Parasomnias & Other Non–Epileptic Events from Sleep

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Normal Sleep Histogram

SEQUENCES OF STATES AND STAGES OF SLEEP ON A TYPICAL NIGHT

Stages

Awake

Hours of Sleep

0 1 2 3 4 5 6 7 8
Normal Sleep–Wake Chemistry

Diagram showing the brain's wakefulness, slow wave sleep (SWS), and rapid eye movement (REM) states.
REM Sleep

REM sleep is a highly activated brain in a paralyzed body!

Three Components:
- EEG activation,
- Muscle atonia, and,
- Episodic bursts of rapid eye movements.

Tonic and Phasic types of REM sleep are distinguished usually for research purposes.

The most commonly used marker of REM sleep \textit{phasic} activity in human beings are:
- bursts of rapid eye movements;
- muscle twitches and,
- cardio–respiratory irregularities often accompany the REM bursts.

The mental activity of human REM sleep is associated with dreaming, based on vivid dream recall reported after approximately 80\% of arousals from this state of sleep.

Inhibition of spinal motor neurons by brainstem mechanisms mediates suppression of postural motor tonus in REM sleep.
NREM–REM Transition
Sleep is entered through NREM sleep.
NREM sleep and REM sleep alternate with a period near 90 minutes.
SWS predominates in the first third of the night and is linked to the initiation of sleep.
REM sleep predominates in the last third of the night and is linked to the circadian rhythm of body temperature.
Wakefulness in sleep usually accounts for less than 5% of the night.
Stage 1: 2% to 5% of sleep.
Stage 2: 45% to 55% of sleep.
Stage 3: 3% to 8% of sleep.
Stage 4: 10% to 15% of sleep.
NREM sleep is therefore 75% to 80% of sleep.
REM sleep is usually 20% to 25% of sleep, occurring in four to six discrete episodes.
Classification of Parasomnias

- ICSD–3
- DSM 5
A. Recurrent episodes of incomplete awakening from sleep, occurring during the first third of the major sleep episode, accompanied by

1. **Sleepwalking**: Repeated episodes of rising from bed during sleep and walking about. While sleepwalking, the individual has a blank, staring face, is relatively unresponsive to the efforts of others to communicate with him or her, and can be awakened only with great difficulty.

2. **Sleep Terrors**: Recurrent episodes of abrupt terror arousals from sleep, usually beginning with a panicky scream. There is intense fear and signs of autonomic arousal, such as mydriasis, tachycardia, rapid breathing, and sweating, during each episode. There is relative unresponsiveness to efforts of others to comfort the individual during these episodes.
B. No or little (e.g., only a single visual scene) dream imagery is recalled
C. Amnesia for the episodes is present
D. The episodes cause clinically significant distress or impairment in social, occupational, or other important areas of functioning
E. The disturbance is not attributable to the physiological effects of a substance (e.g. a drug of abuse, a medication)
F. Coexisting mental and medical disorders do not explain the episodes of sleepwalking or sleep terrors

307.46 (F51.3): **Sleepwalking type**
*Specify if:*
*With sleep-related eating*
*With sleep-related sexual behavior (sexsomnia)*

307.46 (F51.4) **Sleep terror type**
NREM vs REM Parasomnia

NREM Sleep Arousal Disorders
- Sleepwalking
- Sleep Terrors
- (Confusional Arousal) (ICSD–3)

REM Parasomnia
- REM Sleep Behaviour Disorder (RBD)
- Painful Penile Erections
- “Wet Dreams”
Violence and Sleep

**Violent behavior during sleep** refers to injury to oneself or others committed while in a sleep state or while in a state of incomplete awakening from sleep.

Often, violent behavior occurs during an incomplete awakening in the context of a *disorder of arousal.*
NREM Parasomnias

- Sleepwalking
- Sleep Terror
- Confusional Arousal – not in DSM–5

A continuum of the same pathophysiology
Prevalence

- Childhood – up to 15%, Adults – 2%
- Onset – Majority (> 70%) in Childhood/Adolescence – recent data suggest 16.9% Adult Onset (Lopez et al. 2013)
- Usually + Family History
- Genetics – ??
Mainly from **Clinical History** substantiated by Special Investigations (PSG, EEG):

1. SW history characterized by behavioural manifestations, misperception of the environment, impaired judgment, and frequent retrograde amnesia;
2. At least one SW episode annually;
3. At least one episode in the past 6 months
4. No other Organic Cause found on investigation

Lopez et al., 2013
NREM Parasomnia

1. Disorientation on awakening;
2. Confusional behaviour;
3. Retrograde Amnesia – possible for fragments of distorted memory to be retained
4. No Concealment
5. Out of character behaviour
6. Absence of factors suggesting Intent: evidence of pre-planning, motivation, and behaviours reported during the event that suggest conscious motivation all point a non-sleep related cause of the violence.
NREM Parasomnia

7. Trigger factors
A. External
   - Noise
   - Touch and/or Proximity
   - Sedatives (Medication)
   - Sleep Deprivation, Sleep–Wake Cycle Disruption
   - Stress (53%)
   - Intense Physical Activity
   - Alcohol (12%)
B. Internal: Sleep Disordered Breathing (SDB), Periodic Limb Movements (PLMs).
8. Support from Sleep Studies
NREM Parasomnias – Special Investigations

Sleep laboratory studies (polysomnography, PSG) will add value by

1. Excluding or identifying possible Internal Trigger factors or other organic causes for the behaviour, e.g. Sleep Disordered Breathing, Periodic Limb Movements of sleep, etc.

2. To assess the impact of External Triggers (Alcohol, Sleep Deprivation, Noise, Touch)

3. To diagnose other causes of the behaviour e.g. RBD and Epilepsy
Violent Parasomnias – PSG Studies

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Nocturnal Wandering and Violence: Review of a Sleep Clinic Population*

Sleep disorders associated with *automatic behavior*. 

- **Disorders of arousal**
  - Confusional arousals (sleep drunkenness)
  - Somnambulism / Sleep walking
  - Sleep terror
- **REM Sleep Behavior Disorder**
- **Nocturnal Complex seizures**
  - Particularly frontal, infero – mesial temporal
Sleep disorders associated with automatic behavior:

- Hypnagogic Hallucinations
- Automatic behavior associated with
  - Narcolepsy and CNS hypersomnina
  - Obstructive sleep apnea
  - Sleep deprivation (including jet lag)
  - Hypnotic intake
  - Cerebral degenerative disease
REM Sleep Behavior Disorder (RBD)
Definition of RBD

- “RBD is characterized by the intermittent loss of REM sleep electromyographic (EMG) atonia and by the appearance of elaborate motor activity associated with dream mentation”
Dream Enactment Behaviour—abnormal vocalisations, motor behaviour, altered dream mentation (can also occur in OSA and in Sleepwalking)

REM Sleep Without Atonia ie PSG confirmation

Primary or Secondary, to Narcolepsy or Neurodegenerative Disease
Etiology (I)

- Idiopathic RBD (~ 60 %)
- Symptomatic RBD (~40 %)

**Acute RBD**
- *Withdrawal* from alcohol, Meprobamate and nitrazepam) or
- *Intoxication* or OD of biperiden, tricyclic antidepressants, MAOIs, SSRI’s

**Chronic RBD**
- *Medications* – TCAs, SSRIs, SNRIs, Anticholinergics
- *Cerebrovascular* causes (hemorrhage, ischemic)
- *Tumours* (esp. of the pontine area)
Etiology (II)

- Neurodegenerative diseases
  - Parkinson’s disease
  - Dementia (Alzheimer’s disease, Lewy body disease, corticobasal degeneration)
  - Olivopontocerebellar degeneration
  - Multiple system atrophy
  - Amyotrophic lateral sclerosis
- Other causes (narcolepsy, familial, etc.)
Differential Diagnosis of RBD

- Confusional arousals
- Sleepwalking
- Night terrors
- Sleep–related seizures
- Nocturnal panic attacks
- Periodic limb movement disorder
- Obstructive sleep apnea
- Post–traumatic stress syndrome – Nightmares
- Dissociative disorder
- Cardiopulmonary and gastrointestinal disorders
Treatment of RBD

- Safety measures
- Clonazepam (0.5 – 2.0 mg at bedtime)
- Melatonin (3 – 12 mg / night)
- Carbidopa / L–dopa
- Clonidine
- Carbamazepine and Gabapentin
- Tricyclic antidepressants and MAOIs
- Other agents (pramipexole, donepezil, triazolam, clozapine, quetiapine)
REM Sleep Behavior Disorder—Psychiatric Presentations: A Case Series from the United Kingdom

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Rapid eye movement sleep behavior disorder (RBD) has rarely been associated with a psychiatric condition. We report a series of cases of RBD presenting as psychiatric disorders. These patients were assessed at a specialist sleep disorders center and investigated using polysomnography and, where appropriate, magnetic resonance imaging of the brain and neuropsychological tests. These cases of RBD highlight the varying presentations and causes of RBD that may involve psychiatrists, sleep specialists, and primary care physicians. These include idiopathic RBD presenting as depression, antidepressant-induced RBD, and a patient with undiagnosed Parkinson disease presenting with RBD. There is an increasing body of knowledge about RBD. At least 10% of patients with RBD are likely to present with psychiatric symptoms. It is essential that the condition is recognised and distinguished from other causes of sleep interruption. After recognizing the disorder, it is essential that the clinician undertake a thorough assessment, including a sleep history and formal investigation of sleep patterns at a specialized unit.

Key Words: REM Sleep Behavior Disorder, psychiatry, Parkinson’s disease, depression, anti-depressants

Citation: Ebrahim IO; Peacock KW. REM sleep behavior disorder—psychiatric presentations: a case from the United Kingdom. J Clin Sleep Med 2005;1(1):43-47
Differential Diagnoses

- NREM Parasomnia – Status Parasomnicus?
- Epilepsy – NFLE?
- Psychogenic/Non-Epileptic Seizures?
- Combination
Hyperkinetic manifestations in nocturnal frontal lobe epilepsy. Semeiological features and physiopathological hypothesis
Table 1 Seizures in NFLE

Very brief motor seizures
- Bilateral and axial involvement resembling a sudden arousal
- Opening of the eyes
- Sitting up in bed
- Sometimes frightened expression

Hypermotor seizures
- Body movements that can start in the limbs, head or trunk
- Complex, often violent behaviour
- Often with a dystonic-dyskinetic component
- Sometimes with cycling or rocking or repetitive body movements prevalent in the trunk or legs
- The patient may vocalise, scream or swear
- Fear is a frequent expression

Asymmetric, bilateral tonic seizures
- Sustained uncustomary forced position

Prolonged seizures
- Same beginning as above
- Semi-purposeful ambulatory behaviour
- Mimicking sleepwalking
NREM Arousal Parasomnias and Their Distinction from Nocturnal Frontal Lobe Epilepsy: A Video EEG Analysis

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Study Objectives. To describe the semiological features of NREM arousal parasomnias in detail and identify features that can be used to reliably distinguish parasomnias from nocturnal frontal lobe epilepsy (NFLE).

Design. Systematic semiological evaluation of parasomnias and NFLE seizures recorded on video-EEG monitoring.

Patients. 120 events (57 parasomnias, 63 NFLE seizures) from 44 subjects (14 males).

Interventions. The presence or absence of 68 elemental clinical features was determined in parasomnias and NFLE seizures. Qualitative analysis of behavior patterns and ictal EEG was undertaken. Statistical analysis was undertaken using established techniques.

Results. Elemental clinical features strongly favoring parasomnias included: interactive behavior, failure to wake after event, and indistinct offset (all P < 0.001). Cluster analysis confirmed differences in both the frequency and combination of elemental features in parasomnias and NFLE. A diagnostic decision tree generated from these data correctly classified 94% of events. While sleep stage at onset was discriminatory (82% of seizures occurred during stage 1 or 2 sleep, with 100% of parasomnias occurring from stage 3 or 4 sleep), ictal EEG features were less useful. Video analysis of parasomnias identified three principal behavioral patterns: arousal behavior (92% of events); non-agitated motor behavior (72%); distressed emotional behavior (51%).

Conclusions. Our results broadly support the concept of confusion arousals, somnambulism and night terrors as prototypical behavior patterns of NREM parasomnias, but as a hierarchical continuum rather than distinct entities. Our observations provide an evidence base to assist in the clinical diagnosis of NREM parasomnias, and their distinction from NFLE seizures, on semiological grounds.

Keywords: Parasomnia, sleep terror, confusional arousal, somnambulism, nocturnal frontal lobe epilepsy, EEG

Citation: Derry CP; Harvey AS; Walker MC; Duncan JS; Berkovic SF. NREM arousal parasomnias and their distinction from nocturnal frontal lobe epilepsy: a video EEG analysis. SLEEP 2009;32(12):1637-1644.
SHORT COMMUNICATION

Atypical presentation of NREM arousal parasomnia with repetitive episodes

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Facilitation of epileptic activity during sleep is mediated by high amplitude slow waves

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Epileptic discharges in focal epilepsy are frequently activated during non-rapid eye movement sleep. Sleep slow waves are present during this stage and have been shown to include a deactivated (‘down’, hyperpolarized) and an activated state (‘up’, depolarized). The ‘up’ state enhances physiological rhythms, and we hypothesize that sleep slow waves and particularly the ‘up’ state are the specific components of non-rapid eye movement sleep that mediate the activation of epileptic activity. We investigated eight patients with pharmaco-resistant focal epilepsies who underwent combined scalp-intracerebral electroencephalography for diagnostic evaluation. We analysed 259 frontal electroencephalographic channels, and manually marked 442 epileptic spikes and 8487 high frequency oscillations during high amplitude widespread slow waves, and during matched control segments with low amplitude widespread slow waves, non-widespread slow waves or no slow waves selected during the same sleep stages (total duration of slow wave and control segments: 49 min each). During the slow waves, spikes and high frequency oscillations were more frequent than during control segments (79% of spikes during slow waves and 65% of high frequency oscillations, both P ~ 0). The spike and high frequency oscillation density also increased for higher amplitude slow waves. We compared the density of spikes and high frequency oscillations between the ‘up’ and ‘down’ states. Spike and high frequency oscillation density was highest during the transition from the ‘up’ to the ‘down’ state. Interestingly, high frequency oscillations in channels with normal activity expressed a different peak at the transition from the ‘down’ to the ‘up’ state. These results show that the apparent activation of epileptic discharges by non-rapid eye movement sleep is not a state-dependent phenomenon but is predominantly associated with specific events, the high amplitude widespread slow waves that are frequent, but not continuous, during this state of sleep. Both epileptic spikes and high frequency oscillations do not predominate, like physiological activity, during the ‘up’ state but during the transition from the ‘up’ to the ‘down’ state of the slow wave, a period of high synchronization. Epileptic discharges appear therefore more associated with synchronization than with excitability. Furthermore, high frequency oscillations in channels devoid of epileptic activity peak differently during the slow wave cycle from those in channels with epileptic activity. This property may allow differentiating physiological from pathological high frequency oscillations, a problem that is unresolved until now.