

## NREM parasomnias

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### INTRODUCTION

Parasomnias are undesirable physical or behavioral phenomena that occur during entry into sleep, within sleep, or during partial arousals from sleep (American Academy of Sleep Medicine, 2005). The focus of this chapter is confusional arousals, sleepwalking (somnambulism), and sleep terrors. These sleep disorders constitute the prototypic nonrapid-eye-movement (NREM) sleep parasomnias and are collectively termed “disorders of arousal” (Broughton, 1968) because of the autonomic and motor arousal that propels the patient towards partial wakefulness. A summary and comparison of the main features of NREM and REM sleep parasomnias are presented in Table 52.1.

Disorders of arousal are more common in childhood than in adulthood and their prevalence rate decreases significantly with age. However, whereas the occurrence of NREM parasomnias in children is frequently viewed as a relatively benign and common event that will resolve spontaneously, these disorders often pose greater problems, including social inconvenience and sleep-related injury, in affected adults. In fact, injurious NREM sleep parasomnias in adults may be more prevalent than commonly believed (Schenck et al., 1989; Ohayon et al., 1999; Mahowald and Schenck, 2000c).

The symptoms and manifestations of these NREM parasomnias can be considered along a spectrum. For instance, the patient's affective expression can range from calm to extremely agitated, and the actual physical behaviors can range from simple and isolated actions (e.g., sitting up in bed, mumbling, fingering bed sheets) to complex behaviors (e.g., rearranging furniture, inappropriate sexual activity, playing a musical instrument, driving an automobile). Moreover,

an episode can be comprised of two overlapping disorders, such as a sleep terror followed by sleepwalking.

Disorders of arousal share a number of characteristics. Most episodes arise from sudden but incomplete arousal from slow-wave (stages 3 and 4) sleep (Jacobson et al., 1965; Kavey et al., 1990; Espa et al., 2000) and sometimes from stage 2 sleep (Kavey et al., 1990; Zucconi et al., 1995; Joncas et al., 2002). Consequently, these parasomnias tend to occur in the first third of the sleep period when slow-wave sleep (SWS) is predominant. Episodes are generally characterized by misperception and relative unresponsiveness to external stimuli, mental confusion, automatic behaviors, and variable retrograde amnesia. This state is indicative of a high arousal threshold. A common genetic component is also suspected, as a positive family history is often reported by people with an arousal disorder (Hublin et al., 1997, 2001; Hublin and Kaprio, 2003).

Factors that deepen sleep, such as intense physical activity (Vecchierini, 2001), hyperthyroidism (Ajilouni et al., 2005), fever (Dorus, 1979; Kales et al., 1979; Larsen et al., 2004), sleep deprivation (Rauch and Stern, 1986; Mayer et al., 1998; Joncas et al., 2002), and neuroleptics (Charney, 1979; Landry and Montplaisir, 1998) or medications with depressive CNS effects (Lee-Chiong, 2002; Mahowald, 2002), can facilitate or precipitate NREM parasomnias in predisposed individuals. Factors that fragment sleep, including sleep-disordered breathing (Guilleminault et al., 1998; Espa et al., 2002; Guilleminault et al., 2005a), periodic leg movement syndrome (Guilleminault et al., 2003), stress (Kales et al., 1980b; Klackenberg, 1982; Crisp et al., 1990; Ohayon et al., 1999), and environmental or endogenous stimuli (Gastaut and Broughton, 1965; Kales et al., 1966; Broughton and Gastaut, 1974), can

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Table 52.1

## Comparison of common features of NREM and REM sleep parasomnias

	Confusional arousal	Somnambulism	Sleep terrors	Nightmares	RBD
Time of night	First third to half of sleep period	First third to half of sleep period	First third to half of sleep period	Last half of sleep period	Last half of sleep period
Sleep stage	SWS	SWS	SWS	REM	REM without atonia
Associated activity	May sit up in bed	Simple to complex movements. Possible ambulation	Sits, screams. Agitated motor activity	Movements are rare and limited	Behavior that correlates with dream content
Duration	1–15 min	1–30 min	1–10 min	3–20 min	1–10 min
Autonomic activation	Low	Low to moderate	Moderate to extreme	None to moderate	Low to moderate
Recall for the event	Variable amnesia for the event	Variable amnesia for the event	Variable amnesia for the event	Vivid and detailed dream recall	Vivid and detailed dream recall
Full awakening	Uncommon	Uncommon	Uncommon	Common	Common
State after event	Confused/disoriented	Confused/disoriented	Confused/disoriented	Fully awake and functional	Fully awake and functional
Arousal threshold	High	High	High	Low	Low
Reduced in sleep laboratory	Yes	Yes	Yes	Yes	No
PSG findings	Partial arousals from SWS	Partial arousals from SWS	Partial arousals from SWS	REM	Excessive EMG during REM
Potential for injury/violence	Yes	Yes	Yes	No	Yes

EMG, electromyography; PSG, polysomnographic; RBD, REM sleep behavior disorder; REM, rapid eye movement; SWS, slow-wave sleep

have similar effects. Hormonal factors may also influence the frequency with which women experience parasomnias, as sleep terrors and injurious sleepwalking can emerge premenstrually (Schenck and Mahowald, 1995b), and sleepwalking may decrease during pregnancy, particularly in primiparas (Hedman et al., 2002).

Some researchers (Mahowald and Schenck, 1992, 1999, 2005) have cogently argued that a proper understanding of many parasomnias rests on the appreciation that sleep and wakefulness are not always mutually exclusive states and that various variables implicated in generation of wakefulness, REM sleep, and NREM sleep (the three primary states of being) may occur simultaneously, interact dynamically, or oscillate rapidly. It should also be noted that NREM parasomnia-related behaviors are not unlike the natural occurrence of clinically wakeful behavior during physiological sleep documented in the animal kingdom (Almanar and Ball, 1994; Rattenborg et al., 1999).

## CONFUSIONAL AROUSALS

### Clinical features

That people sometimes experience confused awakenings from deep sleep in which they appear to be partially awake and partially asleep was noted over 150 years ago – a condition termed “*ivresse du sommeil*” (French) or “sleep drunkenness”. Other terms used to describe this disorder include *Schaftrunkenheit* (German) and Elpenor syndrome (derived from the story of Elpenor, who broke his neck during such an episode in Homer’s *The Odyssey*).

Confusional arousals, often seen in children, consist of mental confusion or confusional behavior during or following arousals from SWS. They can also occur upon attempted awakening from sleep in the morning or during daytime naps. An arousal will often begin with automatic movements (e.g., playing with bed sheets) and moaning or unintelligible vocalizations, and can progress to thrashing about in bed, violent behaviors towards the self or others, or inconsolable crying. Individuals usually appear confused with slow mentation and have poor reactivity to environmental stimuli; attempts to awaken the person are often unsuccessful and may be met with vigorous resistance. Most episodes last from a few to 15 minutes.

### Prevalence

Confusional arousals are common in infants and young children, and their prevalence in both male and female adults is approximately 3–4% (Ohayon et al., 1999, 2000).

## Pathophysiology

Relatively little is known about the pathophysiology of confusional arousals. A familial pattern appears to exist in families of deep sleepers. Intensified sleep inertia (i.e., a feeling of grogginess after awakening accompanied by a temporarily reduced ability to perform even simple tasks) likely plays a role (Broughton, 2000). One study (Ohayon et al., 1999) of a representative sample in the UK found that people reporting confusional arousals were more likely than individuals without parasomnias to be diagnosed with a mood disorder according to the *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV)*, mood disorder, to consume psychoactive drugs, and to be smokers. An association between confusional arousals and a mood disorder was found in a subsequent epidemiological study (Ohayon et al., 2000). It is possible that symptoms of insomnia or fragmented sleep, which are often associated with mood disorders, result in accumulated sleep deprivation and increased awakenings from NREM sleep, conditions known to facilitate the occurrence of arousal disorders. Similarly, shift or night workers are at higher risk of reporting confusional arousals (Ohayon et al., 2000). People with confusional arousals may also suffer from other sleep disorders, such as obstructive sleep apnea (Ohayon et al., 2000) or hypersomnia (Roth et al., 1972).

## SOMNAMBULISM (SLEEPWALKING)

### Clinical features

Like some other parasomnias, sleepwalking was once thought to be a behavioral manifestation of dreaming-related processes. Sleepwalking is now considered a disorder of arousal involving a physiological dysfunction in the neural regulation of generalized cortical activation. Somnambulistic actions may be complex, such as dressing, playing a musical instrument, or driving a car, and may be performed with substantial dexterity; more often, however, they are mundane, stereotyped, and accompanied by a variable degree of amnesia for the episode.

Far from being a benign condition, somnambulism can result in injury to the sleeper or to others. Adults suffering from somnambulism often consult due to a history of aggressive and/or injurious behavior during sleep (Rauch and Stern, 1986; Schenck et al., 1989; Moldofsky et al., 1995; Denesle et al., 1998). In a significant number of cases, patients report having suffered serious injuries (e.g., contusions, fractures to limbs, rib cage, multiple lacerations) and/or having attacked a bed partner during an episode (Kales et al., 1980c; Rauch and Stern, 1986; Kavey et al.,

1990; Milliet and Ummenhofer, 1999). Furthermore, the number of legal cases of sleep-related violence is on the rise (Cartwright, 2000) and sleepwalking represents one of the leading causes of sleep-related injury (Pareja et al., 2000). In one polysomnographic investigation (Schenck et al., 1989) of 100 consecutive patients consulting for repeated nocturnal injury, 90% received one of two diagnoses: sleepwalking/night terrors (54%) or REM sleep behavior disorder (36%). Reported behaviors during somnambulism and/or agitated sleep terrors included running into walls and furniture, jumping out of windows, leaving the house, driving an automobile, wandering around streets, walking into lakes, climbing ladders, and wielding weapons such as loaded shotguns. A second laboratory investigation of 64 consecutive adult patients with sleepwalking or sleep terrors found that 40% reported a history of sleep-related violence leading to the destruction of property (e.g., breaking of walls, doors, windows, plumbing) or serious self-injury and a further 19% reported harmful but nondestructive behavior (Moldofsky et al., 1995). Similarly, a study of 50 chronic sleepwalkers found that 30% had injured themselves or others in the course of at least one episode (Guilleminault et al., 2005a). Moreover, the fact that the driving motor vehicles, sexual activity, suspected suicide, and even homicide and attempted homicide can occur during somnambulism raises fundamental questions as to the medicoforensic implications of these acts (Oswald and Evans, 1985; Mahowald et al., 1990, 2003, 2005; Broughton et al., 1994; Schenck and Mahowald, 1995a; Rosenfeld and Elhajjar, 1998; Kayumov et al., 2000; Mahowald and Schenck, 2000c; Shapiro et al., 2003).

### Prevalence

The yearly prevalence of somnambulism in children aged 6–16 years ranges from 4% to 17%, peaking at 11–12 years of age, and sleepwalking occurs in 1–4% of adults (Bixler et al., 1979; Klackenberg, 1982; Goldin, 1997; Hublin et al., 1997; Ohayon et al., 1999; Laberge et al., 2000). Although children with sleepwalking tend to outgrow the disorder during mid to late adolescence, somnambulism persists into adulthood in up to 25% of cases (Hublin et al., 1997). In addition, episodes can emerge in adulthood and increase in severity over time (Berlin and Qayyum, 1986; Kavey et al., 1990).

### Pathophysiology

The exact mechanisms that give rise to somnambulism remain unclear. Several general factors have been proposed, including increased psychopathology, genetics,

and deregulation of serotonergic systems. A number of atypical sleep parameters have also been described. The latter are covered in a dedicated section below (see Unusual sleep parameters).

### PSYCHOPATHOLOGY

Traditionally, the presence of somnambulism (with or without concomitant sleep terrors) in adulthood has been viewed as a sign of major psychopathology (Pai, 1946; Kales et al., 1980b; Soldatos et al., 1980). Epidemiological evidence suggests a higher prevalence of psychopathology among adult patients with arousal disorders (Ohayon et al., 1999), and psychopathology has been reported in subgroups of adolescents with sleep terrors and/or sleepwalking (Gau and Soong, 1999). However, several studies have shown that many adult patients do not have a DSM-based (American Psychiatric Association, 1994) Axis I psychiatric disorder, nor do they necessarily present with highly disturbed personality traits (Schenck et al., 1989, 1997; Mahowald and Schenck, 1999; Guilleminault et al., 2005a).

### GENETICS

Several studies have revealed a genetic contribution to sleepwalking. The prevalence of somnambulism is higher in children of parents with a history of sleepwalking, and about 80% of somnambulistic patients have one or more family members affected by the disorder (Abe and Shimakawa, 1966; Kales et al., 1980a; Hori and Hirose, 1995). Barkwin's (1970) study of over 300 twin pairs found that monozygotic (MZ) twins are concordant for the disorder six times as often as dizygotic (DZ) twins. A population-based twin study of 1045 MZ and 1899 DZ pairs showed a considerable genetic effect in adulthood sleepwalking (probandwise concordance 5 times higher in MZ than in DZ pairs), although the effect in childhood sleepwalking was not as pronounced (probandwise concordance 1.5 times higher in MZ than in DZ pairs) (Hublin et al., 1997). HLA-DQB1 typing in sleepwalkers and their families indicates that somnambulism may be associated with excessive transmission of the HLA-DQB1\*05 and \*04 alleles (Lecendreux et al., 2003).

### DEREGULATION OF SEROTONERGIC SYSTEMS

Sleepwalking episodes are four to nine times more common in patients with Tourette syndrome or migraine headaches (Barabas et al., 1983, 1984; Giroud et al., 1987). These observations suggest that somnambulism may be associated with abnormalities in the metabolism of serotonin. That serotonin is involved

in the pathophysiology of sleepwalking has also been hypothesized on the basis that several factors that can precipitate sleepwalking (e.g., sleep-disordered breathing (SDB), certain drugs, fever) implicate the serotonergic system (Juszczak and Swiergiel, 2005).

### Unusual sleep parameters

Sleep architecture and normal cycling among sleep stages are preserved in adult somnambulistic patients. Nevertheless, a number of unusual sleep-related processes have been described including alterations in the cyclic alternating pattern (CAP), an increased number of sudden arousals from SWS, hypersynchronous delta waves, irregular buildup of slow-wave activity, and unique EEG characteristics prior to and during somnambulistic episodes.

#### CYCLIC ALTERNATING PATTERN AND SLOW-WAVE SLEEP AROUSALS

The CAP is a measure of NREM instability that expresses the organized complexity of arousal-related phasic events in NREM sleep (Terzano and Parrino, 2000; Terzano et al., 2001). In comparison with controls, patients with sleepwalking/sleep terrors show increases in the CAP rate (Zucconi et al., 1995). An increased CAP rate was also found in children with chronic sleepwalking and concomitant sleep respiratory disorders (Guilleminault et al., 2005b). Similarly, polygraphic recordings have shown that, compared with controls, sleepwalkers experience a greater number of SWS arousals and brief microarousals (Halasz et al., 1985; Blatt et al., 1991; Espa et al., 2000). The data indicate that an increase in NREM sleep instability and arousal oscillation is a typical microstructural feature of SWS-related parasomnias. These results also suggest that, in addition to being a disorder of arousal, somnambulism is characterized by an inability to maintain stable and consolidated SWS.

#### HYPERSYNCHRONOUS DELTA WAVES

One of the more controversial findings regarding the sleep EEG of patients with sleepwalking/sleep terrors is the presence of hypersynchronous delta activity (HSD), usually described as continuous high-voltage ( $>150 \mu\text{V}$ ) delta waves occurring during SWS or immediately prior to an episode (Kales et al., 1966; Guilleminault and Silvestri, 1982; Kavey et al., 1990; Blatt et al., 1991; Broughton, 2000). Studies of HSD prior to sleepwalking or sleep terror episodes in adult parasomniacs have yielded mixed results (Kavey et al., 1990). Using a different approach to assess prearousal delta activity, one study found that most

behavioral and nonbehavioral arousals from SWS in adult patients were not preceded by a delta-wave buildup and that only 15.5% were preceded by delta-wave clusters (Schenck et al., 1998).

The occurrence of HSD was recently assessed by our group (Pilon et al., 2006) with an array of measures over different EEG derivations during the NREM sleep of somnambulistic patients and controls during normal sleep, following sleep deprivation, and prior to somnambulistic episodes. We found that: (a) HSD was present in 80% of controls during baseline recording and in 90% after sleep deprivation; (b) when compared to control subjects, HSD occurred more frequently during sleepwalkers' sleep EEG; (c) sleep deprivation increased HSD during stage 4 sleep in both groups; and (d) there was no evidence that somnambulistic episodes were immediately preceded by a buildup in HSD or by any HSD-related variables.

Taken together, these findings reinforce the results from previous studies (Schenck et al., 1998; Pressman, 2004) in demonstrating that, regardless of how it is measured, HSD has low specificity for the diagnosis of NREM parasomnias. It is suggested that HSD is related to the expression of the homeostatic process underlying sleep regulation. This hypothesis is consistent with the finding that HSD is more frequent when sleep pressure is at its peak according to the two-process model of sleep (Borbély and Achermann, 2000), and with studies reporting HSD and high-amplitude delta waves during SWS of adults with SDB, another sleep-disordered population characterized by considerable sleep fragmentation and sleep deprivation (Himanan et al., 2004; Pressman, 2004).

The potential neurophysiological mechanisms underlying HSD remain unknown. It can be argued, however, that HSD represents an increased activity of the neural structures involved in the regulation of delta activity during NREM sleep. Delta activity results from the progressive hyperpolarization of the thalamocortical and cortical neurons, and, as sleep advances, serves to protect the brain from incoming sensory stimuli in order to allow more deep sleep; spindles activity prevails at intermediate levels of hyperpolarization, whereas delta activity is seen at a high level of hyperpolarization (Steriade et al., 1993; McCormick and Bal, 1997; Merica and Fortune, 2004). Thus, HSD could reflect more hyperpolarized thalamocortical and cortical neurons compared with regular delta activity.

#### SLOW-WAVE ACTIVITY

EEG slow-wave activity (SWA: spectral power in the 0.75–4.5-Hz band) is a quantitative measure of SWS dynamics and is considered an indicator of sleep depth

or sleep intensity (Borbély and Acherman, 2000). Gaudreau et al. (2000) investigated the power and dynamics of SWA in adult sleepwalkers and controls, and showed that sleepwalkers had significantly less overall SWA power, with the greatest difference occurring during the first NREM cycle. A similar reduction in SWA was also reported in two other studies of patients with sleepwalkers/sleep terror (Espa et al., 2000; Guilleminault et al., 2001). These data indicate that sleepwalkers' frequent awakenings from SWS interfere with the normal buildup of their SWA and provide further evidence for an abnormality in these patients' capacity to sustain stable SWS.

Actual behavioral episodes, however, are immediately preceded by an increase in SWA (Espa et al., 2000) or low delta power (0.25–2.0 Hz) (Guilleminault et al., 2001), a process that may reflect cortical reaction to brain activation.

### EEG during behavioral episodes

Original EEG investigations of experimentally induced somnambulistic episodes found that they could be described in terms of continuous and diffuse nonreactive alpha rhythms or by patterns of low-voltage delta and beta activity (Gastaut and Broughton, 1965; Broughton, 1968). More recently, the EEG associated with minor behavioral events in adult sleepwalkers was described as a pattern of stage 1 sleep without evidence of complete awakening (Guilleminault et al., 2001). Schenck et al. (1998) found that three postarousal EEG patterns characterized the first 10 seconds of most SWS arousals in adults with sleepwalking/sleep terrors: (I) diffuse rhythmic and synchronous delta activity ( $\leq 4$  Hz), most prominent in bilateral anterior regions; (II) diffuse and irregular, moderate to high voltage delta and theta activity intermixed with, or superimposed by, alpha and beta activity; and (III) prominent alpha and beta activity, at times intermixed with moderate voltage theta activity. Irrespective of specific EEG patterns, delta activity was found to be present in 44% of the postarousal EEGs.

We followed up on this study by assessing differences in observed postarousal EEG patterns across behavioral arousals from sleepwalkers as a function of sleep stage (Zadra et al., 2004). The two more frequently observed forms of postarousal activity were patterns II and III. These patterns were also the only two that occurred during stage 2 episodes. Delta activity was present in almost 50% of all episodes from SWS, and in 20% of those from stage 2. The distribution of principal postarousal EEG patterns was also investigated as a function of episode complexity. The results showed that diffuse rhythmic and synchronous

delta activity (pattern I) was more likely to accompany simple somnambulistic episodes than complex ones, and that it occurred only during events emerging from SWS as opposed to stage 2 sleep. There was no evidence of complete awakening during any of the episodes. Examples of patterns I and II are presented in Figures 52.1 & 52.2.

Finally, one single-proton emission computed tomographic study during sleepwalking in a 16-year-old boy with a history of somnambulism suggests that episodes arise from the selective activation of thalamocingulate circuits and the persisting inhibition of other thalamocortical arousal systems (Bassetti et al., 2000). EEG during the episode showed diffused, high-voltage, rhythmic delta activity (i.e., pattern I). The authors supported the view of sleepwalking as reflecting a dissociated state consisting of motor arousal and persisting mind sleep.

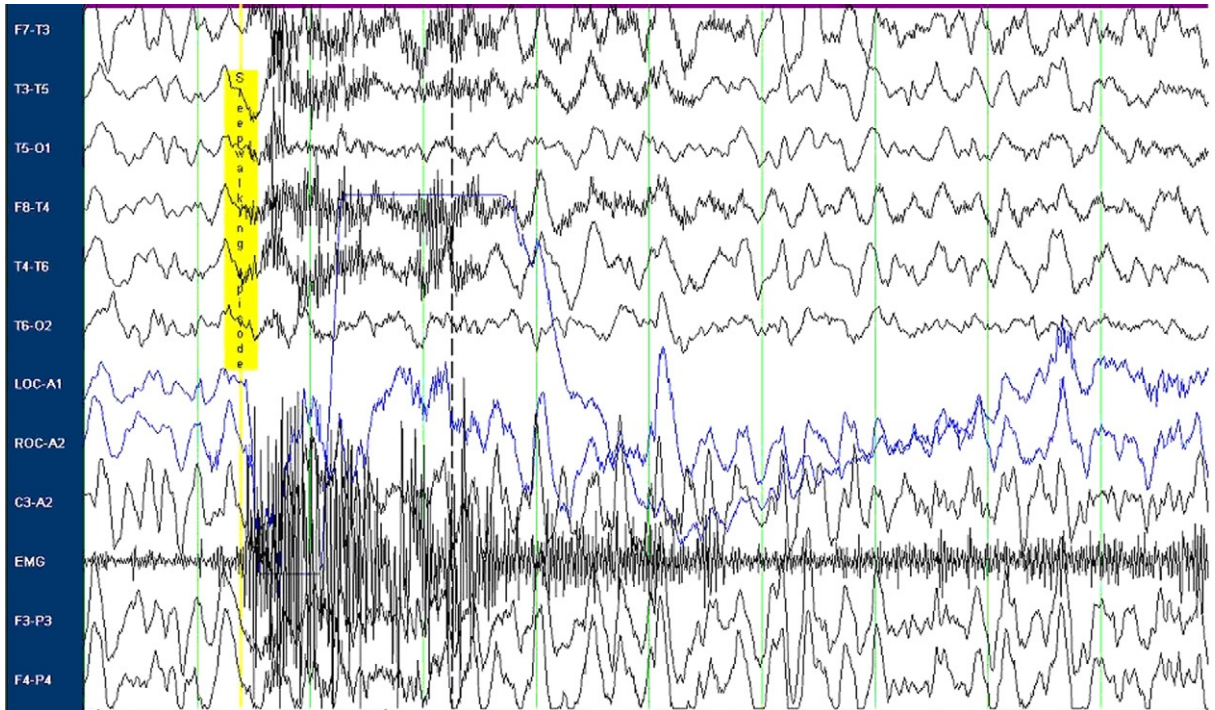
## SLEEP TERRORS

### Clinical features

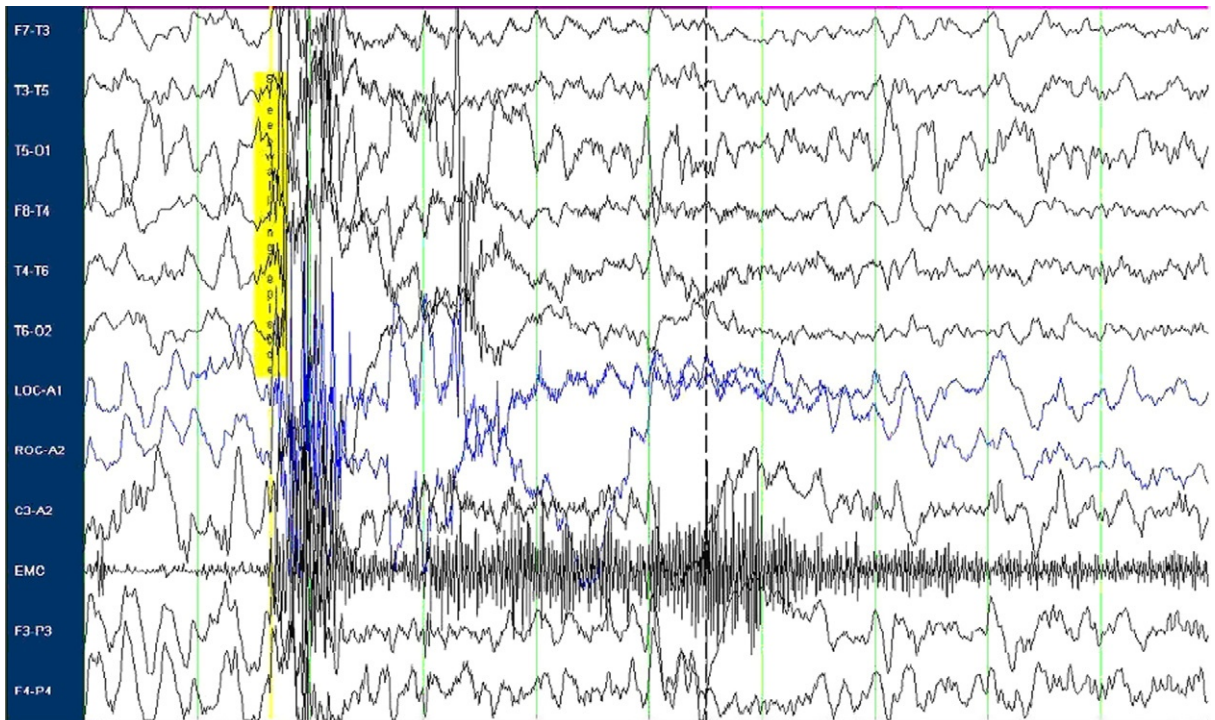
Sleep terrors, also known as night terrors, are sometimes called "*pavor nocturnus*" in children. As outlined by Broughton (2000), the term sleep terror is preferable to night terror, because episodes can occur during daytime sleep or naps. Historically, sleep terrors have been confused with nightmares, a distinct REM sleep parasomnia (see Chapter 53 for details of REM sleep parasomnias). Gastaut and Broughton (1965) first observed polysomnographically that sleep terrors were not associated with REM sleep but rather occurred suddenly during SWS.

Sleep terrors are characterized by a loud piercing scream or cry for help, intense autonomic activation (e.g., tachycardia, tachypnea, flushing of the skin, diaphoresis, mydriasis) inconsolability, and overwhelming anxiety or acute panic. Facial expressions often reflect intense fear. These reactions may be followed by agitated motor activity such as hitting the wall or running about as if reacting to imminent danger (Kales et al., 1980c). In fact, sleep terrors can be accompanied or followed by a sleepwalking episode (Fisher et al., 1973a; Nino-Murcia and Dement, 1987; Broughton, 2000). Somnambulism associated with sleep terrors may be more vigorous and frantic than a typical sleepwalking episode. At the end of a sleep terror, the person may awaken or simply return to sleep without being completely awakened.

Although most sleep terrors are benign, the behavior may be violent and result in considerable injury (Hartmann, 1983; Rauch and Stern, 1986; Schenck et al., 1989), occasionally with medicoforensic implications relating to these acts (Mahowald et al., 1990,



**Fig. 52.1.** Example of postarousal EEG pattern I during a behavioral episode from stage 4 sleep in a 19-year-old man. The EEG shows diffuse and rhythmic delta activity and is most predominant in the anterior regions.



**Fig. 52.2.** Example of postarousal EEG pattern II during a behavioral episode from stage 4 sleep in a 23-year-old woman. The EEG shows irregular delta and theta activity intermixed with faster activity.

2005; Mahowald and Schenck, 2000b; Cartwright, 2004). Sometimes, family members of affected patients can suffer from psychological trauma related to the violent behaviors manifested during sleep terrors, even if they are not physically injured, as suggested in a recent case study (Baran et al., 2003).

The incidence of sleep terrors in the sleep laboratory is lower than in the patient's normal environment (Fisher et al., 1973a; Nino-Murcia and Dement, 1987; Broughton, 2000). However, sleep terrors can be induced precipitously in predisposed individuals by auditory stimulation during SWS (Fisher et al., 1973a; Schenck et al., 1989). This observation has led some researchers to suggest that episodes are not the culmination of ongoing sleep mentation. Although this may be true in many cases, precipitating dream imagery, ranging from a brief frightening image or thought to more elaborate dream-like mentation, has been noted, particularly in adults (Fisher et al., 1974; Schenck et al., 1989; Kahn et al., 1991). Although some of this mental content can be related to "postarousal" events (e.g., fear of dying associated with autonomic activation), there exist numerous examples of imagery occurring during "prearousal" events (Fisher et al., 1974).

### Prevalence

Sleep terrors usually begin in childhood or adolescence but may also emerge in adulthood and tend to persist longer in life than does sleepwalking (Kales et al., 1980c). Many children with sleep terror will report sleepwalking at a later age (Klackenberg, 1987). The overall prevalence of sleep terror in children range from 1% to 18% with a peak prevalence between the ages of 5 to 7 years (Simonds and Parraga, 1982; Klackenberg, 1982, 1987; Salzarulo and Chevalier, 1983; Vela-Bueno and et al., 1985; DiMario and Emery, 1987; Laberge et al., 2000; Schredl, 2001). Sleep terrors have been reported to persist in 7–36% of children affected during adolescence (DiMario and Emery, 1987; Laberge et al., 2000). The prevalence in the general adult population is about 2.2% (Ohayon et al., 1999). This prevalence declines gradually with age, and becomes about 1% at age 65 years and above (Ohayon et al., 1999). There are no significant gender differences in children (Laberge et al., 2000) and adults (Ohayon et al., 1999).

### Pathophysiology

Many parasomniacs present a history of both somnambulism and sleep terrors, and, as previously described, these two NREM parasomnias share many common features. Not surprisingly, factors considered as being operant in the pathophysiology of sleep terrors are similar to

those previously described for sleepwalking. Consequently, they are presented only summarily below.

### PSYCHOPATHOLOGY

As with somnambulism, sleep terrors in adulthood have been described in relation to various psychopathologies, but many studies have shown that such parasomnias can occur in otherwise mentally healthy individuals (Schenck et al., 1989; Espa et al., 2000; Schenck and Mahowald, 2000). That said, when compared to people with confusional arousals or sleepwalking, individuals reporting sleep terrors are the most likely to meet criteria for a DSM-IV disorder, particularly anxiety disorders (Ohayon et al., 1999).

### GENETICS

Although the exact mode of transmission remains uncertain, familial and twin studies suggest a significant genetic contribution to sleep terrors (Hallstrom, 1972; Debray and Huon, 1973; Kales et al., 1980a). Sleep terrors are also more frequent in families with a history of somnambulism, and vice versa (Kales et al., 1980a; Abe et al., 1984). These data and the clinical similarities between these two parasomnias suggest a common genetic predisposition and similar pathophysiological mechanisms. It has been suggested that sleepwalking is a more prevalent and less severe manifestation of the same substrate that underlies sleep terrors (Kales et al., 1980a).

### ORIENTING RESPONSE

The orienting response to auditory stimuli in patients with sleep terrors has been reported to be more intense and persistent than in normal subjects, suggesting a hyperexcitability of the nervous system in these patients (Rogozea and Florea-Ciocoiu, 1983, 1985). Furthermore, there appears to be an association between the severity of sleep terrors and the intensity of the responsiveness change (Rogozea and Florea-Ciocoiu, 1985).

### Unusual sleep parameters

As with sleepwalkers, patients with sleep terrors show normal sleep architecture and cycling among sleep stages (Schenck et al., 1989; Zucconi et al., 1995; Espa et al., 2000). Nevertheless, a number of unusual sleep-related processes have been identified in their sleep microstructure.

### SLOW-WAVE SLEEP AROUSALS

Compared with controls, patients with sleep terrors or a mixture of disorders of arousal (e.g., sleep terror and sleepwalking) show an increased number of arousals during SWS both in children (Benoit et al., 1978)

and in adults (Broughton, 1991; Espa et al., 2000), more SWS to wake transitions (Broughton, 1991), of brief microarousals preceded by EEG slow-wave synchronization (Halasz et al., 1985), and in the number of CAP cycles (Zucconi et al., 1995). The resulting sleep fragmentation is viewed as interfering with normal buildup of SWA, and one study showed that, compared with controls, SWA in patients with disorders of arousal was significantly decreased and showed a slower rate of decay across NREM cycles (Espa et al., 2000). As discussed previously, similar findings have been reported in adult sleepwalkers (Gaudreau et al., 2000; Guilleminault et al., 2001). As suggested by Broughton (1991), these findings indicate the coexistence in patients with disorders of arousal of pressure for deep sleep and of a process resulting in repeated arousals during SWS.

#### **HYPERSYNCHRONOUS DELTA WAVES**

The presence of HSD has also been found to occur more frequently in the sleep EEG of patients with sleep terror than in controls (Halasz et al., 1985; Espa et al., 2000). HSD activity, however, has a low specificity for the diagnosis of NREM parasomnias (Schenck et al., 1998; Pressman, 2004; Pilon et al., 2006). See the section on sleepwalking for a more detailed account of HSD.

#### **EEG ACTIVITY PRIOR TO SLEEP TERRORS**

Of the many findings reported in Fisher's early studies (Fisher et al., 1973a, 1974), one of the most salient is that the severity of the sleep terror, as assessed by heart rate increase and maximum heart rate after arousal, is proportional to the duration of the preceding stage 3–4 sleep episode. This is true both for spontaneously occurring sleep terrors and for sleep terrors induced experimentally by sounding a loud buzzer. Consistent with these results, an EEG mapping study of a patient with chronic sleep terrors found that the EEG preceding sleep terrors contained significantly more delta power in central and frontal regions than control EEG sections, and that prearousal delta power was proportional to the sleep terror's intensity (Zadra and Nielsen, 1998). Similarly, Espa et al. (2000) found that the time course of SWA on the central derivation prior to behavioral episodes in patients with arousal disorder (sleepwalking, sleep terrors, or both) was preceded by an increase in SWA, with the main increase occurring immediately prior to the episode.

#### **EEG during sleep terrors**

The EEG activity during sleep terror is neither fully asleep nor fully awake (Fisher et al., 1973a). The three main postarousal EEG patterns identified by Schenck

et al. (1998) and described in the previous section on sleepwalking also apply to sleep terrors.

### **CLINICAL VARIANTS**

The behaviors manifested during an arousal disorder can be relatively distinct and specialized. Two variants of NREM parasomnias involve sleep-related eating and sleep-related sexual behaviors.

#### **Sleep-related eating disorder**

According to the International Classification of Sleep Disorders (American Academy of Sleep Medicine, 2005), sleep-related eating disorder (SRED) consists of recurrent episodes of involuntary eating and drinking during arousal from sleep with problematic consequences. SRED is classified under the "other parasomnia" section. Episodes typically occur during partial arousals from sleep during the first third of the night, with impaired subsequent recall (Whyte and Kavey, 1990; Schenck et al., 1991, 1993; Schenck and Mahowald, 1994; Winkelman, 1998). Polysomnographic studies have associated SRED with a variety of underlying sleep disorders, the most frequent of which is sleepwalking. SRED may also be associated with restless leg syndrome, periodic limb movements of sleep, obstructive sleep apnea, and circadian rhythm disorders (Schenck et al., 1993; Winkelman, 1998). Most of these sleep disorders can precipitate NREM parasomnia episodes in predisposed individuals (Guilleminault et al., 2003, 2005a) and are known to increase arousals during sleep.

Although SRED can affect both sexes and all ages, it is most common in young adult women (Schenck and Mahowald, 1994). SRED affects up to 4% of college students (Winkelman et al., 1999) and can lead to considerable weight gain (Schenck et al., 1993). A history of other parasomnias, especially sleepwalking, is also common (Winkelman, 1998). Although SRED is usually not associated with the presence of waking eating disorder, some patients present a history of current or past daytime eating disorder such as anorexia nervosa or bulimia (Schenck et al., 1991; Winkelman, 1998), and SRED is reported more frequently in patients with daytime eating disorders than in nonpsychiatric populations (Winkelman et al., 1999).

SRED has been reported in association with medications such as zolpidem (Harazin and Berigan, 1999; Morgenthaler and Silber, 2002; Schenck et al., 2005) and triazolam (Menkes, 1992). Some medications have been reported to be effective for the treatment of SRED such as topiramate (Winkelman, 2003), a combination of dopaminergic and opiate agents (Schenck and Mahowald, 2002a), or pramipexole (Provini et al., 2005).

### Sleep-related abnormal sexual behaviors

Sleep-related abnormal sexual behaviors (SRASBs) consist of inappropriate sexual activity occurring without conscious awareness during sleep (Mahowald et al., 2005). Other terms proposed for these episodes include “atypical sexual behavior during sleep,” “sex-somnia,” and “sleep sex” (Wong, 1986; Buchanan, 1991; Fenwick, 1996; Rosenfeld and Elhajjar, 1998; Alves et al., 1999; Guilleminault et al., 2002; Shapiro et al., 2003). SRASB can range from sexual vocalizations or sexualized bodily movements to violent masturbation or sexual assaults, and have been reported as being markedly different from behaviors normally initiated during the patient’s waking state (Guilleminault et al., 2002; Shapiro et al., 2003). According to the International Classification of Sleep Disorders, 2nd edition (ICSD-2) (American Academy of Sleep Medicine, 2005), SRASB is classified in the Disorders of Arousal section as a clinical subtype of confusional arousal. One polysomnographic study found that disorder of arousals were the most frequent sleep disorders associated with SRASB, and that the condition could also occur in association with REM-sleep behavior disorder (RBD) or NREM complex partial seizures (Guilleminault et al., 2002).

SRASB may give rise to a variety of negative emotions and cognitions including feelings of embarrassment, guilt, shame, or depression, often carries interpersonal consequences (Guilleminault et al., 2002; Mangan, 2004), and has potential medicolegal implications (Guilleminault et al., 2002; Shapiro et al., 2003).

The use of clonazepam, sometimes in association with psychotherapy or stress management, has been reported to be effective for the treatment of SRASB (Guilleminault et al., 2002).

### NREM PARASOMNIAS ASSOCIATED WITH PRIMARY SLEEP DISORDERS

Several lines of evidence indicate that in both children and adult populations sleep terrors and sleepwalking can be secondary to sleep respiratory events, such as obstructive sleep apnea (OSA) and upper airway resistance syndrome, or to other sleep disorders. Parent-reported parasomnias in children with OSA suggest that sleep terrors and sleepwalking are more frequent in sleep-disordered children than in normative controls (Owens et al., 1997). Similar results were reported by Ipsiroglu et al. (2002). A population-based cohort study of preadolescent school-aged children showed that sleepwalking was significantly more prevalent in children with SDB (Goodwin et al., 2004). Guilleminault et al (2003) found that 49 of 84 (58%) prepubertal

children with chronic sleep terrors and sleepwalking also presented with SDB. Two other children presented with restless leg syndrome. Treatment of the precipitating sleep disorder may result in a disappearance of the disorder of arousal (Guilleminault et al., 2003).

Similarly, a recent prospective study of 50 adults with chronic somnambulism found that many patients presented with SDB and that treatment of the SDB with continuous positive airway pressure or surgery controlled the sleepwalking (Guilleminault et al., 2005a). An association between adult sleepwalking and SDB has been noted by others (Ohayon et al., 1999; Espa et al., 2002).

### NREM PARASOMNIAS ASSOCIATED WITH MEDICAL CONDITIONS

Rarely, NREM parasomnias may develop as a result of medical or neurological conditions. *De novo* sleep terrors have been reported in association with a right thalamic lesion (Di Gennaro et al., 2004) and a brainstem lesion (Mendez, 1992). *De novo* somnambulism has been described in patients presenting with thyrotoxicosis caused by diffuse toxic goiter or Graves’ disease (Ajlouni et al., 2001, 2005). Disorders of arousal can also be triggered by medication. These include sedatives/hypnotics (Mendelson, 1994; Harazin and Berigan, 1999), neuroleptics (Charney et al., 1979), lithium (Landry et al., 1999), minor tranquilizers, stimulants, and antihistamines (Huapaya, 1979; Mahowald and Schenck, 2000a).

### DIAGNOSTIC CONSIDERATIONS

The DSM-IV and ICSD-II clinical criteria for sleepwalking and sleep terrors are presented in Table 52.2. Diagnosis of NREM parasomnias can often be made based on a detailed history, including complete description of the time course and content of sleep-related behaviors. Given that variable retrograde amnesia characterizes disorders of arousal, descriptive information from family members or a bed partner can be particularly valuable. Similarly, home video recording may also be helpful in characterizing behavioral manifestations.

### Use of polysomnography

When case presentations involve violent or injurious behaviors, excessive daytime sleepiness, or associated medical or neurological conditions, more extensive evaluations, including polysomnographic study with an expanded EEG montage, may be required. An expanded electroencephalogram electrode array can help differentiate NREM parasomnias from sleep-related seizures

Table 52.2

## Clinical criteria for sleepwalking and sleep terror disorder

DSM-IV diagnostic criteria	ICSD-II diagnostic criteria
<p><i>Sleepwalking disorder (307.46)</i></p> <ul style="list-style-type: none"> <li>A. Repeated episodes of rising from bed during sleep and walking about, usually occurring during the first third of the major sleep episode</li> <li>B. While sleepwalking, the person 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</li> <li>C. On awakening (either from the sleepwalking episode or the next morning), the person has amnesia for the episode</li> <li>D. Within several minutes after awakening from the sleepwalking episode, there is no impairment of mental activity or behavior (although there may initially be a short period of confusion or disorientation)</li> <li>E. The sleepwalking causes clinically significant distress or impairment in social, occupational, or other important areas of functioning</li> <li>F. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition</li> </ul>	<p><i>Sleepwalking disorder</i></p> <ul style="list-style-type: none"> <li>A. Ambulation occurs during sleep</li> <li>B. Persistence of sleep, an altered state of consciousness, or impaired judgment during ambulation demonstrated by at least one of the following:               <ul style="list-style-type: none"> <li>i. Difficulty in arousing the person</li> <li>ii. Mental confusion when awakened from an episode</li> <li>iii. Amnesia (complete or partial) for the episode</li> <li>iv. Routine behaviors that occur at inappropriate times</li> <li>v. Inappropriate or nonsensical behaviors</li> <li>vi. Dangerous or potentially dangerous behaviors</li> </ul> </li> <li>C. The disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder</li> </ul>
<p><i>Sleep Terror Disorder (307.46)</i></p> <ul style="list-style-type: none"> <li>A. Recurrent episodes of abrupt awakening from sleep, usually occurring during the first third of the major sleep episode and beginning with a panicky scream</li> <li>B. Intense fear and signs of autonomic arousal, such as tachycardia, rapid breathing, and sweating, during each episode</li> <li>C. Relative unresponsiveness to efforts of others to comfort the person during the episode</li> <li>D. No detailed dream is recalled and there is amnesia for the episode</li> <li>E. The episodes cause clinically significant distress or impairment in social, occupational, or other important areas of functioning</li> <li>F. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition</li> </ul>	<p><i>Sleep Terror Disorder</i></p> <ul style="list-style-type: none"> <li>A. A sudden episode of terror occurs during sleep, usually initiated by a cry or loud scream that is accompanied by autonomic nervous system and behavioral manifestations of intense fear</li> <li>B. At least one of the following associated features is present:               <ul style="list-style-type: none"> <li>i. Difficulty in arousing the person</li> <li>ii. Mental confusion when awakened from an episode</li> <li>iii. Amnesia (complete or partial) for the episode</li> <li>iv. Dangerous or potentially dangerous behaviors</li> </ul> </li> <li>C. The disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder</li> </ul>

as well as other sleep disorders such as RBD. Polysomnography is also required to identify primary sleep-related disorders (e.g., SDB, periodic limb movement disorder) that may underlie the parasomnia. In all cases, continuous audiovisual monitoring is essential to document behavioral manifestations and to correlate videotaped events with polysomnographic characteristics.

Diagnosing NREM parasomnias with objective instruments such as polysomnography can be difficult as episodes rarely occur in the sleep laboratory. Indirect evidence supporting a diagnosis can take the form of an increased frequency of arousals and microarousals from SWS, but these events are not specific to the arousal disorders. Two techniques that may increase the probability of recording more complex behavioral manifestations are sleep deprivation and the presentation of auditory stimuli during SWS. When compared to baseline recordings, one study found that 40 hours of sleep deprivation resulted in a fivefold increase in the number of somnambulistic episodes recorded in the sleep laboratory (Joncas et al., 2002). In addition, a significant increase in the complexity of the episodes recorded was observed during the recovery night. A subsequent study revealed that 25 hours of sleep deprivation was also effective in increasing both the frequency and the complexity of somnambulistic events recorded in the sleep laboratory (Pilon et al., 2005a). All but two of the 27 patients investigated in these studies had at least one behavioral episode during recovery sleep. The fact that none of the control subjects investigated in these studies experienced nocturnal behavioral manifestations in the laboratory demonstrates that sleep deprivation alone does not lead to somnambulistic episodes, but rather that it increases the probability of somnambulistic behaviors among those so predisposed. These results thus provide further evidence that sleep

deprivation can be a valuable tool that facilitates the polysomnographically based diagnosis of this sleep disorder, and suggest high sensibility for adult sleepwalkers.

Early studies of a small sample of young sleepwalkers found that behavioral events could be induced by standing the child on his or her feet during SWS (Gastaut and Broughton, 1965; Kales et al., 1966; Broughton, 1968; Broughton and Gastaut, 1974). In one study, two episodes were triggered during a child's SWS by calling his name (Kales et al., 1966). Similarly, sleep terrors can be precipitated in predisposed individuals by sounding a loud buzzer (Fisher et al., 1970, 1973a).

It has been suggested that the probability of recording somnambulistic or sleep terror events can be further increased by simultaneously combining factors that deepen sleep (e.g., intense physical activity, sleep deprivation, neuroleptics) with those that fragment sleep (e.g., stress, environmental or endogenous stimuli (Broughton, 1991; Espa et al., 2000; Besset and Espa, 2001). One pilot investigation (Pilon et al., 2005b) assessed this hypothesis in eight adult sleepwalkers and five controls under controlled conditions by combining 25 hours of sleep deprivation with the presentation of auditory stimuli. As shown in Table 52.3, the auditory stimulations precipitated somnambulistic episodes in approximately 15% of the trials administered during sleepwalkers' SWS at baseline and in 40% of the trials presented during their postsleep deprivation SWS. No somnambulistic episodes were induced in controls either during their SWS at baseline (0 in 24 trials) or after sleep deprivation (0 in 29 trials). These results support the hypothesis that the combination of sleep deprivation and external stimulation increases the probability of recording behavioral episodes in the sleep laboratory.

**Table 52.3**

**Characteristics of induced somnambulistic events and of auditory stimulations administered during slow-wave sleep before and after 25 hours of sleep deprivation in eight adult sleepwalkers**

	Baseline sleep	Recovery sleep	<i>P</i>
Total number of induced episodes during SWS	4	15	–
Number of patients experiencing at least one induced episode during SWS	3/8 (38%)	8/8 (100%)	<0.05
Mean (SD) frequency of induced episodes during SWS	0.5 (0.8)	1.9 (0.8)	<0.05
Mean (SD) number of auditory stimulations during SWS	3.5 (2.3)	4.6 (3.2)	ns
Success in inducing an episode with an auditory stimulation during SWS	4/28 (14%)	15/37 (41%)	<0.05
Mean (SD) intensity (in dB) of the auditory stimulations that induced behavioral episodes	56.7 (11.5)	54.0 (12.4)	ns

### Differential diagnosis

Disorders of arousal need to be distinguished from nightmare disorder, RBD, complex partial seizures, and nocturnal panic attacks.

Nightmares are vivid and disturbing mental experiences that generally occur during REM sleep and often result in awakening (American Academy of Sleep Medicine, 2005). They can be distinguished from sleep terrors by their usual occurrence during the second half of the night, when REM is most prominent, and by the recall of detailed dream content. The degree of automatic activation (e.g., palpitations and dyspnea) is much greater during sleep terrors and there is an absence of mental confusion upon awakening from a nightmare as opposed to a sleep terror. Actual screaming or other intense vocalizations that can characterize sleep terrors are rare during nightmares.

RBD is characterized by intermittent loss of REM-sleep atonia and by the appearance of elaborate motor activity associated with dream mentation during REM sleep (Schenck et al., 1986; Schenck and Mahowald, 2002b). Patients usually have a vivid recall of the dreams, which appear to correlate with observed behaviors (Schenck and Mahowald, 2002b). RBD can be also be distinguished from NREM parasomnias by its usual occurrence during the second half of the night and by the absence of mental confusion upon awakening. However, some patients may have behavioral manifestations during both REM and NREM sleep with RBD occurring in combination with a disorder of arousal (Bokey, 1993; Kushida et al., 1995; Schenck et al., 1997). This a condition known as parasomnia overlap disorder (Schenck et al., 1997).

Disorders of arousal and complex partial epileptic seizures share several clinical similarities (e.g., sudden onset, unresponsiveness, retrograde amnesia) and precipitating factors (e.g., sleep deprivation). Nocturnal frontal lobe epilepsy can be particularly difficult to differentiate from NREM parasomnias, especially in children (Zucconi and Ferini-Strambi, 2000). Complex partial seizures usually involve repetitive stereotypical behaviors and patients rarely return to bed. Epileptic seizures may occur in any sleep stages throughout the sleep period. Similar seizure activity may occur during daytime wakefulness. Sleep terrors and seizures may coexist in the same person (Tassinari et al., 1972).

Approximately 50% of all patients with panic disorder have nocturnal panic attacks which are characterized by intense fear or discomfort accompanied by cognitive and physical symptoms of arousal (American Psychiatric Association, 1994). These attacks are comparable to panic attacks experienced in the daytime and they may sometimes be clinically similar to sleep

terrors (Craske and Tsao, 2005). Nocturnal panic attacks usually occur in late stage 2 or early stage 3 sleep (Craske and Tsao, 2005) and, unlike many sleep terrors, patients do not become physically agitated or aggressive during the panic attack. Immediately after a nocturnal panic attack, patients are oriented, can vividly recall their attack, and usually have difficulty returning to sleep (i.e., suffer from insomnia); these features differ from those observed in patients with sleep terrors.

### TREATMENT

Several factors must be taken into account while considering treatment options for disorders of arousal. These include the frequency and chronicity of the episodes, the potential danger to the patient or to others, and the disruptive nature of the disorder for the patient, bed partner, or family. When the episodes are benign and not associated with harm potential, treatment is often unnecessary (Mahowald and Schenck, 1996). Reassuring the patient and significant others about the generally benign nature of the episodes and demystifying the events is sometimes sufficient. Efforts should be made to identify and avoid potential precipitating factors, such as sleep deprivation, stress, and environmental disturbances. Precautions should be taken to ensure a safe sleep environment for patients with sleep terrors or agitated somnambulism. Preventive measures can include the removal of obstructions in the bedroom, securing windows, sleeping on the ground floor, installing locks or alarms on outside doors, covering windows with heavy curtains, using a nightlight, placing barriers in stairways, and removing all sharp or otherwise dangerous objects.

As previously discussed, people reporting sleep terrors or somnambulism may suffer from SDB, including OSA and upper airway resistance syndrome. In these cases, treatment of the primary sleep disorder with nasal continuous positive airway pressure or surgical treatment for the SDB should result in the alleviation and control of the parasomnia.

### Pharmacological treatments

Generally, pharmacological agents should be considered only if the behaviors are hazardous or extremely disruptive to the bed partner or other household members (Nino-Murcia and Dement, 1987). Benzodiazepines (e.g., low doses of clonazepam or diazepam) and tricyclic antidepressants (e.g., imipramine) can be effective (Fisher et al., 1973b; Reid, 1975; Cameron and Thyer, 1985; Cooper, 1987; Schenck and Mahowald, 1996; Remulla and Guilleminault, 2004). Results of a randomized study in children with sleep terrors indicated

satisfactory treatment with L-5-hydroxytryptophan (Bruni et al., 2004). Anecdotal reports suggest that melatonin may also be effective in children (Jan et al., 2004). However, pharmacotherapy does not always result in adequate control of NREM parasomnias such as sleepwalking (Guilleminault et al., 2005a).

### Nonpharmacological treatments

A variety of nonpharmacological treatments has been recommended for long-term management of NREM parasomnias. Hypnosis (including self-hypnosis) has received the most attention, and this relatively brief form of intervention has been found to be effective in both children and adults with sleep terrors or sleepwalking (Clement, 1970; Eliseo, 1975; Taboada, 1975; Reid and Gutnik, 1980; Reid et al., 1981; Gutnik and Reid, 1982; Koe, 1989; Hurwitz et al., 1991; Kohen et al., 1992). Other treatment options include psychotherapy (Kales et al., 1982) and progressive relaxation (Kellerman, 1979). Positive results have also been reported with scheduled or anticipatory awakenings in children with sleep terrors or sleepwalking (Lask, 1988, 1993; Tobin, 1993; Frank et al., 1997; Durand and Mindell, 1999; Durand, 2002). This technique involves briefly awakening the patient approximately 15–30 minutes prior to the expected episode. The procedure is repeated nightly for up to a month. Reports suggest that improvements can be maintained for several months after the end of the treatment and that the intervention may result in a significant reduction in the frequency of these parasomnias. Irrespective of which approach is adopted, treatment should include instructions on sleep hygiene and stress management. The aforementioned safety recommendations should also be provided.

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