Chapter 14
Parasomnias in Children

Paola Proserpio and Lino Nobili

Abstract Parasomnias are undesirable physical events or experiences that occur during sleep. They are classified on the basis of the sleep stage during which each of the parasomnias tends to occur: NREM-related parasomnias also defined as disorders of arousal (confusional arousals, sleepwalking, sleep terrors, and sleep-related eating disorder), REM-related parasomnias (REM sleep behavior disorder, recurrent isolated sleep paralysis, and nightmare disorder), and other parasomnias (exploding head syndrome, sleep-related hallucinations, and sleep enuresis). Parasomnias include several clinical features, with different complexity of behaviors, usually associated with autonomic nervous system changes and skeletal muscle activity.

The current pathophysiological theories consider parasomnias as state dissociation, characterized by the coexistence of wake- and sleeplike activity within cortical and subcortical areas of the brain. Although parasomnias are not usually associated with a primary complaint of insomnia or excessive sleepiness, they are considered clinical disorders because of possible resulting injuries, adverse health, and psychosocial effects.

Most of the parasomnias can be diagnosed based on history alone. Only the REM sleep behavior disorder requires video-polysomnographic documentation as one of the essential diagnostic criteria. However, polysomnographic recordings can be useful also in other parasomnias especially when the differential diagnosis is difficult or in the case of suspected comorbidities with other sleep disorders. Patient education and behavioral management represent the main treatment approaches to the patient with parasomnias. A pharmacological treatment may be useful when episodes are frequent and persist despite resolution of possible inducing factors, are associated with a high risk of injury, or cause secondary consequences.

Keywords Confusional arousals • Sleepwalking • Sleep terrors • REM-related parasomnias • Sleep enuresis • State dissociation • Nocturnal frontal lobe epilepsy

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S. Nevšímalová, O. Bruni (eds.), *Sleep Disorders in Children*,
DOI 10.1007/978-3-319-28640-2_14

Introduction

The term "parasomnia" derives from the Greek "para" meaning "around" and the Latin "somnus" meaning "sleep" and was coined in 1932 by the French researcher Henri Roger who gave a scrupulous descriptions of sleep terror and somnambulistic episodes in his monograph entitled "Les Troubles du Sommeil-Hypersomnies, Insomnies, and Parasomnies."

In accordance with the third edition of International Classification of Sleep Disorder (ICSD3) [1], parasomnias are defined as "undesirable physical events or experiences that occur during entry into sleep, within sleep, or during arousal from sleep." There are several possible ways to classify the parasomnias. The most widely accepted classification is that suggested by the American Academy of Sleep Medicine [1], which is based on the sleep stage during which each of the parasomnias tends to occur (Table 14.1).

The current pathophysiological theories consider parasomnias as state dissociation, characterized by the coexistence of wake- and sleeplike activity within cortical and subcortical areas of the brain. The normally distinct three essential states of human consciousness – wake, NREM sleep, and REM sleep – are modulated by a complex neural system, with functionally distinct but integrated components, that allows an unambiguous separation between these states. However, recent studies showed that brain-sleep state may be spatially nonuniform and sleep and wakefulness may not be temporally distinct behavioral states but rather part of a continuum resulting from the complex interaction between diffuse neuromodulatory systems and intrinsic properties of the different thalamocortical modules [2]. This interaction may account for the occurrence of dissociated activity across different brain structures characterizing both physiological and pathological conditions [3].

Although these disorders occur predominantly or exclusively during sleep, they are not usually associated with a primary complaint of insomnia or excessive sleepiness. However, they are considered clinical disorders because of possible resulting injuries, adverse health, and psychosocial effects. The clinical consequences of the parasomnias can affect the patient, the bed partner, or both.

Parasomnias include several clinical features, with different complexities of behaviors, usually associated with autonomic nervous system changes and skeletal

Table 14.1 Classification of parasomnias

NREM-related parasomnias (disorders of arousal)	REM-related parasomnias	Other parasomnias
Confusional arousals	REM sleep behavior disorder	Exploding head syndrome
Sleepwalking	Recurrent isolated sleep paralysis	Sleep-related hallucinations
Sleep terrors	Nightmare disorder	Sleep enuresis
Sleep-related eating disorder		

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muscle activity. For this reason they are considered a different entity from the "sleep-related movement disorders" that include a wide range of predominantly simple motoric activities (myoclonic, repetitive, rocking, rhythmic, grinding, cramping, fragmentary, dystonic, or dyskinetic movements or tremors) which are not usually associated with dream mentation or experiential concomitants.

Most of the parasomnias can be diagnosed based on history alone. Considering the ten core categories of parasomnias listed in the ICSD3, only the REM sleep behavior disorder requires video-polysomnographic documentation as one of the essential diagnostic criteria. However, polysomnographic recordings can be useful also in other parasomnias especially when the differential diagnosis is difficult or in the case of suspected comorbidities with other sleep disorders.

NREM-Related Parasomnia

NREM-related parasomnia or "disorders of arousal" (DoA) are the subgroup of parasomnias arising from NREM sleep. Diagnostic criteria from the ICSD3 are shown in Table 14.2. This group is composed of confusional arousals, sleep terrors, sleepwalking, and sleep-related eating disorder (SRED). More than one type may coexist within the same patient. These parasomnias occur primarily in childhood and normally cease by adolescence, but the onset or persistence during adulthood is well recognized. Especially in children, they are considered benign phenomena. However sometimes DoA can be characterized by complex behavior with potentially violent or injurious features or can result in the complaint of excessive daytime sleepiness. Evaluation and treatment are therefore recommended for patients whose activities are potentially violent or are very disturbing to other family members. Finally, because other parasomnias, particularly the REM sleep behavior disorder and nocturnal seizures, can perfectly mimic disorders of arousal, extensive

Table 14.2 NREM-related parasomnias and diagnostic criteria

Criteria A–E must be met
A. Recurrent episodes of incomplete awakening from sleep
B. Inappropriate or absent responsiveness to efforts of others to intervene or redirect the person during the episode
C. Limited (e.g., a single visual scene) or no associated cognition or dream imagery
D. Partial or complete amnesia for the episode
E. The disturbance is not better explained by another sleep disorder, mental disorder, medical condition, medication, or substance use
Notes
1. The events usually occur during the first third of the major sleep episode
2. The individual may continue to appear confused and disoriented for several minutes or longer following the episode

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video-polysomnographic recordings can provide corroborative documentation in support of the clinical diagnosis.

Considering that SRED occurs almost exclusively during adulthood, this subtype of NREM parasomnia will be briefly described separately at the end of this chapter.

Epidemiology

NREM parasomnias are generally considered a common pediatric sleep disorder that tends to decrease with development. Same individual may experience more than one type of arousal parasomnias. However, epidemiological figures for these co-occurrences are yet to be determined.

Almost all children have confusional arousals on occasion; in particular, during the preschool age, they frequently experience minor episodes of partial awakening from sleep, which might not even come to parental attention. For this reason, epidemiological studies on confusional arousal are scanty. In children, Laberge et al. [4] found that about 17 % of children between 3 and 13 years are experiencing occasional or frequent episodes of confusional arousals. In another study, Ohayon et al. observed that confusional arousal affected 4.2 % of the general population, decreasing from 6.1 % in the 15–24 age group to 3.3 % in the 25–34 and stabilizing around 2 % after 35 years old [5].

The prevalence of sleepwalking in children ranges from 3 to 14.5 % [6, 7]; most episodes usually resolve after the age of 10 years. A strong familial occurrence has often been reported although the genetical basis of this phenomenon has yet to be clarified. Recently the first genetic locus for sleepwalking at chromosome 20q12-q13.12 has been described [8].

Sleep terrors have the greatest incidence in preschool children. Laberge et al. [4] reported an overall prevalence of sleep terrors of 17.3 % in children between 3 and 13 years. In another longitudinal study, the frequency of this sleep disorder was 39.8 % in age group 2.5–6 years, with a peak at ages 2.5, 3.5, and 4 years [7].

The prevalence of NREM parasomnias in adults is unknown, but mostly represents a continuation of episodes after adolescence, sometimes after having been symptom-free for several years. A recent population-based cross-sectional study in 1,000 randomly selected young adults (18 years and older) showed a lifetime prevalence of confusional arousals of 18.5 % and actual prevalence (in the previous 3 months) of 6.9 %. For sleepwalking these prevalences were 22.4 % and 1.7 %, and for sleep terrors they were 10.4 % and 2.7 %, respectively [9].

Pathophysiology

It is generally considered that disorders of arousal derive from a breakdown of boundaries between wakefulness and sleep regulatory systems. Apart from the phenomenon of state dissociation, other mechanisms seem to contribute to the

appearance of these sleep disorders, such as activation of innate behaviors and locomotor centers, arousal instability, internal and external triggering mechanisms, and genetic and psychopathological influences. Finally, due to the frequent association with perinatal risk factors and developmental comorbidities, a disorder of sleep maturation has been also hypothesized [10].

The Phenomenon of State Dissociation in Arousal Parasomnias

Intracerebral stereo-EEG (S-EEG) investigations conducted in epileptic patients during the presurgical evaluation have shown that physiological NREM sleep can be characterized by the coexistence of wake-like and sleeplike EEG patterns in different cortical areas [11]. These local cortical activations (lasting on average 5–10 s) can occur in the absence of any behavioral manifestation.

In 2009, Terzaghi et al. described an episode of a confusional arousal captured during a S-EEG exploration. During the event, local fast wake-like EEG activations in the motor and cingulated cortices contrasted with the persistence or increase of sleeplike delta activities in the frontal and parietal associative cortices [12]. In a more recent study [13], S-EEG recording during a confusional arousal showed the occurrence of a local activation in the motor, cingulate, insular, temporo-polar, and amygdalar cortices, while a simultaneous persistence of slow waves was observed in the frontal and parietal dorsolateral cortices as well as sleep spindles in the hippocampal cortex. Finally, a third episode of confusional arousal was recently recorded in another drug-resistant epileptic patient during a right temporo–parietal–occipital S-EEG exploration [14]. Interestingly, the nucleus ventralis intermedius (VIM) of the thalamus had also been sampled by one distal electrode contact. During the episode, the activity recorded from the VIM showed a slight decrease in delta power and a clear-cut emergence of beta activity, which normally characterizes wake thalamic EEG.

Interestingly, the electrophysiological patterns observed in these three cases are in accordance with data previously obtained with ictal SPECT in sleepwalking which showed decreased regional cerebral blood flow in the frontoparietal cortices associated with the activation of the cingulate cortex and the absence of a deactivation of the thalamus during sleepwalking [15].

From a speculative perspective, typical features of arousal parasomnias could be explained by the coexistence of an activation of the amygdalo–temporo–insular areas disengaged from the prefrontal control cortex (emotional activation, such as fear), with the persistence of the deactivation of the hippocampal and frontal associative cortices (amnesia for the event). Interestingly, functional studies have shown that sleep deprivation, a condition that can facilitate the occurrence and increase the complexity of somnambulistic events recorded during recovery sleep, can induce an activation of the amygdala, significantly strengthening its connectivity with autonomic activating centers of the brainstem and reducing the connectivity with the prefrontal cortex [6]. The fundamental cause of “pathological” state dissociation is still unknown, but probably influenced by genetic and maturational factors.

The presence of local dissociated states during physiological sleep could suggest a possible adaptive role of this phenomenon. Indeed, a lower arousal threshold during NREM sleep in humans may have been selected, because it increases the probability of survival, facilitating motor behaviors in the case of sudden awakenings. We could hypothesize that subjects with NREM parasomnias could show a pathological increased arousability of some local neuronal networks (such as motor and limbic cortex), in contrast with an increased sleep pressure in other cortical areas. Accordingly, a transcranial magnetic stimulation study found an increased excitability of the human motor cortex during wakefulness in a group of sleepwalkers [16].

There are no definitive data about the involvement of specific neurotransmitters in the pathophysiology of arousal disorders. Sleepwalking may be associated with abnormalities in the metabolism of serotonin considering its frequent association with migraine and Tourette syndrome and the observation that several factors, known to induce the occurrence of sleepwalking, such as fever, lithium, and antidepressants, activate the serotoninergic system. Based on the results of a transcranial magnetic stimulation study in sleepwalkers, an involvement of cholinergic and GABA pathways has been supposed [16]. Finally, a possible role of the hypocretin hypothalamic system, known to play a major role in vigilance state stabilization, cannot be excluded.

Neurophysiologic Features and Alteration of Sleep Continuity in NREM Parasomnias

A peculiar feature of subjects who experience NREM parasomnia is the presence of increased arousals and cyclic alternating pattern (CAP) rate during slow-wave sleep, even on nights without episodes (for detail see “diagnosis” section). The increased number of awakenings determines a chronic condition of intra-night slow-wave activity (SWA) deprivation that is reflected in an alteration of NREM sleep continuity and in a different dynamics of SWA throughout the night. Indeed, during the first sleep cycles, patients with NREM parasomnia show a decrease of SWA values with respect to control subjects and a lack of the typical exponential decaying trend of SWA during the consecutive sleep cycles. Moreover, it has been shown that a SWA rebound and a normalization of the SWA profile in sleepwalkers are not obtained even after sleep deprivation. Contrarily, sleep deprivation results in more awakenings and an increased frequency of clinical manifestations during recovery sleep [6]. These data suggest that abnormal arousal reactions persist in sleepwalkers even after sleep deprivation.

Activations of Innate Behaviors and Locomotor Centers

Animal and human data seem to suggest that part of the “emotional” and motor clinical features of parasomnias could result from a release of inhibition of “central pattern generators” (CPGs) [17]. CPGs are “functional neural organizations” which

regulate innate behavioral automatisms and survival behaviors and located in the spinal cord, mesencephalon, pons, and bulb. The cortex itself can also operate as a CPG, and this could explain the occurrence of previously learned behaviors during arousal parasomnias.

Genetic Influences

Genetic factors have long been suggested to be involved in the occurrence of arousal parasomnias, although the pattern of inheritance of NREM parasomnias is still unknown. About 80 % of sleepwalkers have at least one family member affected by this parasomnia, and the prevalence of somnambulism is higher in children of parents with a history of sleepwalking [18]. A twin study found a concordance rate of sleepwalking 1.6 times greater in monozygotic vs. dizygotic twins for childhood sleepwalking and approximately 5.3 times greater for adult sleepwalking [19]. A small series indicates that somnambulism may be associated with excessive transmission of the HLA-DQB1∗05 and ∗04 alleles [20]. Bisulli et al. found a high frequency of arousal disorders in patients with nocturnal frontal lobe epilepsy (NFLE), suggesting that both disorders can show an abnormal (possibly cholinergic) arousal system as a common pathophysiologic mechanism [21].

Precipitating Influences

There are different conditions that may induce the occurrence of a dysfunction of the limit between sleep and wakefulness, thus triggering the occurrence of arousal parasomnias. Different studies showed that arousal disorders are more likely to occur in genetically predisposed individuals in the presence of an increased pressure for slow-wave sleep and factors favoring arousals or fragmenting sleep [22]. Frequently these two main triggering factors, sleep fragmentation and sleep deprivation, work together in a vicious circle. In particular, factors that deepen sleep encompass different conditions, such as sleep deprivation, fever, and sedative and psychotropic medications (non-benzodiazepine hypnotics, antidepressants, neuroleptics, and sodium oxybate). On the other hand, sleep fragmentation can be caused by sleep disorder (sleep apnea, periodic leg movements), fever, stress, and external stimuli such as noise or touch. Finally, an increased incidence of NREM parasomnia in patients with either nocturnal or diurnal epilepsy has been reported; the increase of arousal instability induced by epileptic discharges may favor the occurrence of NREM parasomnias.

NREM Parasomnias and Psychopathology

Not all studies agree on the extent that psychological factors may contribute to arousal parasomnias. Some studies suggest that NREM parasomnias in childhood are mainly related to developmental and genetic predisposing factors, while their

persistence or onset in adulthood can be triggered by psychological factors [23]. An epidemiological study demonstrated a high percentage of subjects with concurrent diagnoses of parasomnia and mood or anxiety disorders [24]. On the other hand, the observation that a successful treatment of a comorbid depressive disorder in adult patients with night terrors and sleepwalking had no effects on the course of parasomnias seems to suggest that the concurrent psychopathology does not play an essential role. Overall current data suggest a lack of a definitive association between a history of major psychological trauma, severe psychopathology, and sleepwalking/night terrors.

Clinical Features

Although representing distinct disorders, some researchers consider NREM parasomnias as a single continuum, ranging from confusional arousals with low motor and autonomic activation, on the one hand, to sleepwalking characterized by intense motor activity and mild autonomic activation, on the other hand. According to this theory, night terrors fall between these two, with intense autonomic discharge and mild motor activation [25]. Patients who experience one of these three phenomena are prone to demonstrate the others as well. In particular, episodes sometimes combine elements of all three, and a child might display a sequence of confusional arousals in early childhood and sleepwalking later, followed by sleep terrors in late childhood and adolescence. Alternatively, features of all three forms can occur at any one stage of development.

There are common features to these disorders (Tables 14.3 and 14.4). Although they may occur during any NREM sleep stage, these events generally occur out of deep NREM sleep (N3) and, thus, most often take place in the first third of the night when these sleep stages are most represented. Any factor that deepens sleep (sleep deprivation, stress, febrile illness, medications, alcohol) or is associated with arousals (external or internal stimuli, like the presence of sleep-disordered breathing,

Table 14.3 Clinical features of NREM parasomnia

General clinical features
Common in childhood
Decrease with increasing age
Episodes in the first third of the night
A state between sleep and waking during the event, disorientation, and confusion
Presence of triggering factors
Long episode duration (minutes)
Minimal recall of the event
Strong familial pattern

Table 14.4 A comparison of different clinical features of NREM parasomnia

	Confusional arousal	Sleepwalking	Sleep terror
Age of onset	2–10 years	4–12 years	18 months–10 years
Peak time of occurrence	First third of night	First third of night	First third of night
Ictal behavior	Whimpering, some articulation, sitting up in bed, inconsolable	Screaming, agitation, flushed face, sweating, inconsolable	Walking about the room or house, may be quiet or agitated, unresponsive to verbal commands
Motor activity	Low	Complex	Rarely complex
Autonomic activity	Low	Mild	Intense
Complications	Rare (aggressions)	Possible (violence)	Occasional (escape)
Typical duration	<1 min	1–20 min	5–20 min

natural termination of a sleep cycle, mental activity, or others) may increase the occurrence of NREM parasomnias. During the episode, patients are usually unresponsive to the environment, and they are typically completely or partially amnestic after the event, with little or no recall either immediately afterward or the next morning. Finally, the presence of a positive family history is another aspect helpful to identify NREM parasomnia.

Confusional Arousal

Confusional arousals are defined as episodes characterized by mental confusion or confused behavior that occurs while the patient is in bed, in the absence of terror or ambulation outside of the bed [1].

They occur mainly in infants and toddlers (probably most of whom have such episodes to some extent, at least in mild form) and almost invariably before the age of 5.

An episode may begin with mumbling, moaning, or whimpering, gradually increasing movements which then progress to agitated and confused behavior with marked perspiration, crying (perhaps intense, but not screaming), calling out, or thrashing about. Sometimes this causes the child to fall out of bed, although injuries are less likely than in the other arousal disorders. The child's eyes may be open or closed. Talking may also occur. The child may be partially aware of the environment and thus may be confused and combine reality with imagination, e.g., the child may sit up in the bed and put a toy in his mouth thinking that it is a dummy/pacifier. Typically, although appearing to be awake, the child does not respond when spoken to and may seem to "stare right through" his parents. Any forceful attempts to intervene may meet with severe resistance and even aggression.

Each episode usually lasts 5–15 min (sometimes much longer) before the child calms down spontaneously and returns to restful sleep. Enuresis may occur during or after an episode.

Sleepwalking

Sleepwalking is a series of complex behaviors that are usually initiated during arousals from slow-wave sleep and culminate in leaving the bed in an altered state of consciousness.

Actually, sleepwalking, also known as somnambulism, can consist of very complex motor activity, of which walking is just one element. Indeed, the symptoms and manifestations that characterize sleepwalking can show great variations both within and across predisposed patients. Movements can be repetitive and purposeless (e.g., sitting up in bed, pointing at a wall, fingering bedsheets) but also complex and meaningful (e.g., rearranging furniture, cooking or eating, getting dressed, etc.). Eyes are usually open and the sleepwalker's emotional expression can range from calm to extremely agitated. Given the heterogeneous nature of sleepwalking episodes, their duration can vary from a few seconds to dozens of minutes. Associated mental activity often includes misperception and relative unresponsiveness to external stimuli, confusion, perceived threat, and variable retrograde amnesia [6]. In a significant proportion of patients, short, unpleasant dreamlike mentations may occur during sleepwalking episodes [26].

Episodes of sleepwalking in children are rarely violent and their movements are often slow. If restrained, the child may attempt to avoid the other person but does not put up aggressive resistance. On the other hand, the most serious complication of sleepwalking in adulthood is represented by injuries and violent behavior; the number of legal cases of sleep-related violence involving sleepwalking is on the rise [22].

Sleep Terrors

Sleep terrors are characterized by episode of extreme fear or terror and agitation with prominent motor activities that arise abruptly from sleep. The episode begins suddenly with vocalization, which can be screaming or crying, sometimes associated with sitting up in bed, thrashing, agitation, confusion, a facial expression of fear, and sympathetic activation (tachycardia, flushing, mydriasis, and sweating). Patients are only partially responsive to the environment during episodes, and there is little or no recall of the event the morning after the event. Cases reported with violent behaviors appear related more to involuntary contact or provocation by another person, particularly if attempts are made to block or restrain the individual [27].

Sleep terrors usually last a few minutes but can range anywhere from 30 s to 30 min. They can occur more than once a night and up to several times per week.

Diagnosis

As in other sleep disorders, the first step in a clinical encounter with a patient with abnormal behaviors when asleep is taking a good history. An adequate general and hypnic anamnesis with the patient and bed partner is paramount, taken directly or

aided by a questionnaire. There are no available standardized sleep questionnaires for parasomnias; however, other associated symptoms can be assessed by this modality. Sleep questionnaires can be helpful to the physician to collect more quickly extensive information regarding sleep–wake habits. For examples of screening questionnaires for pediatric sleep, see Owens et al. and Archbold et al. [28, 29]. Sleep diaries can highlight irregularities of sleep/wake schedules and help determine whether episodes are triggered by sleep deprivation. Finally, a videotape of a typical episode recorded by parents at home may be very helpful to the clinician.

A clear clinical history can be sufficient to diagnose the presence of NREM parasomnia in the majority of cases, but in others only video-polysomnographic (vPSG) recording can clarify the nature of the disorder. Indeed, although accordingly with the ICSD3, vPSG is not necessary for the diagnosis of arousal disorders, this diagnostic technique is recommended: (1) in cases in which the clinical history is not completely suggestive of NREM parasomnia; (2) in the presence of injurious or extremely disruptive behaviors; (3) when there may be associated sleep disorder (sleep apnea, periodic limb movement, etc.); or (4) when the parasomnia is associated with medical, psychiatric, or neurological symptoms or findings [30, 31].

There are no consistently robust features in terms of overall sleep architecture and normal cycling among sleep stages that can result highly suggestive of patients with NREM parasomnia. However, some unusual sleep-related features have been described as characterizing the sleep of patients suffering from arousal disorders. These include hypersynchronous delta waves, irregular buildup of slow-wave activity, and NREM sleep instability.

The hypersynchronous delta (HSD) activity is defined as continuous high-voltage (>150 microV) delta waves occurring during slow-wave sleep (Fig. 14.1) or immediately prior to an episode [32] and has been investigated for a long time as a possible diagnostic sign of a NREM parasomnia. However, careful studies analyzing HSD prior to arousal disorder episodes have yielded mixed to poor results [32, 33]. Indeed, an increased number of HSD have been reported, but HSD was absent in many sleepwalkers before episodes of complex behaviors [34]. Additionally, HSD is also present in patients without history of sleepwalking but with sleep apnea or periodic leg movements [33–35]. In summary, data indicate that this electroencephalographic pattern does not appear to be a sensitive or specific diagnostic sign for a NREM parasomnia in adults and even less in children [27].

The sleep of patients with arousal disorders is characterized by an inability to maintain consolidated periods of slow-wave sleep probably due to an abnormality in the neural mechanisms responsible for the regulation of this sleep stage [36]. The increased frequency of somnambulic episodes during post-deprivation recovery sleep confirms the view that sleepwalkers suffer from a dysfunction of the mechanisms responsible for sustaining stable slow-wave sleep [37].

The cyclic alternating pattern (CAP) is a phenomenon of changing patterns in sleep that often cycle and alternate every 20–30 s and expresses the organized complexity of arousal-related phasic events in NREM sleep, thus representing a measure of NREM instability [38, 39]. Recently this pattern has been studied in patients

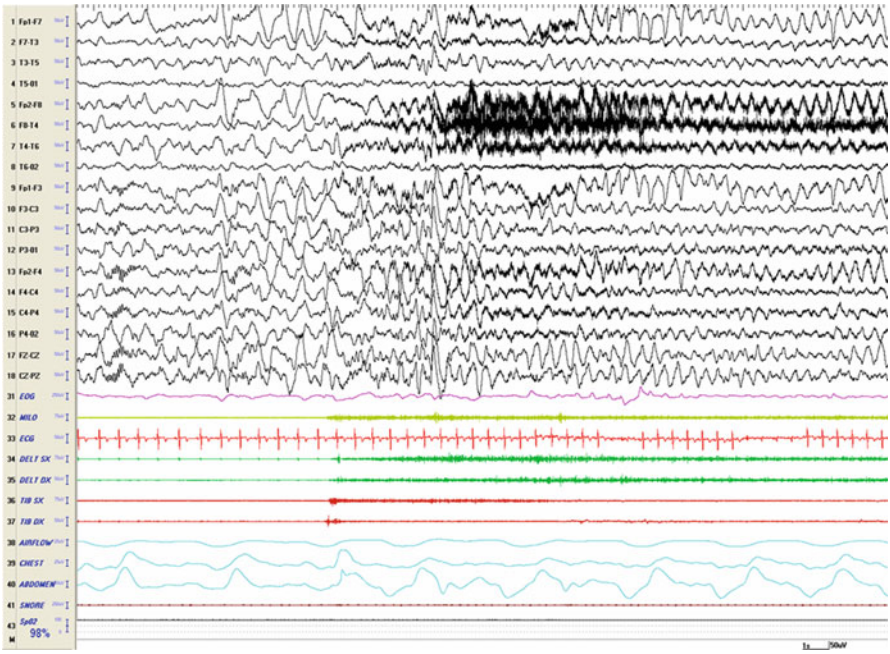

Fig. 14.1 Sleep EEG recording of an episode of confusional arousal in an 8-year-old child, occurring in N3 sleep stage. Notice the presence of a burst of delta waves immediately preceding the onset of the episode (corresponding to the increase of muscle tone) and the persistence of a hypersynchronous delta activity mainly expressed over the frontal regions. *EOG* electrooculogram, *MILO* chin, *ECG* electrocardiogram, *DELT SX* left deltoid muscle, *DELT DX* right deltoid muscle, *TIB SX* left anterior tibial muscle, *TIB DX* right anterior tibial muscle

with NREM parasomnia. The important finding was a higher CAP rate in patients with sleepwalking/sleep terrors in comparison with controls; the instability of NREM sleep in these patients was present also during non-sleepwalking nights [37, 40, 41]. Similarly, polysomnographic recordings have shown that, compared with controls, sleepwalkers experience a higher number of microarousals and arousals during slow-wave sleep [42].

In summary, these data indicate that NREM parasomnias are characterized by an increase in NREM sleep instability and arousal oscillations together with an inability to maintain stable and consolidated slow-wave sleep (for more details see Pathophysiology section).

Differential Diagnosis

NREM parasomnia needs to be distinguished from other parasomnias (in particular RBD and nightmare disorder), nocturnal panic attacks, and sleep-related seizures.

REM Behavior Disorder (RBD)

There is often considerable overlap of features between the disorders of arousal (in particular sleepwalking) and the RBD. However, this overlap can be observed much more frequently in adults than in children, where RBD is very rare. In general, the main features that allow distinguishing RBD from an arousal disorder are the dream enactment behavior, the usual occurrence during the second half of the night, and the absence of mental confusion upon awakening. Some patients may meet the diagnostic criteria for both NREM and REM parasomnias; these patients are diagnosed with "parasomnia overlap disorder."

Nightmares

Nightmares can sometimes resemble sleep terrors (for differential diagnostic features, see Table 14.5). Nightmares occur within REM sleep and are therefore more prominent in the second half of the night; children arousing from a nightmare usually become fully alert quickly, respond positively to comforting, and may offer a detailed description of dream content after awakening the following morning. Compared to sleep terrors, nightmares are characterized by lower levels of autonomic activation (e.g., palpitations or dyspnea), vocalization, and mobility, but are often associated with much more anxiety and subsequently difficulty returning to sleep [43, 44].

Table 14.5 Differential diagnosis between sleep terror and nightmares

	Sleep terror	Nightmares
Peak time of occurrence	First third of the night (from SWS)	Last third of the night (from REM sleep)
Sex	Males > females	In children males = females
Age	4–12 (peak at 5–7)	Any age (frequent at age 3–6)
Prevalence	3–4 % in children	10–20 % in children
Ictal behavior	Heartbreaking cry, screams	Scary awakening
Consciousness	Disoriented, confused	Fully alert after awakening
Vocalization	Common	Rare
Autonomic activity	Intense	Low/mild
Amnesia	Frequent	Absent
Dream recall	Absent	Present (scary vivid dream)
Familial history	Present	Absent
Complications	Potentially injurious and violent	Rarely injurious or violent
Predisposing factors	Sleep deprivation, febrile illness	Stress, traumatic events, personality disorders
Treatment	Safety, avoid predisposing factors, benzodiazepines	None, psycho-/behavioral therapy

Nocturnal Panic Attacks

Nocturnal panic attacks consist in waking from sleep in a state of panic, with intense fear or discomfort. They are frequent in patients with panic disorder, with 44–71 % reporting at least one such attack, and sometimes they can be hardly distinguishable from sleep terrors [45]. As sleep terrors, they can more frequently occur in the first third of the night, during late stage 2 or early stage 3 sleep. However, sleep panic attacks can be distinguished from arousal disorders because patients do not become physically agitated or aggressive during the attack; moreover, immediately after the episodes, they appear oriented, can vividly recall their attack, and usually have difficulty returning to sleep [45].

Nocturnal Frontal Lobe Epilepsy

Nocturnal frontal lobe epilepsy (NFLE) is a syndrome of heterogeneous etiology, encompassing genetic, lesional, and cryptogenetic forms [46–48]. NFLE is usually considered a benign clinical epileptic syndrome because seizures occur almost exclusively during sleep, and in the majority of patients, the pharmacological treatment is effective; however, severe and drug-resistant forms, occasionally associated with mental retardation, have been described [49].

During 1990s, the definition of autosomal dominant nocturnal frontal lobe epilepsy (ADNFLE) was introduced for the first time because of the observation of the occurrence of sleep-related motor seizures in different individuals of the same family [50].

The first genetic studies in ADNFLE families identified different mutations in the gene coding for neuronal nicotinic acetylcholine receptors (nAChRs). In the following years, ADNFLE was quickly recognized as a genetically heterogeneous disorder as most of the described families show mutations in different genes, not involved in the cholinergic system (for a review see Nobili et al. [48]).

The findings of a genetic alteration of the cholinergic system may give some insights into understanding the pathogenesis of this disorder. Indeed, the nAChR is known to exert a modulating effect in the regulation of NREM and REM stability and of arousal oscillations. On the other hand, a mutation of the nicotinic receptors has shown to facilitate the occurrence of an unbalanced excitation/inhibition circuitry within the GABAergic reticular thalamic neurons, thus generating seizures through the synchronizing effect of spontaneous oscillations in thalamocortical connections [51]. Thus, it seems that a genetic alteration observed in NFLE might facilitate both the epileptogenesis and the occurrence of arousal instability [52]. With these assumptions, the high prevalence of parasomnias in the personal and family history of individuals with NFLE [21] might rely on a common alteration of the arousal regulating system.

Considering the clinical aspects, in NFLE patients seizures usually begin before the age of 20 years, with a peak during childhood, although onset during adulthood has been also reported; seizure frequency is usually high. A distinctive characteristic

of NFLE, common to the sporadic and the familial form, is that, in the space of a single night, NFLE patients may show a large number of different sleep-related motor attacks of increasing complexity and duration. These include:

1. Short-lasting (2–4 s) stereotyped movements involving the limbs, the axial musculature, and/or the head [53].
2. Paroxysmal arousals (PAs), characterized by abrupt episodes of arousals (5–10 s in duration) sometimes accompanied by stereotyped movements, vocalization, frightened expression, and fear [54].
3. Major attacks, lasting 20–30 s, characterized by asymmetric tonic or dystonic posturing or complex movements such as pelvic thrusting, pedaling, choreoathetoid, and ballistic movements of the limbs [47].
4. Epileptic nocturnal wandering, consisting of ictal deambulatory behaviors often associated with frightened expression and fear [55].

In recent years it has been shown that sleep-related complex motor attacks may also originate from the temporal lobe [56, 57], the insular–opercular region [58], and the posterior cerebral regions [59].

Considering the electrophysiological features, interictal and ictal scalp EEG abnormalities in NFLE patients are often scanty or absent, probably due to the inaccessibility of much of the frontal lobes to surface electrodes and to the presence of movement artifacts related to seizures [47, 49].

Taking into account the similarities and the possible coexistence of parasomnias in people with NFLE, the differential diagnosis between these disorders appears sometimes complicated, especially if it is only based on anamnestic investigations. A reliable semeiological description of motor events occurring during the night is often difficult to collect from a witness or sleep partner because observers may be lacking or, if present, not fully reliable or awake when attacks occur.

Main anamnestic differences between NFLE and NREM parasomnias are summarized in Table 14.6.

Different questionnaires and scales have been reported in the literature to help differentiate these disorders on the basis of clinical features. Indeed, in 2006 Derry et al. [60] developed the frontal lobe epilepsy and parasomnia (FLEP) scale in order to establish how reliably features from the history might distinguish NFLE from parasomnias. Although initially reported to have a sensitivity of 1 and specificity of 0.9, Manni et al. [61] challenged the usefulness of the scale after studying a tertiary sleep center population. They found that the FLEP scale risked misdiagnosing some patients, especially NFLE subjects presenting episodes of nocturnal wandering. More recently, Bisulli et al. [62] identified two major anamnestic patterns (i.e., dystonic posturing or hyperkinetic behavior) for NFLE diagnosis with a high specificity and unsatisfactory sensitivity. In addition, they found four minor features that may increase the specificity of these clinical items when associated with one of the two major patterns: unstructured vocalization, experience of an aura preceding the motor attack, duration less than 2 min, and a history of tonic–clonic seizures during sleep. This study confirms the weakness of the clinical history alone in differentiating NFLE from parasomnias and underlines the need of future efforts to develop a

Table 14.6 Differential diagnosis between NREM parasomnia and nocturnal frontal lobe epilepsy

	NREM parasomnia	NFLE
Age at onset	3–8 years	Any age (peak in childhood)
Familial history	Frequently present	Possible
Peak time of occurrence	Usually during the first third	Any time
Sleep-stage onset of episodes	NREM sleep (usually N3)	NREM sleep (usually N2)
Frequency during one night	Usually one episode/night	Several episodes/night
Frequency	Sporadic	Almost every night
Duration	1–10 min	Seconds to 3 min
Evolution	Tend to disappear	Stable, increased frequency, rare remission
Predisposing factors	Frequent (sleep deprivation, febrile illness)	Rare
Stereotypic motor pattern	No	Yes
Consciousness	Usually impaired	Usually preserved
Amnesia	Frequent	Unconstant

reliable algorithm to aid physicians in the diagnostic process of paroxysmal motor sleep disorders.

Many experts consider vPSG the "gold-standard" test for diagnosing paroxysmal sleep-related events, but it is expensive, with a limited availability, and does not always capture the event in a single-night recording. Moreover, interictal EEG fail to disclose epileptiform abnormalities in a substantial percentage of NFLE patients [49]. Conversely, interictal epileptiform abnormalities may occur in some parasomnias. Finally, even when the nocturnal episode has been recorded, the diagnosis can remain doubtful because ictal scalp EEG often fails to disclose epileptiform abnormalities or because the episode captured is a minor motor event for which the diagnosis is not reliable, even among experts.

To make video analysis of nocturnal paroxysmal events more reliable, a diagnostic algorithm focusing on the semeiological features of the arousal parasomnias and NFLE has recently been proposed [63]. In their work, Derry et al. noticed that the discrepancy between historical account and recorded events was more evident in NREM parasomnias than in NFLE. Moreover, the clinical features of the initial arousal behaviors (abrupt or slow movements) were indistinguishable between the two conditions, thus confirming that epileptic minor events and paroxysmal arousals cannot be easily differentiable from non-epileptic events on the basis of video-EEG analysis. In contrast, the clinical features of the evolution and the offset of the events could better differentiate NFLE from parasomnias. Finally, the presence of a coherent speech and a verbal interaction with the neighboring individuals during the episode, the possibility to modify the event by the actions of individuals present, and the absence of a clear and distinct offset of the attack seemed to be highly indicative of a NREM parasomnia.

Despite the limits of vPSG, the possibility of analyzing the video of the nocturnal attack remains an important diagnostic tool, making home video recording a useful adjunct [64].

Treatment

To date, no properly powered randomized controlled trials assessing medical and psychological treatment efficacy have been conducted in patients with NREM parasomnia. Indeed, current treatment recommendations are based only on small clinical trials as well as clinical and anecdotal evidence [65–67]. Patients should therefore be advised that prescribed drugs are considered "off-label."

Parasomnic attacks in healthy children and adolescents are often benign and normally require no treatment. Reassuring the patient and significant others about the generally benign nature of the episodes is sometimes sufficient. Especially in the case of sleepwalking, environmental safety issues should be discussed with the parents and represent a first-line approach. Physicians should always evaluate the possible presence of favoring and precipitating factors, including inadequate lifestyle, coexisting sleep disorders, and drugs. Pharmacotherapy should be considered only when the episodes are frequent or dangerous to the patient or others or when they cause undesirable secondary consequences, such as excessive daytime sleepiness, or cause distress to the patient or family.

Reassurance and Environmental Safety

In the majority of NREM parasomnic episodes during childhood, the most disturbing characteristics may sometimes be limited to what is experienced by an observing parent. In these cases, reassurance on the typically benign nature of episodes is often enough. Relatives should be also aware that most affected children outgrow the condition by late adolescence or sooner.

Moreover, parents should avoid any attempt at interrupting the episode because this practice may increase confusion and precipitate a dramatic or even violent reaction. Indeed, efforts to shorten parasomnic events may lead to aggressive behaviors because of the physical proximity and provocation [27, 68]. It is preferable to wait until the episode is over and then guide the child quietly back to bed [67].

Modifications of the environment may be necessary depending on the characteristics of the episodes to minimize the risk of injury. Safety recommendations should be addressed and tailored to each individual. Preventive measures can include locating the patient's bedroom on the ground floor, blocking access to stairs and kitchen, covering windows with heavy curtains, placing mattresses on the floor, using sleeping bags to reduce wandering, and eliminating any potentially dangerous objects [43, 67].

Bedroom door alarms may be used to signal the occurrence of a wandering episode; however it has been shown that a loud stimulus can worsen the behavior of the sleepwalker [69].

Predisposing and Triggering Factors

As mentioned above, all the conditions increasing the amount of slow-wave sleep increase the likelihood of occurrence a parasomnic episode [70]. Indeed, a careful history about sleep patterns and duration should be collected, including evaluation of the night-to-night stability of sleep achieved, periods of relative sleep deprivation, occurrence of recuperative sleep, and nap history [43]. Sleep hygiene should be recommended, including advising routine naps for children <4 years of age to ensure adequate sleep.

Moreover, considering that arousal induced by whatever external or internal stimuli (noise, light, pain, nocturia) could precipitate an episode, specific measures to identify and remove these triggers will lessen the arousals and consequently help in resolution of the parasomnias [67, 71]. In particular, when parasomnic episodes become frequent and intractable or are associated with daytime mood or behavioral disturbance, the possibility of comorbid sleep disordered, especially sleep apnea, but also periodic limb movements and gastroesophageal reflux, must be recognized and treated [67, 71].

Non-pharmacological Treatment: Psychological Interventions and Anticipatory Awakenings

A variety of non-pharmacological treatments has been recommended for long-term management of NREM parasomnias, including hypnosis, autogenic training, relaxation therapy, psychotherapy, and cognitive behavioral therapy. However, the evidence for these methods is based mainly on anecdotal data and case reports [67]. Hypnosis (including self-hypnosis) has been found to be effective in both children and adults presenting with chronic sleepwalking and sleep terrors [72, 73]. However, hypnosis in children can be often difficult, and individuals show varying degrees of susceptibility for this therapeutic approach.

Anticipatory awakening or scheduled awakening is another behavioral technique that can be recommended as an effective therapy when the episodes occur nightly and consistently at or about the same time each night. Since arousal parasomnia tends to be clustered into the first third of night sleep, in particular during slow-wave sleep, momentary awakening of the child by the parents, 15–20 min prior to the usual time of occurrence, may shift the child into a lighter state of sleep, thereby aborting the event. During the scheduled awakening, the parent should comfort the child. Anticipatory awakening seems to be effective in about 60 % of cases [66]. This technique can represent an important therapeutic option, particularly if the family is reluctant to administer a drug to the child and inclined toward non-pharmacologic management. The disadvantages are that it requires nightly vigil and intervention by the parents; also, sometimes the interruption itself may provoke a frank parasomnia [66].

Pharmacological Treatment

Clinicians should consider therapy only if the episodes cause undesirable secondary consequences, such as excessive daytime sleepiness, or cause distress to the patient or family. Pharmacologic interventions include benzodiazepines such as diazepam 5–10 mg or clonazepam 0.5–2 mg [74] and tricyclics such as imipramine or clomipramine [67]. The effectiveness of benzodiazepines may relate to sedative effects or to decreases in slow-wave sleep.

Other serotoninergic antidepressants, in particular paroxetine, have been reported to be particularly effective in the treatment of sleep terrors. On the other hand, paroxetine has been shown to favor episodes of sleepwalking thus suggesting possible distinct pathophysiological mechanisms between sleep terrors and sleepwalking [74].

An open pharmacological trial of L-5-hydroxytryptophan (2 mg/kg at bedtime) suggests its efficacy in the treatment of sleep terrors. L-5-Hydroxytryptophan is a precursor of serotonin that may modify central serotoninergic system dysfunction or enhance production of sleep-promoting factors [75]. Finally, some case studies have suggested that melatonin therapy, at 5 mg, half an hour before bedtime, may be helpful for patients with sleepwalking and sleep terrors [76].

Sleep-Related Eating Disorder (SRED)

SRED consists of "recurrent episodes of involuntary eating and drinking during arousals from sleep, associated with diminished levels of consciousness and subsequent recall, with problematic consequences" [1]. Episodes typically occur during partial arousals from sleep during the first third of the night, with impaired subsequent recall [77]. This disorder is potentially harmful; problematic features of the recurrent sleep-related eating include the following: consumption of abnormal combinations of food or toxic substances, sleep-related injurious behaviors performed while in pursuit of food [78, 79], adverse health consequences (weight gain, various metabolic problems), and daytime sleepiness.

The prevalence of SRED in the general population is unknown. Winkelman et al. reported that 16.7 % of individuals who were part of an inpatient eating disorder program, 8.7 % of those in an outpatient eating disorder program, 4.6 % of college students, 1.0 % of obese individuals in a weight loss program, and 3.4 % of those in an outpatient depression clinic reported behavior consistent with SRED [80]. SRED is found predominantly in women, and the average age of onset is approximately 22–27 years, with a mean of approximately 12–16 years before clinical presentation [80, 81].

A history of other parasomnias, especially sleepwalking, is frequently reported. Patients with SRED share several clinical commonalities with sleepwalkers plus previous or current eating behavior problems. It suggests that they have specialized

a former sleepwalking behavior toward sleep-related eating because they are more vulnerable to eating behavior problems during the daytime [82].

Other sleep disorders can be associated with SRED, in particular restless legs syndrome (RLS), periodic limb movement of sleep (PLMS), and sleep apnea [80, 81]. Most of these sleep disorders can increase arousals during sleep and precipitate NREM parasomnia episodes in predisposed individuals. Several psychiatric conditions have been associated with SRED, including depression, bipolar disorder, anxiety, posttraumatic stress disorder, and history of repeated abuse. Finally, many drugs have been implicated in the initiation of SRED, including zolpidem, triazolam, amitriptyline, olanzapine, and risperidone [83].

Considering the management of SRED, a treatment of possibly associated sleep disorders is essential. Some drugs have been reported to be effective for the treatment of SRED such as topiramate and dopaminergic agents alone or in combination with benzodiazepines (mainly clonazepam) or opiates [83, 84].

REM-Related Parasomnia

REM Sleep Behavior Disorder (RBD)

Physiologic REM sleep is characterized by an activated brain state in combination with skeletal muscle paralysis. In RBD, normal atonia is lost, and patients present recurrent episodes of dream-enacted behaviors that can vary from small hand movements to violent activities, such as punching, kicking, or leaping out of bed. RBD is also associated with electromyography (EMG) abnormalities during REM sleep, including an excess of muscle tone and/or an excess of phasic EMG twitch activity during this sleep stage. A change in the pattern and frequency of dream recall is frequently described, and dreams can often have a negative emotional content. Accordingly with ICSD3, RBD requires polysomnography for making a diagnosis [1]. The key features of RBD on polysomnography are preserved chin electromyographic tone, or "REM sleep without atonia" (RSWA), and video evidence of motor dream enactment in the form of increased physical activity, including aggressive or violent behaviors.

RBD has been considered for a long time a parasomnia that occurs almost exclusively in elderly men. However, it is now recognized as a disorder of all ages and both sexes. In adults, there is clear association of RBD with synucleinopathic degenerative disorders such as Parkinson disease, dementia with Lewy bodies, and multiple system atrophy. In a minority of cases, RBD represents a side effect of treatment with drugs such as antidepressants (tricyclics, selective serotonin reuptake inhibitors (SSRIs), and selective noradrenaline reuptake inhibitors) and lipophilic beta-blockers. Different animal and human studies have suggested that lesions or dysfunction in REM sleep and motor control circuitry in the pontomedullary structures cause RBD phenomenology, and degeneration of these structures might

Table 14.7 Etiology of rapid eye movement sleep behavior disorder in childhood

Hypersomnias of central origin
Narcolepsy type 1
Narcolepsy type 2
Idiopathic hypersomnia
Neurodevelopmental–neurodegenerative disorders
Autism
Attention deficit disorder
Smith–Magenis syndrome
Moebius syndrome, juvenile
Parkinson disease
Tourette syndrome
Neurofibromatosis type 1
Structural brainstem abnormalities
Pontine glioma
Chiari malformation type 1
Drugs
Selective serotonin reuptake inhibitors
Tricyclic antidepressants

explain the presence of RBD years or decades before the onset of parkinsonism or dementia in people who develop neurodegenerative disorders [85].

Cases of RBD in childhood and adolescence are very infrequent, and the literature is composed only of single case reports or small case series [86–88]. Because of the rarity of these forms and the lack of long-term follow-up data, little is known about the natural history of early-onset RBD. RBD in children is virtually never idiopathic and is usually associated with narcolepsy or idiopathic hypersomnia, neurodevelopmental–neurodegenerative disorders, or structural brainstem abnormalities or represents a side effect of pharmacological agents, such as SSRI agents (Table 14.7).

RBD can occur in both children and adults with narcolepsy [89]. The prevalence of this parasomnia in narcolepsy, especially when associated with cataplexy, seems to be high, ranging from 36 to 60 % [90, 91]. RBD usually develops after hypersomnia and cataplexy onset; however, in a very few patients, especially during childhood, RBD can represent the first manifestation of the disease [92]. RBD in narcoleptic patients shows some distinct phenotypic features with respect to other RBD patients [90]. In particular, clinical severity is usually less aggressive and violent with a predominance of elementary jerks rather than complex behaviors and vocalizations; moreover there is no male predominance and an earlier onset. It has been hypothesized that hypocretin/dopaminergic system deficiency may lead to motor dyscontrol during REM sleep in narcolepsy, as hypocretin/dopaminergic neurons have wide projections to different nuclei that regulate REM sleep atonia (e.g., subcoeruleus nucleus) and the emotional content of dreams (e.g., central nucleus of the amygdala) [88].

In another group of patients, childhood RBD is associated with neurodevelopmental disabilities, such as autism. In autistic children, a decrease activation of GABA transmission has been described. GABA represents the main neurotransmitter of the ventral gigantocellular nucleus, and its decrease might predispose to a loss of the inhibition of spinal motor neurons during REM sleep [88].

Finally, RBD can be induced by specific drugs, such as selective serotonin reuptake inhibitors. During physiologic REM sleep, the serotoninergic neurons descending to motor neurons cease firing, leading to hypotonia. In this perspective, drugs that stimulate the serotonin system can induce RBD, possibly because they prevent normal sleep-related hypotonia [85].

RBD treatment is basically symptomatic. The main indications for RBD management encompass the preventive measures, a reevaluation of drugs that can precipitate or worsen RBD, and the use of drugs aimed at blunting the motor-behavior manifestations. Although no randomized double-blind trials exist, two agents have been shown to be beneficial: clonazepam (0.5–2 mg at bedtime) and melatonin (3–12 mg at bedtime). Clonazepam seems to have a suppressing effect on phasic locomotor activity and a positive influence on mental dream activity. The mechanism by which melatonin can restore the REM-related muscle atonia remains substantially unknown.

Considering childhood RBD, treatment is either not mentioned in some reports or given at unspecified dosages; however, in the short term, it seems to be modestly responsive to benzodiazepines or melatonin [88].

Recurrent Isolated Sleep Paralysis

Recurrent isolated sleep paralysis (RISP) is defined as "an inability to perform voluntary movements at sleep onset (hypnagogic or predormital form) or on waking from sleep (hypnopompic or postdormital form) in the absence of a diagnosis of narcolepsy" [1]. During the episodes consciousness is preserved and full recall is present. Hallucinations such as a feeling of the presence of others nearby, pressure on the chest, or hearing footsteps are common. A single episode usually lasts seconds to minutes and spontaneously resolves or can be halted by external auditory or tactile stimulation from a bed partner. The sensation of being paralyzed can cause intense anxiety. Although diaphragmatic function is not affected, difficulties in breathing may be reported.

RISP is considered to represent a condition of state dissociation, with a persistence of REM sleep into wakefulness.

Due to the differences in the definition used and in sampling methods, estimates of the prevalence vary widely, between 6 and 40 %. The lifetime prevalence of sleep paralysis, based on a large systematic review, is estimated to be 7.6 % of the general population, 28.3 % of students, and 31.9 % of psychiatric patients [93]. No consistent sex differences have emerged and the mean age of onset is 14–17 years.

Main predisposing factors for RISP are sleep deprivation and irregular sleep–wake schedules. The episodes seem to occur most frequently in the supine position. An association with anxiety/psychiatric disturbances has been described.

The management of RISP consists in reassurance about the benign nature of the episodes and in the avoidance of sleep deprivation and other triggering factors. Recurrent episodes may be treated with REM-suppressing agents such as low doses of tricyclic agents, clonidine, or clonazepam.

Nightmare Disorder

Nightmare disorder is characterized by "recurrent, highly dysphoric dreams, which are disturbing mental experiences that generally occur during REM sleep and that often result in awakening" [1]. Full alertness upon arousal and intact recall of the frightening dream are generally characteristic of the episodes. As REM sleep is more represented in the second half of the night, episodes tend to occur more frequently in the early hours of the morning. Nightmare content consists often in dream sequences that seem vivid and real. Emotions are characteristically negative and most frequently involve anxiety and fear but also anger, rage, embarrassment, and disgust. Monsters or other fantastical imageries often characterize the dreams of young children, whereas adolescent and adults may experience more realistic images derived from daytime stressors or traumatic events. Dream descriptions in preschool age children are usually short and simple, while older children may elaborate the dream content by adding fantastic features. There is rarely any physical movement during dreams because of the REM-induced atonia; somatic manifestations of anxiety such as tachycardia, sweating, and tachypnea may occur. The episodes are generally brief but there may be post-awakening anxiety and difficulty returning to sleep.

Occasional nightmares are very common in children, ranging from 60 to 75 % [94]. However, occasional nightmares do not constitute a nightmare disorder. In preadolescent children the prevalence of nightmare disorder was estimated to be 1.8–6 % [95]. Nightmare onset typically occurs between ages 3 and 6. The prevalence of nightmares decreases as children aged, although they are still common among adults. Close to a third of adults with recurrent nightmares have onset of the symptom during childhood. Nightmares are commonly seen in those who have been physically or sexually abused and in those suffering from posttraumatic stress disorder. A strong association with anxiety disorders has been also described.

Therapy with beta-blockers and dopaminergic agonists and withdrawal from REM-suppressing medications, such as selective serotonin reuptake inhibitors, tricyclic antidepressants, hypnotics, and alcohol, may precipitate or increase the severity of nightmares.

The diagnosis of nightmares is relatively simple, although it is important to ascertain with detail the main features of the event to rule out other sleep disorders, particularly sleep terrors (for details see the Differential Diagnosis section in NREM Parasomnia chapter).

Occasional nightmares do not require specific treatments but only behavioral suggestions such as reassurance of children about the unreal nature of dreams and avoidance of television viewing within 2–3 h of bedtime. Recurrent nightmares may benefit from psychological, pharmacological, or combined treatments, although studies in this field are scanty.

Rescripting techniques, in which parents/therapist and children discuss the dream and invent less frightening ending, can be helpful. Similarly, desensitization toward dream content can help the child to feel more in control of the nightmares, which may also serve to reduce anxiety. Encouraging children to write about or draw their dreams may also yield positive results.

Prazosin, risperidone, and trazodone are the most widely used drugs in treating nightmares.

Other Parasomnias

Exploding Head Syndrome (EHS)

Exploding head syndrome is characterized by a "sudden, loud imagined noise or sense of a violent explosion in the head occurring as the patient is falling asleep or waking during the night" [1, 96]. The abnormal sensation usually lasts a few seconds and is usually accompanied by a sense of fright. There have been reports of associated perceptions of a flash of light, a myoclonic jerk, or a brief stab of head pain. Patients range from having one episode in a lifetime to recurrent episodes per night. In this last case, an insomnia complaint may develop as a result of the recurring arousals.

In the majority of patients, predisposing factors are not recognized; however, some subjects report increased numbers of attacks when under personal stress or overtired. EHS can precede other neurological conditions, such as migraine attacks or sleep paralysis.

There are little systematic epidemiological data on this sleep disorder. It has been hypothesized to have a typical age of onset of over 50 years and to be more common in women and in those suffering from ISP. However, a recent study conducted in 211 undergraduate students using semi-structured diagnostic interviews assessing for both EHS and ISP showed that 18 % of the sample experienced lifetime exploding head syndrome and 16.6 % presented recurrent episodes without a female prevalence. An association with ISP was found in 36.89 % of subjects [97].

The neurophysiologic mechanisms underlying EHS are unknown. An asynchronous switch-off of different cortical regions (visual, acoustic, motor), leading to a prominent burst of neuronal activity, is the most popular pathogenetic hypothesis.

The cornerstone of management in EHS is reassurance and education, as this is a benign condition that remits over time in most patients. Some case reports describe the efficacy of tricyclic antidepressants (clomipramine) and calcium channel blockers (flunarizine) in patients with recurrent EHS.

Sleep-Related Hallucinations

Sleep-related hallucinations are "hallucinatory experiences that occur at sleep onset (hypnagogic) or on awakening from sleep (hypnopompic)" [1]. They are predominantly visual but may include auditory, tactile, or kinetic phenomena. Complex nocturnal visual hallucinations may represent a distinct form of sleep-related hallucinations. They typically occur following a sudden awakening, without recall of a preceding dream. They usually take the form of complex, vivid, relatively immobile images of people or animals, sometimes distorted in shape or size. These hallucinations may remain present for many minutes but usually disappear if ambient illumination is increased.

Hypnagogic and hypnopompic hallucinations can be associated with narcolepsy, but a high prevalence in the normal population is also described. Studies reported a prevalence of 25–37 % for hypnagogic hallucinations and of 7–13 % for hypnopompic hallucinations. Both hypnagogic and hypnopompic hallucinations are more common in younger persons and occur slightly more frequently in women than in men.

On the contrary, complex nocturnal visual hallucinations are often associated with a variety of underlying disorders typical of the elderly, such as visual loss (Charles Bonnet syndrome), Lewy body disorders, and pathology of the mesencephalon and diencephalon (peduncular hallucinosis).

Little objective information is available regarding the management of sleep-related hallucinations. Most often reassurance is sufficient. Tricyclic antidepressants have been suggested for hypnagogic and hypnopompic hallucinations.

Sleep Enuresis

Sleep enuresis (SE) is characterized by "recurrent involuntary voiding that occurs during sleep. In primary SE, recurrent involuntary voiding occurs at least twice a week during sleep after 5 years of age in a patient who has never been consistently dry during sleep for six consecutive months. SE is considered secondary in a child or adult who had previously been dry for six consecutive months and then began wetting at least twice a week. Both primary and secondary enuresis must be present for a period of at least three months" [1]. Primary and secondary SE is considered distinct phenomena with different etiologies and courses. SE is defined as *monosymptomatic* when the subject has no associated daytime symptoms of bladder dysfunction (such as wetting, increased voiding frequency, urgency, jiggling, squatting, and holding maneuvers). But, usually, when a meticulous history is obtained, the majority of children have at least some light daytime void symptoms, and their SE is classifiable as *non-monosymptomatic* [98].

SE is not specific to one stage of sleep and can occur during either NREM or REM sleep. Most enuretic episodes happen during the first half of the night.

From a developmental point of view, complete control of the bladder at night is usually achieved by the age of 5 years; thus bed-wetting in toddlers is physiologic.

The prevalence of NE is between 6 and 10 % at age 7, decreasing to 2 % at 15 years and 0.5–2 % in adults. Approximately 75–90 % of patients with SE have a primary form, while 10–25 % have secondary NE. SE is more frequent in boys than in girls under 11 years of age. After 11 years there is no difference between sexes. This sex-related difference can be due to a different time of sex-related brain or bladder development. The spontaneous annual remission during childhood is about 15 %, and this natural history should be kept in mind when counseling parents about the prognosis of the disorder.

There seems to be a strong genetic predisposition for primary SE. The reported prevalence is 77 % when both parents were enuretic as children and 44 % when one parent has a history of enuresis.

Sleep disorders that fragment sleep such as sleep apnea and periodic leg movements are frequently associated with SE, and treatment of these disorders may cure or reduce their incidence.

While primary SE is a typical childhood disorder, secondary SE can occur at any age.

Indeed, secondary SE is more commonly associated with organic factors such as the following: urinary tract infections, malformations of the genitourinary tract, extrinsic pressure on the bladder (such as chronic constipation or encopresis), medical conditions that result in an inability to concentrate urine (diabetes mellitus or insipidus, sickle cell disease), increased urine production secondary to excessive evening fluid intake (caffeine ingestion, diuretics, or other agents), neurologic diseases (spinal cord abnormalities with neurogenic bladder or seizures), and psychosocial stressors (parental divorce, neglect, physical or sexual abuse, and institutionalization).

Current pathophysiological model hypothesizes that SE results from three interrelated factors: nocturnal polyuria, decreased nocturnal bladder storage ability, and poor arousal to the stimulus of a full bladder. In particular, different studies hypothesized that children with primary SE should show a delay in achieving the normal increase in vasopressin release during sleep, thus developing nocturnal polyuria that exceeds the bladder capacity. If these children do not arouse to the sensation of a full bladder, primary SE can occur. In particular, children with enuresis are often described as “deep sleepers”, and their arousal threshold seems to be more elevated in all sleep stages with respect to controls [98].

The management of NE starts from some simple strategies, such as lifting or wakening, rewarding dry nights, bladder training (including retention control training), and fluid restriction.

Alarm systems that alert and awaken the child if any moisture is detected are considered a first-line treatment, and its effect seems to be more gradual but sustained with respect to drugs [99].

The established drug therapy of polyuric bed-wetting is desmopressin, a synthetic analog of the antidiuretic hormone arginine (vasopressin) that decreases nocturnal urine production and increases urinary osmolality. Desmopressin is particularly helpful for short-term use, when a rapid response is needed and seems

to have some positive effects in about 70 % of treated children [99]. Finally imipramine and oxybutynin may control enuresis by decreasing the parasympathetic tone of the bladder detrusor muscle.

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