30 minutes or so.

The prototypical RMD, namely head banging, was first described in children in 1905 independently by German and French workers who coined the terms '*jactatio capitas nocturna*' and '*tics dans le sommeil*', respectively. Head banging is common and may affect up to 5% of young children. It is seen more often in males and is more persistent if there is developmental disability in which case similar rocking movements may also be witnessed during wakefulness. Indeed, many adult subjects may exhibit similar patterns of movement in quiet wakefulness such as when listening to music. Secondary injuries are very rare although soft tissue damage or dermal scarring has been reported. Body rocking is even more prevalent as a rhythmic movement of sleep (around 15% of young children) but is rarely perceived as a problem and usually resolves before five years of age. In children, an association of RMD with attention deficit hyperactivity disorder has been reported although sample sizes were small.

In a minority of subjects, RMD persists into adulthood although prevalence figures are difficult to establish as it is relatively rare for the phenomenon to present to a sleep clinic. Of interest, in adults, movements may be more varied and can be seen even in deep non-REM sleep or even REM sleep when all significant movements would normally be suppressed. The diagnosis is usually clear simply from a description, ideally with a video recording, without the need for detailed polysomnography. However, investigations may sometimes usefully demonstrate a degree of sleep disruption or an association of RMD with other sleep disorders such as periodic limb movements or apnoea-induced arousals.

The mechanisms behind RMD remain obscure although a variety of behavioural or psychological theories have been espoused, especially for childhood forms. A form of anxiety relief has been thought likely even if there is no overt psychopathology. Of possible relevance, RMD, like many sleep disorders, usually worsens during stressful periods. However, most authorities regard RMD as a 'tic-like' phenomenon or simply as a 'bad habit' which may be under partial voluntary control, at least in the early stages.

5. PHENOMENA NOT RELATED TO SLEEP STAGE

Sleep-related bruxism

Sleep bruxism (SB) is probably an exaggerated form of normal masticatory movements or 'chewing automatisms' commonly witnessed during sleep with additional tooth grinding as a potentially troublesome feature. Well over 50% of normal subjects will have episodes of rhythmic masticatory muscle activity 1–2 times per hour during sleep, defined as three or more consecutive rhythmic contractions at 1 Hz frequency.

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These contractions are sustained or phasic in which case the disturbing sounds of tooth-grinding occur. Although SB has a prevalence of around 8% in adults, many subjects are unaware that they grind their teeth at night and the bed partner is the main complainant. However, excessive tooth wear, dental or temporomandibular pain, and a strong association with migrainous and other headache syndromes are frequent observations. Of note, tooth-grinding has been seen in over 50% of children diagnosed with tension-type headaches.

Secondary SB has been associated with a wide variety of medications and drugs of abuse, including most psychoactive agents and central stimulants. Selective serotonin reuptake inhibitors (SSRIs) and haloperidol have been most strongly implicated. Hyperkinetic disorders such as generalised dystonia and Huntington's disease have also, not surprisingly, been linked to significant SB. Furthermore, SB tends to worsen when sleep patterns are non-specifically poor, either in the context of psychological distress or when there are frequent arousals due to obstructive sleep apnoea, for example. It is rare for SB itself to trigger a full arousal from sleep and it seems the jaw activity is simultaneous or secondary to EEG arousals. In general, SB also appears commoner in those with hypervigilant or highly motivated personalities. It is not infrequently picked up incidentally on polysomnographic recordings as a 1 Hz interference or movement artefact seen throughout the EEG traces.

Hypnagogic foot tremor

Hypnagogic foot tremor and the related phenomenon alternating leg muscle activation represent benign movement disorders commonly witnessed in subjects who are drowsy or in light sleep. Although often overlooked, regular dorsiflexion of either one foot or both feet in alternation with a frequency of 1–2 Hz may be seen as incidental findings in around 5% of polysomnograms, usually around the sleep-wake transition. Rarely, these movements may disturb the bed partner but usually they are viewed as somewhat curious or even amusing phenomena, potentially similar in nature to rhythmical movement disorders of sleep, simply requiring reassurance and no active treatment.

6. NOCTURNAL SEIZURES

Not infrequently, patients with established epilepsy exhibit seizures from the state of sleep. There is often no clear pattern or link to any particular sleep stage although seizures from REM sleep seem extremely rare. If the events reflect generalised tonic-clonic seizures, the diagnosis is usually clear either from witnessed descriptions or paraclinical features such as tongue biting, incontinence and musculoskeletal pain or stiffness on awakening, following events. Diagnostic difficulty most commonly arises if the seizures are complex partial in nature, especially if arising from the frontal lobe. Indeed, frontal lobe epilepsy (FLE) may occur exclusively from sleep and can sometimes run in families with established mutations of (nicotinic) cholinergic receptors. If a witnessed description of the events and their timing is available, a confident diagnosis of nocturnal FLE can usually be made, although video analysis, even recorded in the home environment, is often invaluable. Non-REM sleep parasomnias may mimic FLE but, if detailed information on the nature and pattern of events is available, distinction is usually possible without recourse to prolonged overnight polysomnography (Table 1).

The most valuable discriminatory features of nocturnal FLE are their frequency (often several a night), their precise stereotypical nature, and the motor features that most often occur at seizure onset (typically, dystonic posturing of an arm or head turning). The behaviours that accompany FLE are often complex and bizarre, including leg cycling, head shaking and truncal rhythmical movements. The attacks may appear to evolve in a precise stereotypical fashion. Partial awareness and responsiveness may be seen and, paradoxically, in contrast to non-REM sleep parasomnias, the offset of seizure activity occurs quickly with no obvious sequelae. Unlike non-REM parasomnias, episodes of FLE are very unlikely to last more than two minutes.

Certain features of FLE may be shared with types of non-REM sleep parasomnia and are not considered to be diagnostically discriminatory. These include emotionally charged or aggressive behaviour and ambulation.

CONCLUSION

Sleep is not simply the absence of wakefulness and some movement during the night is clearly within a spectrum of normality. Occasional minor limb or body jerks, shifting of position every 20 minutes or so and even sleep talking are considered normal variants. However, a considerable array of movements and behaviours from the state of sleep may be vigorous or disturbed enough to be considered abnormal and worthy of a diagnostic label. Although our understanding of underlying mechanisms and treatment options remains rudimentary, the classification of sleep-related movement disorders has evolved. Perhaps surprisingly, the vast majority of such phenomena can be diagnosed with reasonable precision without recourse to sophisticated and expensive investigations, provided an adequate history is available. Sleep remains a highly mysterious state but its disorders are increasingly recognised and occasionally understood.

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SENIOR FELLOWS' CLUB PRIZE

The Senior Fellow's Club Prize for 2013 has been won by B Dobb for the paper on 'A pilot survey of decisions by acute medicine staff after thunderclap headache'. This paper can be read in issue 3, 2013 at http://www.rcpe.ac.uk/sites/default/files/Dobb.pdf

A prize of £250 will be awarded to the first-named (or corresponding) author of an original research paper on a clinical topic, deemed by a panel of judges to be the best paper by a doctor-in-training (i.e. pre-consultant level) published in *The Journal of the Royal College of Physicians of Edinburgh* in 2014. The best paper will be selected by a panel of judges, including a senior Fellow, an active clinician and a member of the editorial team.

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