Chapter 4
Diagnosing Parasomnias

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Introduction

Parasomnias are undesirable or unusual motor, behavioral, and/or experiential events that occur predominantly during (or in the transitions from and to) sleep [1]. If violent or dangerous, parasomnias can injure the patient (or those who attempt to intervene), and when frequent, they can disrupt the sleep/wake schedules and daytime functioning of the patient, bed partner, and/or family. Many parasomnias are impaired sleep state synchronization or "state dissociation" disorders [2, 3]. Transitions between wakefulness, non-rapid eye movement (NREM) sleep, and rapid eye movement (REM) sleep usually occur smoothly and completely, but when more gradual or oscillating rapidly, the physiological markers of one sleep state can linger or intrude into another [2].

A prototypical state dissociation disorder is narcolepsy with cataplexy in which (1) cataplexy is the sudden onset of REM sleep atonia while awake in response to an emotion-laden event; (2) sleep paralysis is an early or lingering appearance of REM sleep atonia, and (3) hypnic hallucination fragments of REM sleep dreams persist into wakefulness. Other state dissociations include sleepwalking (SW), sleep terrors (STs), REM sleep behavior disorder (RBD), sleep paralysis, and sleep inertia. Incomplete declarations of sleep/wake state of one sleep/wake state into another may also explain out-of-body experiences [4], lucid dreaming [5, 6], visual hallucinations in patients with Parkinson's disease (PD) [7], and alien abduction [8].

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S. V. Kothare, A. Ivanenko (eds.), *Parasomnias,*
DOI 10.1007/978-1-4614-7627-6_4,

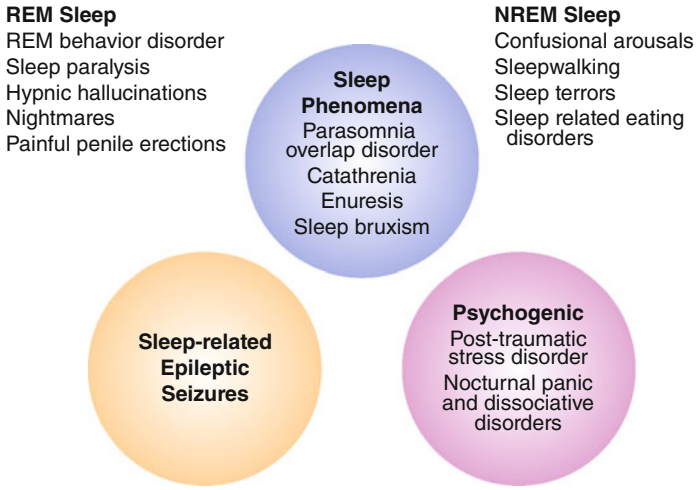

Fig. 4.1 Wide range of parasomnias that sleep specialists encounter

Diagnosing Parasomnias

When asked to diagnose paroxysmal events or abnormal movements during sleep, begin by asking whether these only occur around or during sleep [9]. If these also occur when awake, consider whether the patient has a movement disorder when awake. Contrary to older teachings, most diurnal movement disorders (including tremor, dystonia, chorea, hemiballismus, and myoclonus) persist or intermittently recur in sleep (albeit more intermittent and reduced in frequency and duration than when awake).

Then obtain a detailed description of the events regarding (1) stereotyped or variable; (2) consciousness is preserved before, during, and/or after them; (3) number and time(s) of occurrence related to sleep onset; (4) precipitating factors; (5) recall of the events; (6) potential to cause injury; (7) daytime consequences of them including cognitive slowing or daytime sleepiness; and (8) sleep/wake habits of the individual searching for irregular sleep/wake schedules and partial sleep deprivation.

Next obtain thorough medical, sleep, social, and family histories, specifically asking whether they have a prior personal and/or family history of parasomnias or sleep/wake problems. A physical examination should follow, searching for a movement disorder when awake, dementia, confusion, depression, anxiety, upper airway or body habitus at risk for obstructive sleep apnea (OSA), and underlying cardiac, pulmonary, neurodegenerative, or peripheral nerve disorders.

Based on the clinical descriptions provided by observers (often unreliable because they are aroused after the onset of an event), determine whether the nighttime movements are simple or complex. Box 4.1 summarizes the differential diagnosis of paroxysmal nocturnal events (PNEs) referred to sleep specialists for diagnosis. Figure 4.1 shows the wide range of parasomnias that sleep specialists encounter. Figure 4.2 provides a flow chart for assessing parasomnias on the basis of their time of occurrence and most salient clinical feature.

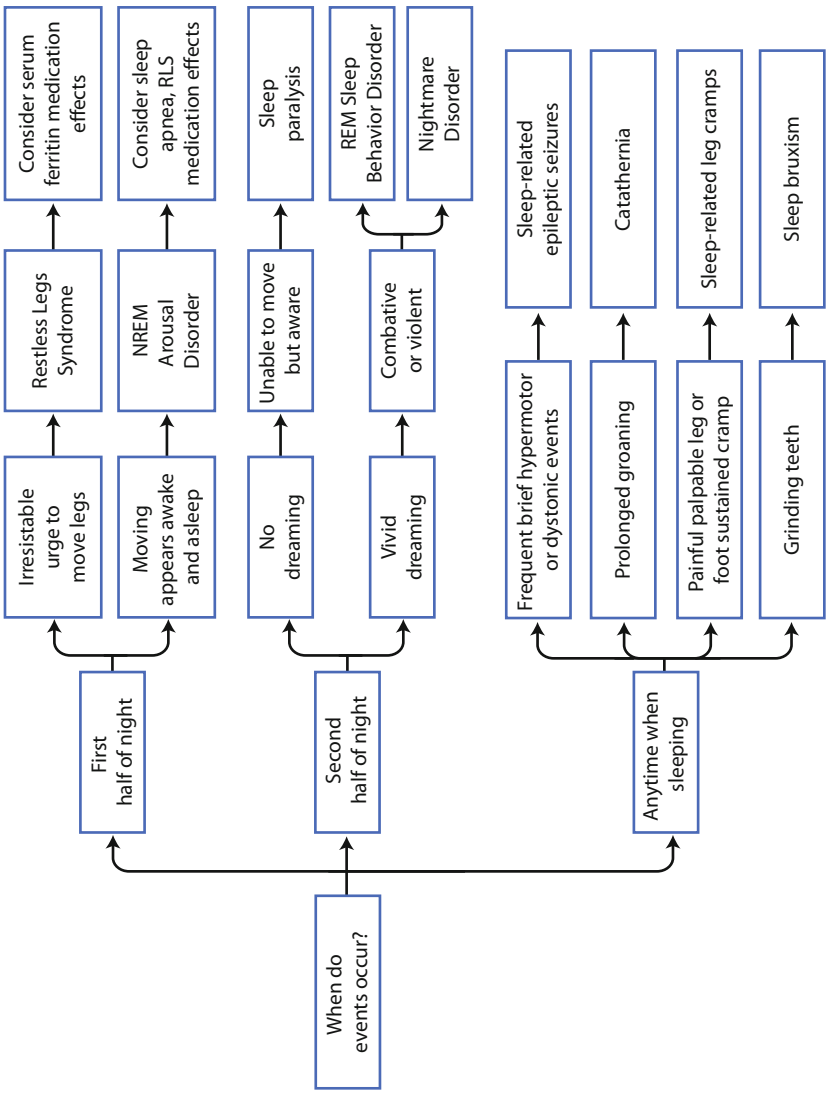

Fig. 4.2 Flow chart for evaluating parasomnias according to the time of occurrence and salient clinical features

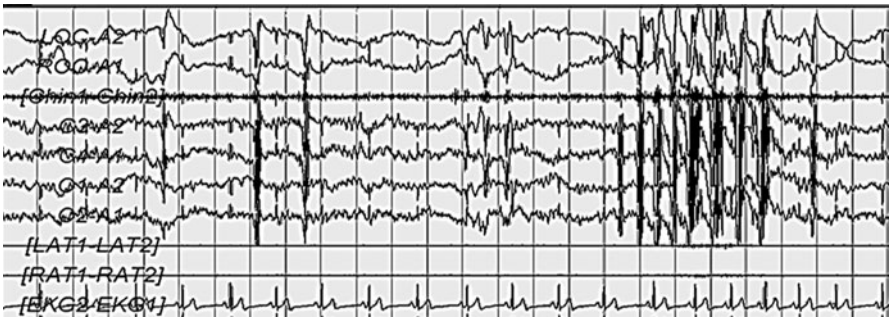

Fig. 4.3 Sleep bruxism

Age-Related Prevalence and Risk Factors for Parasomnias Contribute to Our Diagnostic Expectations

Parasomnias are most common in children and decrease in prevalence with increasing age [10]. Approximately 85 % of children between the ages of 30 months and 6 years talk in their sleep, 46 % have sleep bruxism (SB), 40 % have STs, 25 % have nocturnal enuresis, 15 % sleepwalk, and 9 % exhibit head banging or body rocking. Forty percent of 6- to 16-year-old children have at least one episode of an NREM arousal parasomnia (disorders of arousal (DoA)) (most between ages 11 and 12 years) [10]. However, only 2–3 % of children have more than one DoA per month. The majority of children who sleepwalk stop SW by age 13 years, but it persists in 24 % of frequent sleepwalkers. If one parent has a history of sleepwalking, the risk of a child developing sleepwalking behaviors is 40–45 %; if both parents are affected, then the risk of a child developing this behavior increases to 60–65 %. Somniloquy (sleeptalking) occurs in 50–80 % of children, but in half the cases, sleeptalking occurs only a few times a year. Ten percent of children with sleeptalking, talk nightly. The risk for sleeptalking increases with stress, sleep deprivation, alcohol, and fever, and in those with other primary sleep disorders (DoA, sleep-disordered breathing (SDB), and/or RBD).

SB is another parasomnia that is much more prevalent in children and tends to persist in a larger percent of adults than other parasomnias. SB occurs in one-third of children, 8 % of young and middle-aged adults, and 3 % of the elderly. Figure 4.3 shows an example of SB. Major risk factors for SB in adults include emotional stress, tobacco or caffeine use, heavy alcohol use, type A personality, and other sleep disorders such as sleep apnea or periodic leg movements in sleep (PLMS). SB is probably an extreme audible expression of rhythmic masticatory muscle activity (RMMA). RMMA occurs in most normal subjects when sleeping, and grinding of teeth during sleep probably represents a central pattern generator emerging during sleep.

Adults are far less likely to sleepwalk. A recently published large prospective cross-sectional study of 19,136 adults in the general US population found 3.6 % reported “nocturnal wandering” (NW within the previous year, but only 1 % had ≥ 2

episodes per month [11]. NW in adults tended to be a chronic condition: 81 % reported having episodes of NW for more than 5 years. A family history of NW was reported by 31 % of participants who reported NW. Other risk factors for frequent NW (≥ 2 per month) were OSA (odds ratio [OR] 3.9, circadian rhythm disorder (OR 3.4), insomnia disorder (OR 2.1), alcohol abuse/dependence (OR 3.5), major depressive disorder (OR 3.5), and obsessive compulsive disorder (OR 3.9) using over-the-counter sleeping pills (OR 2.5) or selective serotonin reuptake inhibitors (OR 3.0).

Chronic RBD is a complex parasomnia characterized by abnormal and often violent motor behaviors and complex vocalizations in which patients appear to enact their dreams while in REM sleep [12]. Chronic RBD usually presents after age 50 years, although any age group can be affected [13]. Chronic RBD is much more common in men [14–17]. RBD is also common in individuals with narcolepsy-cataplexy [18–20]. RBD can be misdiagnosed as sleep-related epilepsy, agitated SW, nocturnal panic attacks, nocturnal hallucinations, agitated delirium in intensive care units, sundowning, and/or intentional spouse abuse.

RBD can be idiopathic or secondary. Secondary RBD can be related to neurodegenerative disorders, other neurological disorders, sleep disorders, or medications, including withdrawal states [21]. Chronic symptomatic RBD in older adults is most often associated with an α-synucleinopathy, which manifests as either dementia with Lewy bodies (DLB), PD, or multiple system atrophy (MSA) [16, 22–31]. RBD is often the *first* clinical sign of an α-synucleinopathy, preceding other early nonmotor signs (olfactory dysfunction and depression) of parkinsonism and/or dementia by years or decades [17, 16, 32–34]. Prospective studies found that 38–65 % of patients with idiopathic RBD (iRBD) followed longitudinally subsequently developed a α-synucleinopathy most often 7–13 years after the onset of RBD [16, 34–36]. The 5-year risk for developing a neurodegenerative disease in patients with iRBD was 18 %, increasing to 41 % and 52 % at 10 and 12 years, respectively [36].

The challenging clinical problem is to identify which older adult with iRBD will develop a neurodegenerative disease (in case preventative treatments were available). Early biomarkers found in many patients with iRBD before signs of parkinsonism and/dementia develop that may increase the likelihood that they will later develop a neurodegenerative disease include impaired olfaction [37]; greater echogenicity of the substantia nigra [37]; impaired decision making [38]; markedly reduced sympathetic cardiac ^{123}I-MIBG (metaiodobenzylguanidine) uptake [39]; significant gray matter volume reduction on brain MRI in the anterior lobes of cerebellum, tegmental pons, and left parahippocampal gyrus [40]; and decreased regional cerebral blood flow in the precuneus, limbic lobe, and cerebellar hemispheres [41].

Some patients with dream-enactment behaviors and unpleasant dreams are found to only have severe OSA ("pseudo-RBD"); the abnormal behaviors observed in both REM and NREM sleep were not associated with REM sleep without atonia (RSWA), and continuous positive airway pressure (CPAP) therapy eliminated the abnormal behaviors, unpleasant dreams, daytime sleepiness, and snoring [42]. RBD has also been observed in 10–15 % of patients with narcolepsy with cataplexy [18, 43, 44] and RSWA without clinical RBD in others [43]. RBD in patients with narcolepsy needs to

be distinguished from SW and periodic limb movements during sleep (PLMS) and abnormal dreaming, all more common in these patients. Medications prescribed to treat their cataplexy can induce or aggravate RBD [44–46]. One study found epilepsy coexisting with RBD in 13 % of 80 older adults with epilepsy [47]. Suffice it to say, separating these out can be challenging. RBD and RSWA when seen in children, adolescents, or younger adults warrants consideration of narcolepsy with cataplexy, Tourette's syndrome, medication effect, or an autoimmune or limbic encephalitis [48, 49].

Parasomnias Common in Normal Individuals

Four particularly common parasomnias occur in normal individuals and most often require only reassurance of their usually benign nature. These are sleep starts, hypnic hallucinations, sleep paralysis, and rhythmic movement disorder (RMD). Sleep starts (also known as hypnic jerks) occur in the wake to sleep transition with one or two abrupt myoclonic flexion jerks often accompanied by a feeling of falling, a sensory flash, and/or dream-like imagery. Sleep starts occur occasionally in 70 % of the adult population and are associated with insufficient sleep.

Hypnic hallucinations are vivid perceptual experiences, which most often occur at sleep onset (95 % of the time), but also occur rarely upon awakening. Most people report a sensation of hearing voices or feeling someone else is nearby. Approximately 70 % of people have experienced these symptoms and are more frequent and severe in those with irregular sleep/wake schedules or narcolepsy with cataplexy. Excessive caffeine or other stimulant use, intense work or exercise, and emotional stress can increase the intensity of hypnic hallucinations.

Sleep paralysis is a transient inability to move despite being fully awake during a transition between sleep and wakefulness. It represents a brief persistence of the skeletal muscle motor suppression of REM sleep lingering into wakefulness. It occurs most often and with greatest frequency in individuals who have narcolepsy with cataplexy, particularly in the transition from REM sleep to wakefulness. Fewer than 10 % of adults, but as many as 40 % of teens and college students, have had at least one episode of sleep paralysis, most often triggered by sleep deprivation. Isolated sleep paralysis without other features of narcolepsy also runs in families [50].

RMD are episodes of rhythmic head banging, body rocking, or leg rolling (often accompanied by humming or chanting), which usually occur just before sleep onset, but may persist into NREM 1 or 2 sleep, and rarely recur in REM sleep [51–53]. Nearly 66 % of 9-month-old infants (both neurodevelopmental normal and abnormal) exhibit RMD, but the prevalence falls to 8 % at age 4 years. RMD that persists in later childhood is more likely to occur in children with neurodevelopmental, psychiatric, and/or autism spectrum disorders. Sleep specialists every few years encounter an adult with RMD, most have had it since childhood, but rare cases report it beginning following head injury or encephalitis. A case series of 24 adults with RMD found that RMD first began in childhood in all cases, and a family history was rare [51]. In some, OSA triggered arousals with a recurrence of their movements. Figure 4.4 shows a polysomnography (PSG) of a 40-year-old man with RMD.

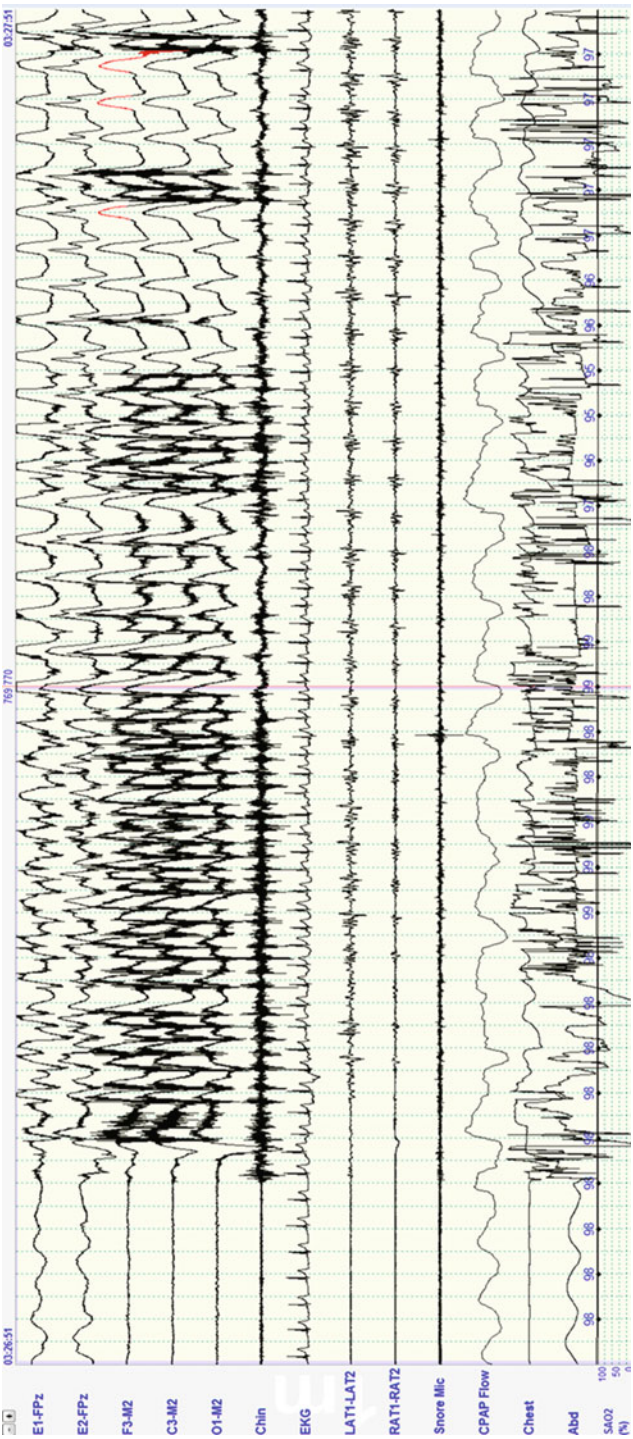

Fig. 4.4 Rhythmic movement disorder

Another common RMD is hypnagogic foot tremor (HFT), which is a rhythmic foot movement found in 5 % of healthy adults. It may involve one or both feet. Figure 4.5 shows a PSG of a 52-year-old subject with symptoms of HFT since early adulthood. HFT is a benign finding that rarely disturbs the patient but may disturb the patient's partner.

Indications for Video-Polysomnography When Evaluating Parasomnias

Clinical practice parameters published by the American Academy of Sleep Medicine (AASM) recommend video-polysomnography (video-PSG) to evaluate PNEs that are (1) unusual or atypical because of age of onset, time of night, duration, or particular accompanying motor behaviors (e.g., stereotyped, repetitive, dystonic, or focal); (2) frequent ($\geq$ 2–3 nights per week); and (3) potentially injurious, and/or disruptive to the patient or family. Video-PSG is warranted to differentiate atypical nocturnal behaviors from nocturnal seizures, to help identify when OSA or other sleep disorders are causing or contributing to frequent parasomnias, enuresis, or to control epileptic seizures [1, 54].

A PSG is unnecessary if the nocturnal behavior events in a *young* child are typical, noninjurious, infrequent, and not disruptive to the child or family [1, 54]. Common, uncomplicated, noninjurious parasomnias (such as SW, STs, sleeptalking, nightmares, bruxism, and enuresis) can usually be diagnosed by a clinical history [1, 54]. Typical NREM 3 arousal disorder features in a young child, which usually do not need a PSG for diagnosis, are summarized in Box 4.2.

One caveat, though: STs or SW events that are unusually frequent (occurring more than 2–3 times per week) warrant PSG to identify another sleep disorder precipitating them (most often OSA, occasionally periodic limb movement disorder). OSA was found on overnight PSG in 58 % of 84 prepubertal children who had STs and/or SW, restless legs syndrome (RLS) with PLMS in two [55]. Tonsillectomy in 43 eliminated both the OSA and parasomnias. Two had RLS, and its treatment with pramipexole eliminated their confusional arousals, restless legs, and PLMS.

Video-PSG is usually warranted in adults with suspected DoA, which begin or recur in adulthood, occur more than 2–3 times per week, are potentially injurious, could be seizure-related but the initial clinical evaluation and a standard electroencephalogram (EEG) inconclusive, and/or are accompanied by symptoms suggestive of SDB, periodic limb movement disorder, suspected or known epilepsy, and/or excessive daytime sleepiness. Video-PSG in adults with frequent SW/STs often identifies concomitant OSA, occasionally PLMs. On occasion, the PNE proves to be sleep-related epilepsy.

On occasion, video-PSG is requested to evaluate forensic cases where parasomnias and/or other sleep disorders or drugs are a contributing factor [56–59]. Violent behaviors arising from sleep are reported by 2 % of the adult population [60]. Violent or potentially injurious parasomnias include DoA, RBD, sleep-dissociative disorders, and nocturnal seizures with postictal confusion. A single video-PSG is likely to

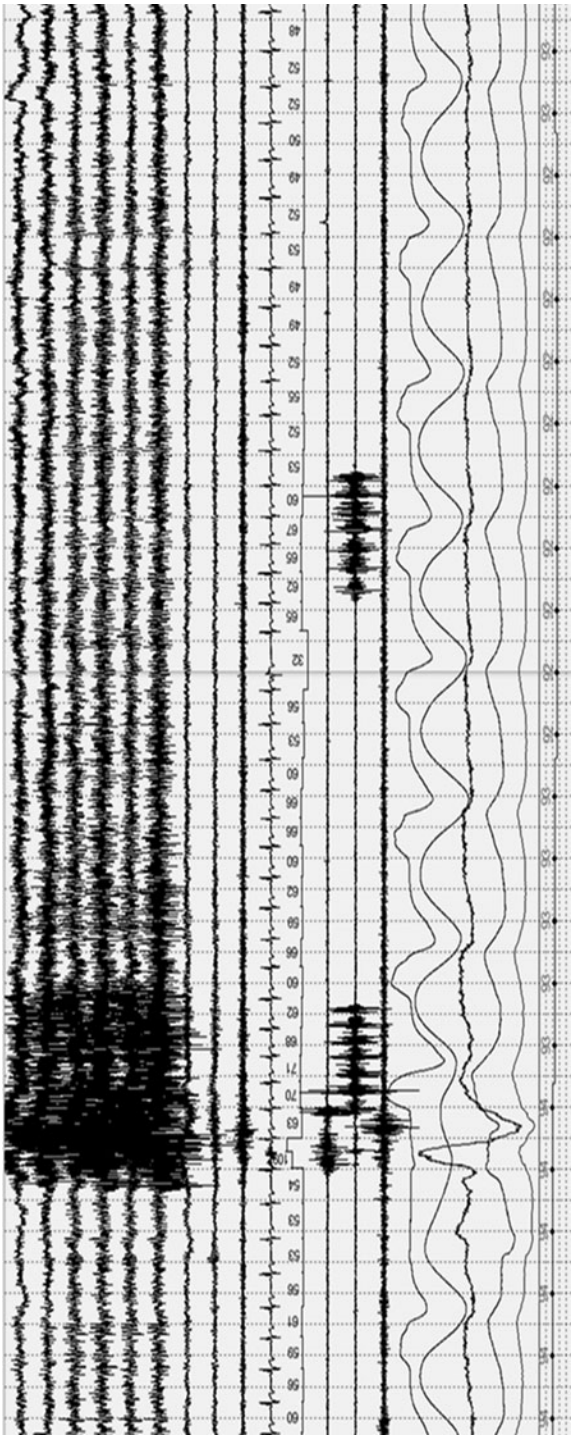

Fig. 4.5 Hypnagogic foot tremor

identify RBD and OSA and less likely to capture a DoA or sleep-dissociative event. However, whether the event(s), which prompted the evaluation, were triggered by the parasomnia or sleep disorders often remains uncertain. Box 4.3 summarizes guidelines proposed to assist in determining the putative role of an underlying sleep disorder in a specific violent act [56].

Diagnostic Algorithm for Evaluating Suspected Sleep-Related Seizures

If you suspect sleep-related seizures and the patient has not had a routine outpatient EEG, request one. However, epileptiform abnormalities are far more likely to be found on routine EEG in children than adults with epilepsy. Epileptiform activity will be found on the first routine EEG in approximately half of the children with clinically diagnosed epilepsy [61–63] but in only 3 % of the studied US veterans (mean age 55 years, range 21–97 years) [64].

Many studies have been published over a half century debating whether partial () or total sleep deprivation increases the likelihood that interictal epileptiform discharges (IEDs) will be found in a routine EEG. Some argue that sleep deprivation does not offer greater activation than sleep alone, whereas others assert total sleep deprivation activates IEDs. We agree with investigators who argue that sleep deprivation only increases the likelihood that NREM sleep will be recorded. Confirming this view, a prospective study of 820 EEGs recorded in children showed the following: (1) NREM sleep was observed in 57 % of sleep-deprived, 44 % of partially sleep-deprived, and 21 % of non-sleep-deprived pediatric EEGs; (2) a sixfold increased yield of recording NREM 2 sleep with sleep deprivation and 2.8-fold increase with partial sleep deprivation; (3) the OR that IEDs would be found was *not* increased by the presence of sleep or the use of total or partial sleep deprivation; and (4) the *only* significant effect of sleep deprivation was to increase the odds that sleep would occur [65]. Sleep is likely to occur with only partial sleep deprivation [66]. A better mix of sleep yield and cost containment is to request a child older than 2 years to stay awake 2 h later than usual the night before the EEG, performing sleep-deprived EEGs in the morning, and request children younger than 2 years to have no naps on the day of the EEG.

If sleep is not easily obtained in a routine outpatient EEG in a child, consider oral melatonin as a sedative (since conscious sedation with choral hydrate is rarely done now). A recent study found that melatonin induced sleep in 80 % of 70 children within a mean of 25 min [67]. We are far less concerned about sleep deprivation in adolescent or adult. A sufficient number of studies in adults provide evidence that total sleep deprivation remains an easy and cost-effective strategy to increase the likelihood of recording IEDs [68–70].

If the routine outpatient EEG obtaining sleep is nondiagnostic and the child's paroxysmal events are daily (even if only in sleep), consider a 4- or 8-h daytime recording in the EEG laboratory (with partial sleep deprivation or sleep deprivation the night before). Recording of 4–8 h in 230 children captured and confirmed the

nature of the paroxysmal events in 80 % of children whose events occurred on a daily basis [71].

If a patient's spells occur only at night and are frequent (2–3 times per week, better yet 1–3 times per night), consider ordering a video-PSG with expanded EEG before prolonged inpatient video-EEG monitoring, especially if concomitant OSA or RBD is suspected. NFLE is one of the few partial epilepsies that sleep specialists may confirm by a single night of video-PSG because patients with NFLE tend to have multiple seizures. Patients with NFLE from the same case series mentioned earlier averaged 3 ± 3 (range 1–20) seizures per night of video-PSG and a mean of 20 ± 11 seizures per month (61 %, > 15 seizures per month) [72].

If the first (or second) routine video-EEG with sleep is normal and your clinical suspicion for a sleep-related epilepsy remains, request continuous inpatient video-EEG monitoring (long-term monitoring (LTM)) for 2–5 days when (1) the nocturnal behaviors do not occur nightly or every other night; (2) a primary sleep disorder (e.g., OSA or childhood RLS) is unlikely; (3) a history exists of postictal agitation or wandering, and/or (4) cooperation of the patient is questionable. The likelihood of recording the typical epileptic or nonepileptic paroxysmal events in LTM ranges from 45–96 % if the patient is having ≥ 1 event per week [71, 73, 74].

Technical Considerations and Challenges of Video-Polysomnography to Diagnose Parasomnias

The AASM practice parameters recommend the following for video-PSGs done to diagnose parasomnias: (1) "additional EEG derivations in an expanded bilateral montage" to diagnose paroxysmal arousals or other sleep disruptions thought to be seizure-related when the initial clinical evaluation and results of a standard EEG are inconclusive; (2) recording surface EMG activity from the left and right anterior tibialis and extensor digitorum muscles; (3) obtaining good audiovisual recording; (4) having a sleep technologist present throughout the study to observe and document events; and (5) sleep specialists who are not experienced or trained in recognizing and interpreting both PSG and EEG abnormalities should seek appropriate consultation or should refer patients to a center where this expertise is available [1].

It is probably best to record at least 18 channels of EEG during video-PSG despite the only two published studies evaluating how many channels of EEG are needed to identify seizures in a video-PSG found that recording 18 channels of EEG during video-PSG did not improve the ability to recognize frontal lobe seizures [75, 76]. The ability to recognize frontal lobe seizures by EEG alone was not helped by more EEG channels, slower screen times, or midline electrodes. Temporal lobe seizures during sleep were most likely to be identified if EEG channels were placed over the temporal regions.

We sometimes encounter unanticipated IEDs on a PSG. Determining whether epileptiform activity is unilateral, bilateral, asymmetric, or shifting left to right is far easier if we remap the standard PSG EEG montage referencing the ipsilateral

electrode to the contralateral mastoid (e.g., F4-M1, F3-M2, C4-M1, C3-M2, O2-M1, O1-M2 shown in Fig. 4.6a) to the ipsilateral mastoid (i.e., F4-M2, F3-M1, C4-M2, C3-M1, O2-M2, O1-M1 in Fig. 4.6b). Routinely recording the alternative EEG derivations (Fz-Cz, Cz-Oz) can be useful for detecting electrographic seizure activity over the CZ in nocturnal frontal lobe epilepsies because artifact is often least there. Scoring sleep studies in patients with epilepsy can be difficult especially when IEDs are frequent, even more difficult when their sleep spindles are of low amplitude or dysmorphic, and/or when inappropriate alpha intrusions are present [77].

Patients with RBD characteristically exhibit excessive tonic activity in their chin electromyogram (EMG) and/or excessive phasic activity in their chin and/or limb EMG. In routine PSG, we only record EMG from the chin and anterior tibialis leg muscles. We add surface EMG electrodes to the wrist extensors when recording patients with suspected RBD [1, 78–80]. The excessive motor activity and RSWA typical of RBD will usually be found in a single night of PSG. A retrospective analysis of video-PSG recorded in 55 adults with RBD who have at least two consecutive video-PSGs found that RBD could be diagnosed in 95 % of patients by recording and carefully analyzing the amounts of REM-related EMG activity, RSWA, and motor events observed on video-PSG [81]. No significant difference in the amounts of phasic and tonic EMG activity during REM sleep between nights 1 and 2 were found, but dream-enactment behaviors were most susceptible to night-to-night variability.

The habitual nocturnal event may not be captured by one night of in-laboratory video-PSG, particularly if the events are DoA [80, 82, 83]. Investigators from Montreal were able to trigger 1–3 SW/ST events in all 10 adults with SW/STs by having them remain awake for 25 h and repeatedly ringing a loud buzzer (most often 40–90 db) during NREM 3 sleep. *However,* clinical confirmation of RSWA and/or RBD may be missed by a single night of video-PSG.

Video-Polysomnographic Features of the Parasomnias Most Often Encountered in the Sleep Laboratory

The parasomnias most likely to be recorded in a sleep laboratory include DoA, RBD, and sleep-related hypermotor seizures (usually nocturnal frontal lobe epilepsy).

NREM Arousal Disorders

DoA most often occur 90–180 min after sleep onset in the transition from NREM 3 (occasionally NREM 2) sleep to wakefulness or REM sleep [84, 85]. They typically last for a few minutes, are nonstereotyped, and can be provoked by sensory stimuli (OSA, a loud noise, or bright light half-awakening the patient). During these events, the patient appears confused, disoriented, and is slow to respond. Their eyes are open (as opposed to closed during RBD or NFLE); visual inspection functions but objects are often misidentified (e.g., trying to use the bedside water glass as a telephone

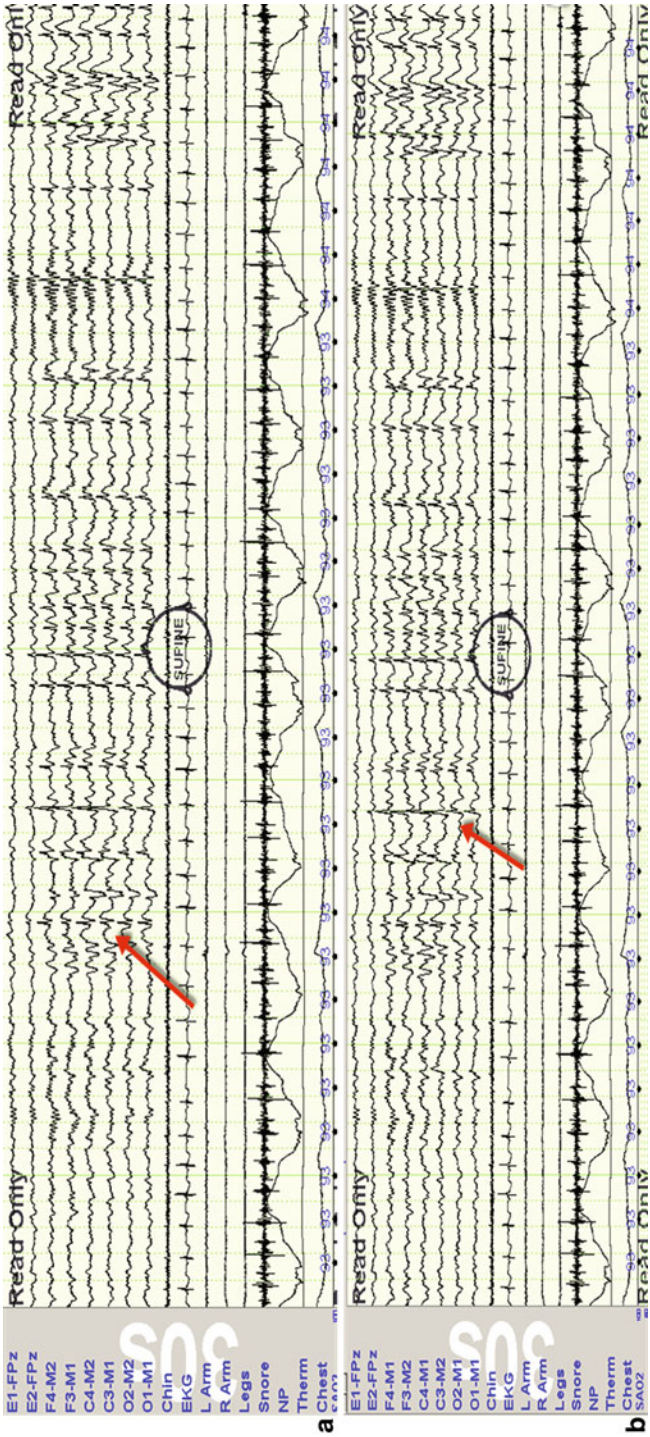

Fig. 4.6 **a**, **b** Unexpected rolandic spikes in NREM 2 sleep in a 7-year-old child

receiver, the closet as a bathroom). They have little or no responsiveness to their external environment and exhibit automatic behaviors. They are difficult to arouse from an event, and if aroused, they recall only fragmentary dream-like images (often of being trapped or attacked). The onset of DoA event is best identified by the appearance of tachycardia from NREM 3 sleep: the acceleration of the heart rate is typically greatest for an ST or agitated sleepwalking, moderate for confusional arousal, and least for passive SW.

During a confusional arousal, the patient often suddenly sits up in bed, may then fumble with bedclothes, thrash, flail or kick, moan, whimper, and/or utter often unintelligible words. Passive SW often begins as a confusional arousal, but the patient leaves the bed, walking toward a sound, light, or a particular room. While SW, the person may eat, urinate in a closet or next to the toilet, or walk outside. Sleep-related eating and sexual behaviors (often atypical from the individual's usual behavior when awake) have also been reported [57, 86].

STs and agitated SW often begin with a blood-curdling scream or cry, the patient exhibiting severe agitation, greater fear, more vocalization, and marked sympathetic arousal with mydriasis, tachycardia, tachypnea, and sweating. They flee their bed screaming or crying, run through the house, down the stairs, or out the front door. They may recoil and have increased agitation when touched or held; innocent attempts by bystanders to touch or direct them may then lead to injury (to themselves or others). Agitated sleepwalkers are more often adolescents or adults. Even violent DoA usually last a few to rarely as long as 30 min, often followed by a calm return to bed or sleep somewhere else in the house or outside. Episodes end with a return to sleep and retrograde amnesia for the events (although some adults can recall fragments of some events). Violent DoA in adults can cause injury to a patient or bedpartner, and self-injury during a DoA may be misdiagnosed as suicide [87].

EEG during a DoA event in 38 adult sleepwalkers (mean age 29 years, 55 % men) was characterized by either regular rhythmic hypersynchronous delta or theta activity, or high amplitude delta intermixed with alpha or beta activity [85]. Figure 4.7 shows a confusional arousal recorded from NREM 3 sleep in a 7-year-old boy. Studies comparing sleep microarchitecture and EEG power in adults with SW or STs with controls report that patients with SW/STs have (1) increased number of brief arousals from NREM 3 sleep especially during the first NREM cycle of a night; (2) reduced delta power of the slow wave activity especially during first NREM cycle; (3) slower decay of EEG delta power of NREM 3 sleep across recurring cycles of NREM sleep; and (4) alterations in cyclic alternating pattern during NREM sleep consistent with increased NREM 3 sleep instability. More work is needed to see if individuals with DoA can be identified by abnormalities in their sleep microarchitecture.

REM Behavior Disorder

RBD episodes usually appear in the first 90 min after sleep onset, typically last 1–5 min, and recur 3–5 times at 90–120 min intervals across an entire night of sleep during recurring periods of REM sleep. As opposed to DoA, patients with RBD

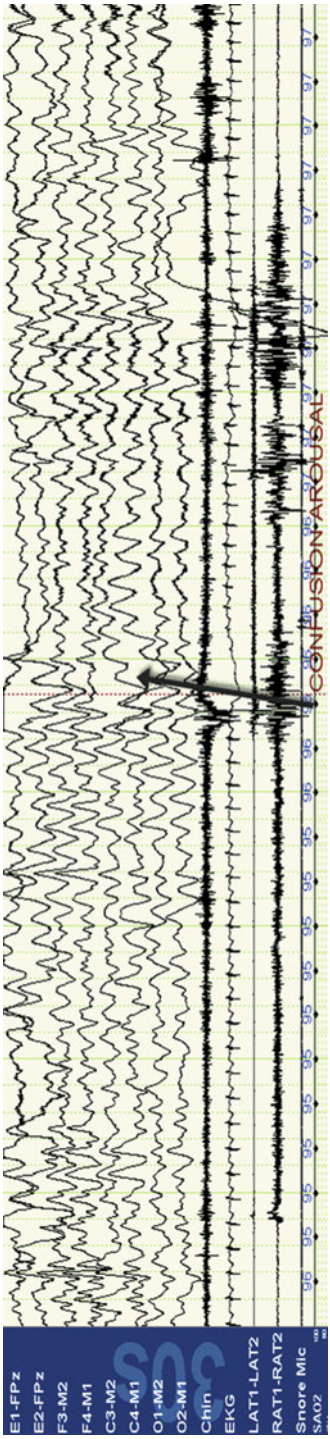

Fig. 4.7 Confusional arousal from NREM 3 sleep in a 7-year-old boy

are easily aroused from an event. Once aroused, they are able to recount dreams (if not too demented) that correspond to the observed behaviors [88, 89]. Their eyes are typically closed during events. Their heart rates most often do *not* increase during RBD events. A recent study found that heart rate responses were reduced in patients with RBD associated with parkinsonism and iRBD compared with controls [90]. Impaired heart rate responses probably represent an early loss of autonomic function.

RBD behaviors are often more plentiful and severe at the end of the night when REM sleep is most plentiful [91]. Paroxysmal motor RBD behaviors were more likely to occur in phasic portions of REM sleep (when rapid eye movements and saw tooth waves are seen) rather than in tonic REM sleep [92]. A case-control study of five PD patients with RBD found that limb jerking was the most common behavioral expression of RBD [78]. Motor behaviors during RBD dream-enactment events are much more frequent than vocalizations [91].

RBD motor behaviors can be simple (talking, shouting, excessive jerking of limbs or body) or complex (arm flailing, slapping, kicking, sitting up, leaping from bed, running, crawling, gesturing, swearing) [93]. A case-control video-PSG study found that 75 % of RBD motor events lasted < 2 s, 83 % were simple, 14 % complex, 11 % associated with vocalizations, and 4 % violent in the patient with RBD. RBD averaged 54 ± 23 limb movements per 10 min of REM sleep compared with 4 ± 2 per hour in healthy age- and gender-matched controls. Because RBD behaviors are typically brief, we recommend carefully reviewing the video when RSWA is most intense.

Motor behaviors during REM sleep in the control subjects were usually simple (91 %). Motor movements during RBD tend to be repetitive, quick, jerky, not self-centered, and rarely involve the environment in an appropriate manner. A specific posture of the hand (limp wrist with flexed digits) during grasping movements was seen in video-PSG of 48 % of 65 RBD patients [94]. The motor behaviors of RBD behaviors were similar whether the patient had PD, narcolepsy, or iRBD [94]. Patients with RBD can also enact nonviolent dreams: singing, dancing, saluting, marching, clapping, or snapping their fingers [95]. Speech in RBD events can vary from mumbling to logical sentences. De Cock et al. found that 38 % of 53 PD patients moved much better and had louder, more intelligible speech during their RBD episodes than when awake [96].

RBD is also regarded as a dream disorder: patients report that the content of their dreams become increasingly violent and disturbed. Their dreams often involve frighteningly unfamiliar people or animals, confrontation, attacking or chasing themes, and the behaviors often depict the sleeper defending himself. The personality, temperament, and behavior of RBD patients when awake are typically incongruent with the nocturnal aggressive behaviors. Of note, D'Agostino et al. demonstrated that the dreams of RBD patients were no more violent than those of the general population, and the "mild" waking temperament is an early subtle sign of the apathy that is commonly described in the context of neurodegenerative disorders [97].

Video-PSG confirmation of RSWA is required to diagnose RBD [1]. RBD is diagnosed by (1) excessive amounts of phasic and/or tonic submentalis and/or excessive

phasic limb EMG activity during REM sleep on video-PSG; (2) the presence of abnormal REM sleep clinical dream enactment behaviors during video-PSG and/or a clinical history of sleep-related injurious, potentially injurious, or disruptive behaviors; and (3) exclusion of substance abuse, other medical, neurological, psychiatric or sleep disorder, or medication(s) that may better explain the sleep disturbance [98–101].

Criteria for scoring RSWA was published by the AASM in 2007 and were based on limited literature available then [98]. A 30-s epoch of REM sleep is regarded as containing excessive *tonic* activity when the amplitude of the chin EMG is of higher amplitude than its lowest amplitude during NREM sleep for 50 % or more of the epoch. Excessive *phasic* EMG activity in REM sleep is scored by subdividing the 30-s PSG epoch into ten consecutive 3-s mini-epochs, identifying and tallying the number of 3-s mini-epochs that contain phasic EMG activity lasting 0.1–5.0 s, which is at least four times as high as the baseline EMG activity. If five or more 3-s mini-epochs of a 30-s epoch of REM sleep contain excessive phasic EMG activity, the REM sleep epoch is regarded as containing excessive phasic EMG activity. If a video-PSG contains excessive EMG activity during REM sleep but the patient has no clinical history suggestive of dream-enactment behaviors and none seen on the PSG, we say the PSG shows RSWA. However, RBD behaviors may not be captured by a single night of PSG.

Unfortunately, the AASM rules for scoring RSWA in a PSG do *not* specify which (and how many) skeletal muscles should be recorded during a video-PSG to confirm RBD or RSWA. Recent studies show that excessive phasic EMG activity and RSWA during REM sleep is more frequent in distal than proximal limb muscles and more frequent in upper limbs than lower limbs. RSWA cannot be scored on the basis of chin EMG alone, but requires recording and scoring of excessive phasic EMG activity in the upper and lower distal limb muscles. One study recorded *13* different muscles in 17 RBD patients (nine with PD) to determine which combination of muscles provides the highest rates of phasic EMG activity during REM sleep in patients with RBD. They found that the greatest amounts of excessive phasic EMG activity were observed in the mentalis, flexor digitorum superficialis, and extensor digitorum brevis muscles [102]. This combination of muscles detected 82 % of all mini-epochs containing phasic EMG activity, whereas only 55 % of excessive phasic activity would be scored if only the chin EMG was recorded. Another recorded EMG activity from *five* muscle groups (mentalis, left/right anterior tibialis, and left/right brachioradialis) found that the greatest amounts of excessive phasic EMG activity were recorded from the mentalis and brachioradialis muscles [103].

The AASM rules for scoring RSWA and RBD in a PSG do not indicate what percentage of 30-s epochs of REM sleep need to contain RSWA to diagnose it [78, 104–108]. A retrospective analysis of video-PSG data in 80 patients with iRBD and 80 age- and gender-matched controls found that a *tonic* chin EMG density (percentage of 2-s mini-epochs of REM sleep) $\geq$ 30 %, phasic chin EMG density $\geq$ 15 %, and $\geq$ 24 leg movements per hour of REM sleep would correctly identify RBD in 82 % of their 80 patients but misidentified it in one control [109]. Another study suggested that $\geq$ 10 % of REM sleep spent with elevated EMG tone or phasic burst activity

would confirm a diagnosis of clinical RBD (based on the receiver–operator curves) providing a sensitivity of 89 % but a specificity of only 57 % [110]. The percentage of PSG epochs of REM sleep containing RSWA may increase with duration of the underlying disorder: one study of 11 patients with iRBD found that the chin tonic EMG activity during REM sleep increased from 30 to 54 % when a second PSG was recorded a mean of 5 years later [111]. Figure 4.8 shows an example of RSWA recorded in a 79-year-old man.

Sleep-Related Hypermotor Seizures

Clinical features that warrant concern for sleep-related epileptic seizures are as follows: (1) events occur any time in the night, just after falling asleep, or shortly before awakening in the morning; (2) multiple events a night; and (3) occasional occurrence of these events when awake or during a brief nap. Sleep-related hypermotor seizures are often initially misdiagnosed as DoA [112–120]. Two-thirds of nocturnal hypermotor seizures emanate from the frontal lobe, one-third from the temporal lobe [121, 122], and a few from the insular region [123]. Clinical features of nocturnal hypermotor seizures are summarized in Box 4.4 [72, 120, 124–127]. Patients with NFLE often have attacks of varying severity, and minor ones are hard to distinguish from arousals seen in healthy normal controls; these are summarized in Table 4.1 [72, 128]. Normal nonepileptic arousals from sleep are best distinguished by motor behaviors that are slower, fewer, less repetitive, less stereotyped, and not associated with dystonic features [128].

Because NFLE seizures are usually brief in duration, most do not leave their beds. Occasionally, longer NFLE seizure may lead to "wandering." Focal seizures that lead to ictal or postictal "wandering" most often emanate from the temporal lobe, particularly the right temporal, and most often begin during wakefulness [121, 122, 129–131]. Studies suggest sleep (or epilepsy) specialists will most likely identify nocturnal events as NFLE if these have an abrupt explosive onset from NREM 2 sleep and hypermotor or asymmetric dystonic posturing accompany them [132]. Many patients with NFLE may complain that their seizures disrupt their sleep (and often they do). Fifty percent of 33 patients with NFLE complained of nocturnal awakenings compared with 22 % of controls; 36 % of patients complained of EDS (11 % of controls), and those who complained of EDS were more likely to report frequent nocturnal awakenings [133].

Be advised that 80 % of adults with NFLE have no IEDs in their EEGs when awake or asleep, 20–54 % have no scalp-recorded ictal EEG activity during many or most of their seizures, and 25 % have normal interictal and ictal EEGs [72, 120]. The lack of scalp ictal EEG activity in NFLE has been attributed to following reasons: (1) muscle artifact often obscures the tracing; (2) events often last < 20–30 s; (3) little or no postictal slowing; and/or (4) the epileptic focus is "buried" in the mesial frontal or inferior frontopolar regions "hidden" from scalp EEG recordings. Given this, the diagnosis of NFLE in these patients is confirmed by recording multiple seizures, noting their relatively stereotyped nature and clinical semiology.

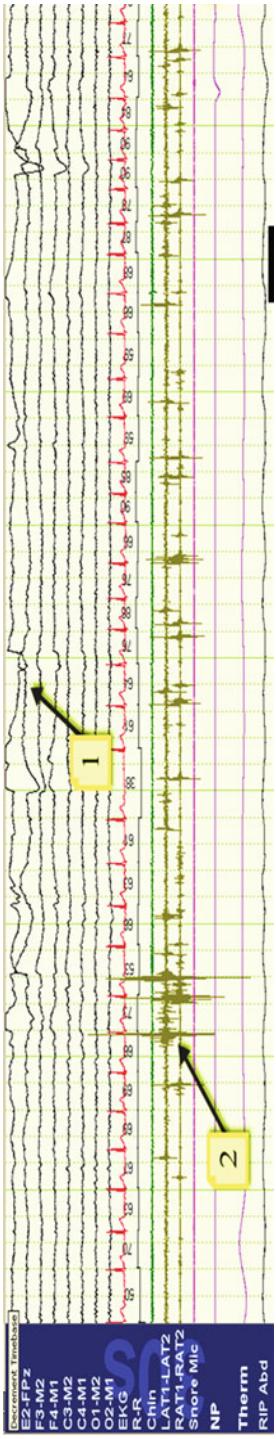

Fig. 4.8 RSWA recorded in a 79-year-old man

Table 4.1 Nocturnal frontal lobe epilepsy characterized by paroxysmal attacks of increasing complexity

Attack type	Duration	Clinical features
Minor attacks	2–4 s	Stereotyped head, axial, or limb movements
Paroxysmal arousals	5–10 s	Abrupt arousal accompanied by trunk and head elevation often with vocalization and a frightened expression
Major attacks (formerly called "nocturnal paroxysmal dystonia")	20–30 s	Begin as an abrupt arousal and rapidly progress to bipedal automatisms, rhythmic twisting movements of trunk, bizarre repetitive hypermotor behaviors, and/or asymmetric dystonic or tonic postures
Episodic nocturnal wanderings	> 1–3 min	Begin as a paroxysmal arousal process followed by leaping from bed, walking, running, screaming, or loud vocalization; often accompanied by fear and bizarre behaviors

Ictal EEG activity may accompany 80 % of NFLE seizures (although it is typically brief and rapidly obscured by muscle artifact). When ictal EEG activity is present in NFLE, it can be unilateral low-voltage paroxysmal fast activity, rhythmic theta activity, or flattening of the EEG over the frontocentral, frontocentral-temporal, frontotemporal or parasagittal regions. A unilateral onset may be followed by early or late spread of the ictal activity to the contralateral hemisphere. When ictal EEG activity in NFLE is bilateral, it is usually maximal over the frontal, frontocentral, or frontotemporal regions, begins as either rhythmic fast or low-voltage fast activity, and sometimes lateralizes to the side of the epileptogenic focus. An example of a nocturnal frontal seizure recorded in our sleep laboratory is shown in Fig. 4.9.

Alternative Methods for Diagnosing Parasomnias

Derry et al. (2006) designed a frontal lobe epilepsy and parasomnias (FLEP) scale to assess the likelihood that a PNE was likely to be NFLE based on the clinical history alone [134]. The FLEP consists of a series of questions based on an initial series of cases and clinical expertise. Responses to the questions asked in the FLEP scale that favored nocturnal seizures were as follows: events last < 2 min, occur ≥ 3–5 times per night, and behaviors during these events are highly stereotyped. Highly variable clinical semiology or onset after age 55 years lessens the likelihood that a PNE is NFLE. A patient with a score of zero or less on the FLEP scale is very unlikely to have epilepsy, and any patient with a score of $> +3$ is very likely to have epilepsy, whereas video-EEG or PSG monitoring is needed for those with an FLEP score of $+1$ to $+3$. For NFLE, the FLEP scale had a sensitivity of 71 %, a specificity of 100 %, a positive predictive value of 100 %, and a negative predictive value of 91 % [134]. Manni et al. (2008) found the FLEP scale usually identifies NFLE but it is less reliable for differentiating SW from epileptic nocturnal wandering and distinguishing RBD from epilepsy [135]. The FLEP scale in 71 subjects (mean age 54 ± 21 years,

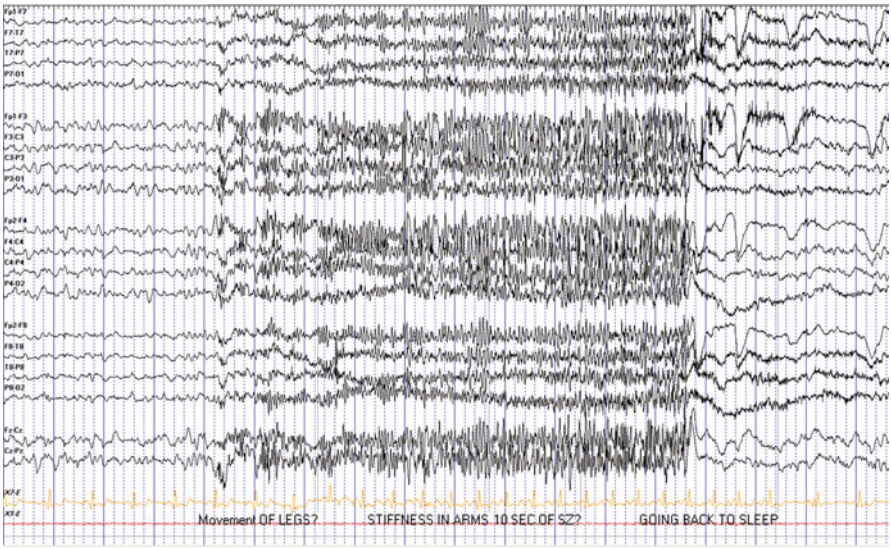

Fig. 4.9 A nocturnal frontal lobe seizure

11 had DoA, 14 NFLE, and 46 iRBD) incorrectly diagnosed four (6 %) of the patients (specifically NFLE patients who had epileptic nocturnal wandering). FLEP scores were in the equivocal range (+ 1 to + 3) in 31 % of the patients requiring video-PSG or video-EEG. Consider asking the family to record home video of the nocturnal events [136–140]. These are simple to request and obtain if events are frequent enough, but most often, the crucial beginning of an event is lost.

The RBD Screening Questionnaire (RBDSQ) is available in English, German, and Japanese and validated in these populations [141–144]. It consists of ten items with 13 yes/no questions (maximum possible score 13). An RBDSQ score of 5 or more showed a sensitivity of 92 % and specificity of 93 % for RBD when subjects were compared with the general population, but a sensitivity of 96 % and specificity of 57 % when it was used in patients with PD.

Future Directions

We need to understand (1) the mechanisms which underlie cortical excitation emerging from sleep in patients with sleep-related epilepsies; (2) the role of sleep in sudden unexpected death in patients with epilepsy; (3) the influence of circadian rhythms and chronotypes on different epilepsy syndromes; (4) whether IEDs and seizures, when sufficiently frequent, impair long-term development and cognitive functioning in neonates and children; (5) whether treating OSA in patients with epilepsy improves seizure control; and (6) why sleep macro- and microarchitecture is altered in patients with epilepsy. More specific guidelines for scoring RSWA in a PSG are sorely needed.

The link between sleep DoA and epilepsy has been elucidated through advancements in monitoring and evaluation techniques utilized during sleep studies. It is evident that treatment strategies can begin from either a neurological or a sleep medicine approach. However, it is also clear that further study into the structural and chemical etiologies of both disorders will be necessary to understand how to best improve overall function and that a multidisciplinary model will best serve patients with these disorders.

Practical Points

1. Parasomnias are most common in children and decrease in prevalence with increasing age; most resolve in the teenage years.
2. Parasomnias that occur > 2–3 times per week warrant a PSG to identify another sleep disorder or seizure disorder that may be a precipitating factor.
3. Most diurnal movement disorders (including tremor, dystonia, chorea, hemiballismus, and myoclonus) persist or intermittently recur in sleep.
4. RBD (dream enactment) should prompt a referral to a sleep specialist or neurologist as it may be related to a sleep disorder and is associated with development of a neurodegenerative disease.

Case Example

A 67-year-old man presented with his wife of 40 years who after waking up, found that he was standing on his feet, holding a television above his head, and about to throw it against the wall. His wife reports that his nighttime movements began 5 years ago. The movements would range from waving an arm in the air to punching and kicking gestures and occurred a few times per month. Recently, the behaviors occur almost nightly. A few times, she was awakened because of aggressive physical contact. This behavior became so disruptive and frightening to the wife that she decided to sleep in another room. She reported that at times she hears yelling or the sounds of someone punching the bed; however, she is too frightened to go into the bedroom to see what her husband is doing. She does not understand how such a kind and gentle man can become so violent at night. It was only after constant urging from their children and the most recent event that she was willing to discuss this issue. The patient reported that he dreams of someone attacking him or his family and finds himself walking up from the dream defending the people he loves. The patient states that he has felt fatigued over the past year, but attributes this to getting older. He also feels depressed at times when it takes him longer to complete tasks compared with that taken a “few years ago.”.

Hematologic studies were normal; however, the physical examination revealed a mild resting tremor in the left hand, decreased expression on the face, and slow but steady ambulation. No paucity of speech or rigidity was present.

Diagnosis: early Parkinson’s disease

This case presents classic findings of RBD, which is specific for dream enactment due to the loss of the normal atonia of REM sleep. Behaviors include movement of a limb, vocal expressions, and violent behavior. Individuals affected are typically men over the age of 50 years, and events are often paradoxical to normal wake personality and behaviors. In this patient, RBD was a precursor to the development of PD. The likelihood of developing a neurodegenerative disease (PD, DLB, and MSA) within 5 years is 42 % in individuals with RBD. This patient was referred to a neurologist for ongoing care. In the interim, the patient was started on pharmacy grade melatonin at 3 mg, 1–2 h prior to his anticipated bedtime. Melatonin at doses of 0.5–12 mg is $>$ 80 % effective in controlling RBD behaviors and promotion of the atonia of REM. In follow-up, the wife reported that the bedtime movements have stopped and she is able to sleep by his side again. This case demonstrates a common barrier to diagnosis of RBD, in that patients may be reluctant to discuss this problem with their provider. It is important to ask patients over the age of 50 years and their families if dream enactment occurs as the treatment has great potential to improve the quality of life for those affected.

Box 4.1: Differential Diagnosis of Paroxysmal Nocturnal Events

- NREM arousal disorder (confusional arousal, sleep walking, sleep terror);
- Sleep-related epilepsy;
- REM sleep behavior disorder (RBD) and pseudo-RBD due to obstructive sleep apnea;
- Nightmare disorder and post-traumatic stress disorder;
- Sleep-related panic attacks;
- Sleep-related dissociative disorder;
- Sleep-related choking, laryngospasm, or gastroesophageal reflux;
- Sleep-related rhythmic movement disorder (often with vocalization);
- Sleep-related expiratory groaning (catathrenia);
- Sudden death when sleeping due to myocardial infarction, Brugada syndrome, untreated OSA, sudden unexpected death in epilepsy, and trauma;
- Sleep bruxism, rhythmic masticatory muscle activity and faciomandibular myoclonus;
- Sleep-related hypnagogic foot tremor.

Box 4.2: "Typical" NREM 3 Arousal Disorder Features in Young Children Who Usually Do Not Need a PSG for Diagnosis

- Occur in first third of the night when NREM 3 predominates;
- Appear confused and disoriented;
- Exhibit automatic motor behaviors and autonomic disturbances, which suggest sympathetic activation;
- Difficult to arouse from episode;

- Positive family history of sleepwalking/sleep terrors;
- Fragmentary hypnic imagery or no recall of event;
- Cannot be consoled;
- May resist intervention;
- Moderate to high likelihood of injury in agitated sleepwalking or sleep terrors;
- Little or no responsiveness to external environment.

Box 4.3: Guidelines to Assist in Determining the Role of a Sleep Disorder in a Specific Violent Act (58)

1. A reasonable basis (by history or video-PSG) to suspect a sleep disorder. Similar episodes with benign or morbid outcome should have occurred previously;
2. Duration of the violent act was brief (minutes);
3. Violent behavior had an abrupt onset, was impulsive, senseless, and without apparent motivation;
4. Victim was someone merely present, and who may have been stimulus for the arousal;
5. Immediately following return of consciousness, the individual exhibits perplexity or horror, without attempt to escape, conceal, or cover-up the action.
6. Evidence of lack of awareness of the individual during the event;
7. Usually some degree of amnesia during the event, which may not be complete;
8. In NREM arousal events, the act may occur on awakening usually 1 h after falling asleep, may occur upon attempts to awaken the patient, and may have been potentiated by alcohol, sedative/hypnotic, or prior sleep deprivation.

Box 4.4: Clinical Features of Nocturnal Hypermotor Seizures

- An abrupt, often explosive, onset awakening from grossly undisturbed NREM 2 sleep;
- Asymmetric dystonic or tonic postures;
- Thrashing, pedaling, and kicking of the lower extremities;
- Tend to be “fairly” stereotyped in appearance for the individual patient;
- Brief (typically lasting 20-30 s, less than 1–2 min);
- Patients are often aware during the seizure, but say they cannot control their movements or vocalizations;
- No postictal confusion or amnesia;
- Twenty percent have no accompanying scalp-recorded ictal EEG activity.

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