

The Oxford Handbook of Sleep Disorders

Section II. Sleep Disorders

Epidemiology, Classification, and Assessment

Parasomnias II: Sleep terrors and somnambulism

(Chapter 27)

Antonio Zadra, PhD & Mathieu Pilon PhD

Department of Psychology, Université de Montréal

and

Centre d'étude du sommeil et des rythmes biologiques,

Hôpital du Sacré-Cœur de Montréal

Address for correspondence:

Antonio Zadra, PhD
Department of Psychology
Université de Montréal
C.P. 6128, succ. Centre-ville
Montreal, Quebec, CANADA H3C 3J7

E-mail: antonio.zadra@umontreal.ca

Tel.: (514) 343-6626

Fax: (514) 343-2285

Abstract

Sleepwalking (somnambulism) and sleep terrors are known as disorders of arousal and constitute two of the most frequent and impressive NREM sleep parasomnias. This chapter discusses the clinical presentation, etiology, polysomnographic findings, diagnostic considerations, prevalence, and treatment options associated with these two parasomnias. Sleepwalking and sleep terrors share many characteristics. Most episodes arise from slow-wave sleep and are characterized by relative unresponsiveness to external stimuli, mental confusion, automatic behaviours, and variable retrograde amnesia. Episodes can be precipitated by factors that intensify or fragment sleep. Factors suggested as being operant include unusual sleep parameters, elevated psychopathology, deregulation of serotonergic systems, and a strong genetic component. A variety of treatments have been recommended but well-designed controlled clinical trials are lacking.

Keywords:

Sleepwalking; somnambulism, sleep terrors, parasomnia, genetics; treatment

List of Abbreviations:

- CAP: cyclic alternating pattern
- CNS: central nervous system
- DSM: Diagnostic and Statistical Manual
- DZ: dizygotic
- EEG: electroencephalography
- HSD: hypersynchronous delta activity
- ICSD: International Classification of Sleep Disorders
- MZ: monozygotic
- NREM: non-rapid-eye-movement
- PLMS: periodic sleep movements in sleep
- PSG: polysomnography
- RBD: REM-sleep behavior disorder
- REM: rapid eye movement
- SRED: sleep related eating disorder
- SRASB: sleep related abnormal sexual behaviors
- SWS: slow wave sleep

Parasomnias are undesirable physical, behavioral or experiential phenomena that occur during entry into sleep, within sleep, or during partial arousals from sleep (American Academy of Sleep Medicine, 2005). Depending on their exact manifestations, frequency and intensity, parasomnias can be considered normal sleep phenomena, especially when occurring during childhood, and may not significantly impact sleep quality or quantity, or daytime functioning. While some parasomnias (e.g., recurrent isolated sleep paralysis, sleep-related groaning) may cause relatively little enduring distress, others (e.g., nightmares, REM sleep behavior disorder, sleepwalking, sleep terrors) can have significant consequences, including marked psychological distress, self injuries and sleep disruption in the patient and a mixture of concern and apprehension in family members.

This chapter focuses on two prototypic NREM sleep parasomnias, namely sleepwalking (somnambulism) and sleep terrors. Together with confusional arousals, sleepwalking and sleep terrors are collectively termed “disorders of arousal” (Broughton, 1968) because of the autonomic and motor arousal which drives the patient towards a state of partial wakefulness from sleep. As will be described in this chapter, disorders of arousal can give rise to a wide range of adverse consequences. A summary and comparison of the main features of NREM and REM sleep parasomnias are presented in Table 1.

Insert Table 1 about here

Although disorders of arousal vary along dimensions of emotional, autonomic and motor activation, they nevertheless share a number of characteristics. NREM parasomnias are common in children and tend to decrease in frequency with age. Episodes generally arise out of slow wave sleep (SWS; stages 3 and 4 of NREM sleep) but also from stage 2 sleep. As a consequence, NREM parasomnias tend to take place in the first third of the night when slow-wave sleep is predominant. Symptoms and manifestations of the disorders of arousal can be considered along a spectrum, but most episodes are typically characterized by misperception and relative unresponsiveness to external stimuli, mental confusion, automatic behaviors, perceived threat and variable retrograde amnesia. Conditions that intensify sleep such as intense physical activity, hyperthyroidism, fever, sleep deprivation and neuroleptics or medications with depressive CNS effects can facilitate or precipitate NREM parasomnias in predisposed individuals. Factors that fragment sleep, including sleep-disordered breathing, periodic leg movement syndrome, stress and environmental or endogenous stimuli can have similar effects. Finally, a common genetic component is suspected as people with an arousal disorder often have a positive family history for one of the three disorders.

Sleepwalking

Clinical features

That people sometimes engage in complex ambulatory behaviors during sleep has been known for centuries. For example, Shakespeare's *Macbeth* contains a widely known episode of somnambulism during which a guilt-ridden Lady Macbeth tries to wash imaginary bloodstains from her hands while speaking of the crimes she and her husband

have committed. Interestingly, this early 17th century depiction of somnambulism is consistent with the medical view that existed until the mid 1960s which held that sleepwalking represented a dissociative state related to dreaming, and possibly related to the enactment of traumatic experiences.

The symptoms and manifestations that characterize sleepwalking can vary greatly both within and across patients. A sleepwalker's emotional expression can range from neutral or calm to extremely agitated. Similarly, actual behavioral manifestations can range from clumsy or simple and isolated actions such as sitting up in bed, pointing at a wall, fingering bed sheets, and walking about a room to surprisingly complex and organized behaviors such as rearranging furniture, cooking or eating, getting dressed, and playing a musical instrument. The sleepwalker's eyes are usually open throughout an episode but they may appear to have glassy stare. Sleepwalkers may spontaneously return to bed or eventually lie down to sleep elsewhere. Given the widely heterogeneous nature of sleepwalking episodes, their duration can vary from a few seconds to dozens of minutes. Reported behaviors in more extreme cases, including driving motor vehicles, inappropriate sexual activity, suspected suicide, and even homicide and attempted homicide, have raised fundamental questions as to the medico-forensic implications of these acts (Andersen, Poyares, Alves, Skomro & Tufik, 2007; Broughton et al., 1994; Mahowald, Schenck, & Cramer Bornemann, 2005; Schenck & Mahowald, 1995a). See Chapter 17 for information on forensic aspects of sleep disorders.

Although sleepwalking is often characterized in terms of its automatic behaviors and retrograde amnesia, ongoing work into the phenomenology of somnambulism indicates that perceptual, cognitive and affective dimensions can play an important role in

the subjective experience of adult sleepwalking. One study (Zadra, 2008) of 68 adult patients consecutively referred to a sleep disorders clinic for chronic sleepwalking found that perceptual elements from the sleeper's actual environment during somnambulistic episodes were sometimes (25%), often (37%) or always (16%) recalled. Forty-seven patients (69%) reported that various forms of mental content or sleep mentation (e.g., images, thoughts, emotions) often or always accompanied their episodes. Furthermore, the displayed behaviors were construed by most patients as being motivated by an intrinsic sense of urgency or underlying reason that accounts for their actions during actual episodes.

Similarly, laboratory investigations of adult sleepwalkers suggest that when available, patients' phenomenological contents are broadly consistent with the observed behaviors during an episode. For instance, as part of a study on the use of forced arousals from auditory stimuli to experimentally induce somnambulistic events (Pilon, Montplaisir & Zadra, 2008), one patient experienced an episode during which he quickly removed his pillow and frantically examined the back of his bed. The patient, who had recently become a dad, later reported that that he believed that his newborn had fallen behind the bed. In the same study, three patients who experienced induced episodes from the auditory stimuli reported hearing a voice asking them to carry out a precise action that precipitated the behavior. For example, one patient suddenly looked up at the ceiling with a fearful expression, started pointing about with one hand, and then proceeded to remove her electrodes with agitation. She later reported hearing someone tell her to tear away the electrodes or she was going to suffocate because the electrodes were attached to the ceiling.

Although these observations should not be taken as indicating that patients' mental content directly precipitates somnambulism in and of itself, such reports raise important questions as to the role that phenomenological contents play in how somnambulistic episodes are experienced and unfold. In addition, these findings suggest that somnambulism should not be assumed to necessarily reflect automatic behaviors in the pure sense of the term and that morning recall of sleepwalking episodes may be greater than generally believed. Clinicians and researchers may gain valuable information by investigating these dimensions of sleepwalkers' experiences.

Whereas the occurrence of sleepwalking in children is frequently viewed as a relatively benign condition, somnambulism in adults can result in serious injury to the sleeper or others. Adults suffering from somnambulism often consult due to a history of aggressive and/or injurious behaviour during sleep (Moldofsky, Gilbert, Lue, & MacLean, 1995; Rauch & Stern, 1986; Schenck et al., 1989). 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 (Kavey, Whyte, Resor, & Gidro-Frank, 1990; Milliet & Ummenhofer, 1999; Rauch & Stern, 1986). 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, Schenck, & Mahowald, 2000).

In one polysomnographic (PSG) investigation (Schenck et al., 1989) of 100 consecutive patients consulting for repeated nocturnal injury, 90% received one of two diagnosis: sleepwalking/night terrors (54%) or REM sleep behaviour disorder (36%). Reported behaviours 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 (Moldofsky et al., 1995) 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 another 19% reported harmful but non-destructive behaviour. Similarly, one study of 50 chronic sleepwalkers found that 30% had injured themselves or others in the course of at least one episode (Guilleminault et al., 2005).

Etiology

As previously mentioned, sleepwalkers were once thought to be in a dissociative state related to dreaming. Following Broughton's landmark contribution (Broughton, 1968) and the early work of Kales and his collaborators (Kales, Jacobson, Paulson, Kales, & Walter, 1966), sleepwalking was conceptualized as a disorder of arousal. However, why patients experience difficulties transitioning from sleep to full arousal is not fully understood and the exact pathophysiological mechanisms of somnambulism remain unclear. Several general factors have been proposed, including atypical sleep parameters, psychopathology, genetics, and deregulation of serotonergic systems.

Unusual sleep/EEG parameters

No consistently robust differences exist between adult somnambulistic patients and control subjects in terms of their overall sleep architecture and normal cycling among sleep stages. Nevertheless, a number of atypical sleep-related processes have been

described as characterizing the sleep of patients suffering from somnambulism, including NREM sleep instability, hypersynchronous delta waves (HSD), irregular build-up of slow-wave activity (SWA), and unique EEG characteristics prior to and during somnambulistic episodes. Each of these sleep-related variables are discussed in turn.

When compared to controls, adults and children with sleepwalking/sleep terrors show increases in the cyclic alternating pattern rate (Zucconi, Oldani, Ferini-Strambi, & Smirne, 1995), a measure of NREM instability which expresses the organized complexity of arousal-related phasic events. An increased cyclic alternating pattern rate was also reported in children with chronic sleepwalking and concomitant sleep respiratory disorders (Guilleminault, Lee et al., 2005). Several studies have also shown that sleepwalkers experience an unusually high number of arousals and brief microarousals out of slow wave sleep (Blatt, Peled, Gadoth, & Lavie, 1991; Espa, Ondze, Deglise, Billiard, & Besset, 2000; Pilon et al., 2008). Sleepwalkers' fragmentation of NREM sleep may reflect a milder manifestation of their presumed arousal dysfunction. NREM sleep instability and arousal oscillation may thus represent a typical microstructural feature of parasomniacs' NREM sleep and play a role in triggering sleepwalkers' abnormal motor episodes. These findings also suggest that in addition to being a disorder of arousal, somnambulism is characterized by an inability to maintain stable and consolidated SWS.

Finally, when compared to baseline recordings, sleep deprivation (with recovery sleep being initiated in the morning) significantly increases the number of SWS awakenings in sleepwalkers but not in controls (Pilon et al., 2008). A significant increase in patients' arousals is not observed when their recovery sleep is initiated during the night (Joncas, Zadra, Paquet, & Montplaisir, 2002). These results indicate that sleepwalkers are

particularly vulnerable to increased homeostatic sleep pressure following sleep deprivation when sleep is initiated at a circadian time of increasing wake propensity.

Many articles and textbooks present as fact that HSD, usually described as continuous high voltage (> 150 uV) delta waves, characterizes the SWS EEG of patients with sleepwalking/sleep terrors, especially prior to episode onset. However, careful studies of HSD and other forms of delta-activity prior to sleepwalking episodes have yielded mixed to poor results (Kavey et al., 1990; Pilon, Zadra, Joncas, & Montplaisir, 2006; Pressman, 2004; Schenck, Pareja, Patterson, & Mahowald, 1998). For example, one study (Schenck et al., 1998) found that most behavioural and nonbehavioral arousals from SWS in adult patients were not preceded by a delta wave build-up and that only 15.5% were preceded by delta wave clusters.

The occurrence of HSD was investigated (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 are immediately preceded by a build-up in HSD or by other HSD-related variables.

Taken together, these findings reinforce the results from previous studies (Pressman, 2004; Schenck et al., 1998) in demonstrating that regardless of how it is

measured, HSD occurs in the slow-wave sleep of normal controls and has low specificity for the diagnosis of NREM parasomnias.

HSD, however, may be related to the expression of the homeostatic process underlying sleep regulation and particularly of delta activity during NREM sleep. 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 & Acherman, 2000) and with studies reporting HSD and high amplitude delta waves during SWS of adults with sleep-disordered breathing, another sleep disordered population characterized by considerable sleep fragmentation and sleep deprivation (Himanen, Joutsen, & Virkkala, 2004; Pressman, 2004).

EEG slow-wave activity (SWA: spectral power in the 0.75 to 4.5 Hz band) is a quantitative measure of SWS dynamics and is considered an indicator of sleep depth or sleep intensity. 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 1st NREM cycle. A similar reduction in SWA was also reported in two other studies of sleepwalkers/sleep terror patients (Espa et al., 2000; Guilleminault, Poyares, Aftab, & Palombini, 2001). These data indicate that normal SWA build-up is impeded by sleepwalkers' frequent awakenings from SWS and provide further evidence for an abnormality in these patients' capacity to sustain stable SWS.

Spectral analysis of sleep EEG signals prior to behavioral episodes in patients with sleepwalking and/or sleep terrors indicate that the onset of a parasomnia is likely to be preceded by an increase in SWA (Espa et al., 2000) or in low delta power (0.25-

2.0Hz) (Guilleminault et al., 2001), with the later peaking about 10 to 12 seconds prior to an episode. Similarly, one recent investigation (Jaar, Pilon, Montplaisir, & Zadra, 2009) reported increases in the density of delta and slow delta waves during approximately the last 30 seconds prior to episode onset. These processes may reflect cortical reactions to brain activation.

Turning to postarousal EEG activity, early studies reported that the EEG recorded during somnambulistic episodes was characterized by continuous and diffuse nonreactive alpha rhythms or by patterns of low-voltage delta and beta activity (Broughton, 1968; Gastaut & Broughton, 1965). 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 and coworkers (1998) described three postarousal EEG patterns that characterized the first 10 seconds of most SWS arousals in adults with sleepwalking/sleep terrors: I) diffuse rhythmic and synchronous delta activity, most prominent in bilateral anterior regions and with a typical frequency of 2.2 Hz, a typical amplitude of 85 μ V, and a typical duration of 20 sec, II) diffuse and irregular moderate-to-high voltage delta and theta activity intermixed with alpha and beta activity, and III) prominent alpha and beta activity. Irrespective of specific EEG patterns, delta activity was present in 44% of the post-arousal EEGs.

More recently, these patterns were assessed during behavioral arousals in adult sleepwalkers (Zadra, Pilon, Joncas, Rompre, & Montplaisir, 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 slow-wave sleep and 20% of those from stage 2 sleep.

Pattern I (diffuse rhythmic and synchronous delta activity), which only occurred during events emerging from SWS, was more likely to accompany simple somnambulistic episodes than complex ones.

Examples EEG activity associated with the spontaneous onset of somnambulistic episodes in the laboratory are presented in Figures 1 and 2.

Insert Figures 1 and 2 about here

Psychopathology

The presence of somnambulism (with or without concomitant sleep terrors) in adulthood has often been viewed as reflecting important psychopathology or personality disorders (e.g., Kales et al., 1980; Pai, 1946; Soldatos, Vela-Bueno, Bixler, Schweitzer, & Kales, 1980). In early childhood, the occurrence of somnambulism can be associated with separation anxiety (Petit, Touchette, Tremblay, Boivin, & Montplaisir, 2007) and reports indicate that anxiety may increase episode occurrence in both children and adults (Cirignotta, Zucconi, Mondini, Lenzi, & Lugaresi, 1983; Crisp, Matthews, Oakey, & Crutchfield, 1990; Rosen, Mahowald, & Ferber, 1995). When compared to controls, higher levels of anxiety disorders have been reported in subgroups of adolescents with sleep terrors and/or sleepwalking (Gau & Soong, 1999). One self-report epidemiologic study found a significantly higher prevalence of concurrent DSM-IV anxiety disorders in adult subjects with sleepwalking (12.7%) and sleep terrors (34.2%) when compared to those with no parasomnia (4.7%) (Ohayon, Guilleminault, & Priest, 1999). Similar percentages across study groups were found for concurrent mood disorders. Grouped as

a whole, mental disorders were found in approximately 50% of people with sleep terrors as compared to about 20% of sleepwalkers. These epidemiological findings also revealed that almost 50% of adults with arousal disorders report having experienced a stressful event in the past 12 months (e.g., divorce or separation from a spouse, bereavement) and that they are significantly more likely than controls to describe their lives as being highly stressful (Ohayon, Guilleminault, & Priest, 1999). Finally, it must be stressed that many adult patients with sleepwalking and/or sleep terrors do not present with a DSM-based Axis I psychiatric disorder nor with highly disturbed personality traits and show good psychosocial functioning (Guilleminault, Kirisoglu et al., 2005; Mahowald & Schenck, 1999; Schenck, Boyd, & Mahowald, 1997; Schenck et al., 1989). Furthermore, successful treatment of an Axis I disorder usually does not impact sleepwalking or sleep terror frequency (Schenck et al., 1989).

Genetic predisposition

A strong genetic component to somnambulism has been well documented. About 80% of somnambulistic patients 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. One twin study (Barkwin, 1970) of over 300 pairs found that monozygotic (MZ) twins are concordant for the disorder six times as often as dizygotic (DZ) twins. A population-based twin study (Hublin, Kaprio, Partinen, Heikkila, & Koskenvuo, 1997) of 1045 MZ and 1899 DZ pairs showed a considerable genetic effect in adulthood sleepwalking (probandwise concordance 5 times higher in MZ than DZ pairs) although the effect in childhood sleepwalking was not as pronounced (1.5 times higher in MZ than DZ pairs). 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

Serotonin has been hypothesized to be involved in the pathophysiology of sleepwalking on the basis that certain factors implicating the serotonergic system (e.g., fever, certain drugs such as paroxetine and amitriptyline) can precipitate sleepwalking (Juszczak & Swiergiel, 2005). In addition, sleepwalking episodes are four to nine times more common conditions associated with abnormalities in the metabolism of serotonin such as Tourette syndrome or migraine headaches (Barabas, Matthews, & Ferrari, 1984; Giroud, Nivelon, & Dumas, 1987).

Other laboratory findings

Hormonal factors may influence the frequency with which women experience sleep terrors and injurious sleepwalking as these can emerge premenstrually (Schenck & Mahowald, 1995b) while sleepwalking can decrease during pregnancy, particularly in primiparas (Hedman, Pohjasvaara, Tolonen, Salmivaara, & Myllyla, 2002).

Rarely, sleepwalking may develop as a result of medical or neurological conditions. One recent study (Poryazova et al., 2007) reported 6 cases of adult onset (de novo) sleepwalking in patients with Parkinson disease. De novo somnambulism has also been described in patients presenting with thyrotoxicosis caused by diffuse toxic goiter or Graves' disease (Ajrlouni et al., 2005). Disorders of arousal can also be triggered by medication, including sedatives/hypnotics, neuroleptics, lithium, minor tranquilizers, stimulants, and antihistamines.

A transcranial magnetic stimulation study (Oliviero et al., 2007) examined motor cortex excitability during wakefulness in 8 sleepwalkers and 18 controls. Sleepwalkers showed significant hypoexcitability of some inhibitory circuits as revealed by reduced short interval intracortical inhibition, cortical silent period duration, and short latency afferent inhibition.

Finally, a SPECT investigation of a sleepwalking episode recorded from a 16-year old man with a history of somnambulism revealed an increase of 25% in rCBF in the posterior cingulate and anterior cerebellum compared to SWS without episodes (Bassetti, Vella, Donati, Wielepp, & Weder, 2000). The authors suggest that variations in the motor and emotional manifestations of sleepwalking may be related to different activation patterns of the cingulate cortex as it modulates behavior in response to emotional processes. A decrease in rCBF in frontoparietal associative cortices was also noted in comparison to the wakefulness pattern of normal subjects. Since this pattern occurs in normal SWS, this finding suggests that the brain is indeed sleeping during somnambulism. These pilot findings thus support the notion that sleepwalking is a dissociated state consisting of motor arousal and persisting mind sleep.

Assessment and Differential Diagnosis

The diagnostic criteria for sleepwalking and sleep terrors in the American Psychiatric Association's DSM-IV (APA, 1994) are presented in Table 2. Key assessment issues to cover in the clinical history for NREM parasomnias are presented in Table 3.

Insert Tables 2 and 3 about here

Diagnosis of sleepwalking can often be made based on a detailed clinical history, including complete description of the time course and content of sleep-related behaviors. Given that variable retrograde amnesia characterizes many somnambulistic episodes, descriptive information from family members or a bed partner can be particularly valuable. Similarly, home video recording can also be helpful in characterizing behavioral manifestations. It should be noted, however, that sleepwalking and sleep terrors can co-occur (Hublin, Kaprio, Partinen, & Koskenvuo, 2001; Kavey et al., 1990; Schenck et al., 1989) and that the two conditions are sometimes difficult to differentiate (Schenck et al., 1998).

When case presentations involve violent or injurious behaviors, excessive daytime sleepiness, or associated medical or neurological conditions, more extensive evaluations, including overnight PSG with an expanded EEG montage, may be required. Continuous audiovisual monitoring is indispensable to document behavioral manifestations and to correlate monitored events with polysomnographic parameters. Sleep laboratory investigations can be useful in ruling out other disorders (e.g., nocturnal seizures, episodic nocturnal wandering, RBD) or in identifying primary sleep-related disorders (e.g., sleep disordered breathing, periodic limb movement disorder) that might underlie the parasomnia, but no validated sleep protocol exists to directly confirm the diagnosis for sleepwalking.

The principal difficulty in diagnosing sleepwalking directly with PSG is that behavioral events rarely occur in the laboratory (Blatt et al., 1991; Joncas et al., 2002; Kales et al., 1966). In addition, when partial episodes do develop, they are usually less

complex than what is described for the patient's home environment and the presence of bed rails typically raised on either side of the patient's bed can also dissuade or impede patients from actually leaving their beds. Some polysomnographic features (e.g., frequent arousals from SWS, presence of HSD) have been proposed as indirect evidence supporting the diagnosis, but these variables show poor sensitivity and specificity (Pilon et al., 2006; Pressman, 2007b).

Sleepwalking can be secondary to sleep respiratory events, including obstructive sleep apnea and upper airway resistance syndrome, or other sleep disorders such as periodic limb movements in sleep (PLMS). An association between sleepwalking/sleep terrors and sleep disordered breathing has been described in several studies (Espa, et al., 2002; Guilleminault, Palombini, Pelayo, & Chervin, 2003; Ohayon et al., 1999), including one population-based cohort study of pre-adolescent school-aged children (Goodwin et al., 2004). Treatment of the precipitating sleep disorder (e.g., continuous positive airway pressure for sleep disordered breathing) can result in a disappearance of the disorder of arousal (Guilleminault, Kirisoglu et al., 2005; Guilleminault et al., 2003). However, some studies find that that a majority of adult sleepwalkers referred to a sleep disorders clinic by their treating physician for suspected somnambulism do not suffer from comorbid sleep disorders (Zadra et al., 2008).

Somnambulism needs to be distinguished from REM sleep behavior disorder (RBD). 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. Patients usually have a vivid recall of the dreams that appear to correlate with observed behaviors. 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. This condition is known as parasomnia overlap disorder (Schenck et al., 1997) and a recent case report (Nadège et al., 2009) indicates that this particular parasomnia can be secondary to a right lesion of the brainstem's pontine tegmentum.

Finally, it should be noted that disorders of arousal and complex partial epileptic seizures share several clinical similarities, including sudden onset, unresponsiveness and retrograde amnesia. In addition, these conditions are all sensitive to factors that deepen sleep, such as sleep deprivation. Nocturnal frontal lobe epilepsy can be particularly difficult to differentiate from NREM parasomnias, especially in children (Zucconi & Ferini-Strambi, 2000). Complex partial seizures usually involve repetitive stereotypic behaviours 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 deprivation and experimental induction of episodes

As previously noted, diagnosing somnambulism with objective instruments such as polysomnography can be difficult as even partial episodes rarely occur in the sleep laboratory. The probability of recording more complex behavioral manifestations can be increased via sleep deprivation and the presentation of auditory stimuli during SWS. Two studies found that 24 and 38 hours of sleep deprivation significantly increased the frequency of somnambulistic events in sleepwalkers (Joncas et al., 2002; Mayer, Neissner, Schwarzmayer, & Meier-Ewert, 1998) while one study did not find an increase

in the number of episodes after 36 hours of sleep deprivation (Guilleminault, Leger, Philip, & Ohayon, 1998). These inconsistencies may be due, in part, to the limited number of patients investigated (7 to 10). A recent investigation (Zadra et al., 2008) of 40 consecutive sleepwalkers (including 10 with either PLMS or mild sleep apnea syndrome) revealed that 25 hours of sleep deprivation was also effective in increasing both the frequency and complexity of somnambulistic events recorded in the sleep laboratory. Combining data from all 40 patients shows that recovery sleep resulted in one or more episodes being recorded from 36 (90%) of the sleepwalkers.

Thus, a relatively short period of sleep deprivation resulting in daytime recovery sleep can effectively facilitate the emergence of somnambulistic behaviors. 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 in predisposed individuals.

It had been suggested that the simultaneous combination of factors that deepen sleep (e.g., sleep deprivation) with those that fragment sleep (e.g., environmental or endogenous stimuli) increases the chances of sleepwalkers experiencing an episode. This hypothesis was recently tested in a study that investigated the effects of forced arousals from experimental auditory stimuli in 10 adult sleepwalkers and 10 control subjects during normal sleep and following post-sleep deprivation recovery sleep (Pilon et al., 2008). The mean frequency of all somnambulistic events recorded across sleepwalkers' PSG assessments is presented in Figure 3. Forced arousals during slow-wave sleep were successful in experimentally inducing somnambulistic episodes in the adult sleepwalkers

and, as predicted, sleep deprivation significantly increased the forced arousals' efficacy. In fact, while no somnambulistic episodes were induced in controls, the presentation of auditory stimuli during daytime recovery sleep resulted in all 10 patients experiencing one or more induced episodes.

These results support the hypothesis that via its homeostatic pressure for increased SWS, sleep deprivation facilitates the occurrence of sleepwalking in predisposed individuals and that this effect can be augmented by incorporating forced arousals.

Insert Figure 3 about here

Medico-legal considerations

As previously described, the clinical presentation of somnambulism can include instances of violent behaviors towards others. There has been an increase in the number of legal cases involving sleep-related violence and sleepwalking represents a significant cause of sleep-related injury. However, there is considerable debate regarding the validity of scientific evidence sometimes presented to support a sleepwalking defence (Cartwright, 2004; Ebrahim & Fenwick, 2008; Pressman, 2007a; Pressman et al., 2009; Pressman, Mahowald, Schenck, & Bornemann, 2007; Pressman, Schenck, Mahowald, & Bornemann, 2007). For instance, whereas alcohol is reported as increasing the possibility of sleepwalking (Ebrahim et al., 2008) a review of this literature indicates that there no actual scientific support for such a statement (Pressman et al., 2007). Although considerable progress has been made in identifying and grouping factors that predispose, prime or trigger sleepwalking (Pressman, 2007a, 2007b), many forensic aspects of sleep

medicine, and of parasomnias in particular, are still in their infancy. A detailed account of forensic aspects of sleep disorders is presented in Chapter 17.

Prevalence and developmental aspects

Sleepwalking is more common in childhood than in adulthood as most children will experience, at least temporarily, one or more of the NREM sleep parasomnias during childhood or early adolescence. One study (Petit et al., 2007) of a representative sample of preschoolers aged 2.5 to 6 years found an overall prevalence of sleepwalking of 14.5% with no significant age effects and new cases of somnambulism appearing equally at all ages. The peak incidence of sleepwalking (approximately 17%) is around 11 to 12 years of age (Klackenberg, 1982). Between the approximate ages of 3 to 9 years, persistent sleepwalking appears to be more common in young boys than in young girls (Petit et al., 2007; Shang, Gau, & Soong, 2006), but gender differences disappear in older children and adolescents (Klackenberg, 1982; Laberge, Tremblay, Vitaro, & Montplaisir, 2000). Most children with sleepwalking will outgrow the disorder during mid to late adolescence, but somnambulism can persist into adulthood in up to 25% of cases (Hublin et al., 1997). Two prospective longitudinal studies of children (Laberge et al., 2000; Petit et al., 2007) revealed high rates of co-occurrence for sleepwalking and sleep terrors. Specifically, approximately 41% of children with persistent somnambulism at age 6 also suffered from sleep terrors (Petit et al., 2007) while at 11 years of age, 17% of sleepwalkers also experienced sleep terrors (Laberge et al., 2000). In addition, children with sleep terrors can develop sleepwalking at a later age and the two parasomnias can occur within the same episode (e.g., sleep terror followed by sleepwalking). However, one epidemiological investigation of parasomnias in people between 15 and 100 years of

age found a co-occurrence of night terrors and sleepwalking in only 0.3% of the overall sample (Ohayon et al., 1999).

Sleepwalking can also develop during adulthood and subsequently increase in severity over time. The prevalence of sleepwalking in adults is approximately 2% to 4% with no significant gender differences (Goldin, 1997; Hublin et al., 1997; Ohayon et al., 1999). However, there is some evidence to suggest that given comparable histories, males are more likely than females to experience somnambulistic behaviors in the sleep laboratory (Zadra et al., 2008). Lifetime prevalence for sleepwalking may be considerably higher (8.5%) in adult psychiatric populations (Lam et al., 2008).

Treatment options

Treatment is often unnecessary when the episodes are benign and not associated with potential injury. In this case, reassuring the patient/family about the benign nature of the episodes and demystifying the events is often sufficient. However, attention should be paid to potential precipitating factors, such as sleep deprivation, stress and environmental disturbances, so that such factors can be avoided. When patients present with a history of agitated somnambulism or sleep terrors, precautions should be taken to ensure a safe sleep environment. Preventative 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 highlighted in a recent review article (Harris & Grunstein, 2009), controlled clinical trials for the treatment of somnambulism are lacking. Hypnosis (including self-hypnosis) has been found to be effective in both children and adults presenting with

chronic sleepwalking (Hauri, Silber, & Boeve, 2007; Hurwitz, Mahowald, Schenck, Schluter, & Bundlie, 1991; Reid, Ahmed, & Levie, 1981). In children, the preferred treatment for somnambulism consists of a behavioral technique called anticipatory or scheduled awakening. Parents keep a diary of their child's episodes and determine the approximate time at which the episodes typically occur. They will then awaken their child about 15-20 min before the episode's typical time of occurrence for a period of one month. Based on several case reports, it appears that this simple intervention can yield rapid results and that benefits are maintained for several months after the end of the treatment (Durand & Mindell, 1999; Frank, Spirito, Stark, & Owensstively, 1997; Tobin, 1993).

As previously discussed, people reporting sleep terrors or somnambulism may suffer from sleep disordered breathing, including obstructive sleep apnea and upper airway resistance syndrome. In these cases, treatment of the primary sleep disorder with nasal CPAP or surgical treatment for the sleep-disordered breathing should result in the alleviation and control of the parasomnia.

Pharmacological treatment should be considered only if the behaviors are hazardous or extremely disruptive to the bed partner or other household members. Benzodiazepines (clonazepam or diazepam) and tricyclic antidepressants (imipramine) can be effective (Remulla & Guilleminault, 2004). However, pharmacotherapy does not always result in adequate control of sleepwalking (Guilleminault et al., 2005). Treatment should always include instructions on sleep hygiene, avoidance of sleep deprivation, and stress management.

Clinical variants:**Sleep-related eating disorder and sleep-related sexual behaviors**

Sleep related eating disorder (SRED) and sleep related abnormal sexual behaviors (SRASB) are two variants of NREM parasomias involving relatively distinct and specialized behaviors. Given the increasing clinical and research attention these disorders have attracted, they are briefly reviewed below.

Sleep related eating disorder

According to the International Classification of Sleep Disorders (AASM, 2005), SRED, which is classified under the ‘other parasomnia’ section, consists of recurrent episodes of involuntary eating and drinking during arousal from sleep with problematic consequences. Episodes, which are typically both frequent and chronic, tend to occur during partial arousals from NREM sleep during the first third of the night and are characterized by varying degrees of awareness and subsequent recall (Howell, Schenck, & Crow, 2009; Schenck, Hurwitz, O'Connor, & Mahowald, 1993; Winkelman, 1998). PSG studies have associated SRED with a variety of underlying sleep disorders, the most frequent of which is sleepwalking (Schenck et al., 1993; Winkelman, 1998).

Although SRED can affect both sexes and all ages, it is most common in young adult women (Schenck & Mahowald, 1994), particularly in those with a history of current or past eating disorder. SRED affects up to 4% of college students (Winkelman, Herzog, & Fava, 1999) and can result in a variety of adverse consequences, including health problems and considerable weight gain (Schenck et al., 1993). SRED can also occur in association with medications such as zolpidem (Harazin & Berigan, 1999; Morgenthaler & Silber, 2002; Schenck et al., 2005) or triazolam (Menkes, 1992).

When comorbid sleep disorders are present, treatment should focus on their control. Although based on limited evidence, medications reported to as being effective for the treatment of SRED include topiramate (Winkelman, 2003) and dopaminergics (e.g., levodopa, pramipexole, bupropion) (Howell, Schenck, & Crow, 2009)

Sleep related abnormal sexual behaviors

SRASB consist of inappropriate sexual activities occurring without conscious awareness during sleep (Andersen et al., 2007; Mahowald et al., 2005; Schenck, Arnulf, & Mahowald, 2007). Other terms proposed for these episodes include sexsomnia and sleep sex. 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 (Andersen et al., 2007; Guilleminault, Moscovitch, Yuen, & Poyares, 2002; Shapiro, Trajanovic, & Fedoroff, 2003; Schenck et al., 2007).

According to the ICSD-II (AASM, 2005), SRASB is classified in the Disorders of Arousal section as a clinical subtype of confusional arousal. Disorders of arousal, and sleepwalking in particular, are the most frequent sleep disorders associated with SRASB although it could also occur in association with RBD or NREM complex partial seizures (Andersen et al, 2007; Guilleminault et al., 2002; Schenck et al., 2007). As detailed in a recent review, however (Schenck et al., 2007), a wide range of sleep related disorders may be associated with SRASB.

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 important clinical and

forensic implications (Andersen et al., 2007; Guilleminault et al., 2002; Schenck et al., 2007).

The use of clonazepam, sometimes in association with psychotherapy or stress management, has been reported to be effective for the treatment SRASB (Guilleminault et al., 2002; Schenck et al., 2007). When associated sleep disorders are present, their treatment should be prioritized.

Sleep terrors

Clinical features

Sleep terrors (also known as night terrors, *pavor nocturnus* in children, and incubus attacks in adults) are “arousals from slow-wave sleep accompanied by a cry or piercing scream and autonomic nervous system and behavioral manifestations of intense fear” (AASM, 2005). As outlined by Broughton (2000), the term sleep terror is preferable to night terror since episodes can occur during daytime sleep or naps. Historically, sleep terrors have been confused with nightmares, a distinct REM sleep parasomnia (see Chapter 26 for details on nightmares). Gastaut and Broughton (1965) first observed polysomnographically that sleep terrors were not associated with REM sleep but rather occurred suddenly during SWS. A comparison of the main features of NREM and REM sleep parasomnias can be found in Table 1.

Sleep terrors typically occur within 90 minutes after sleep onset, are characterized by a loud piercing scream or cry for help, intense autonomic activation (e.g., sweating, flushing of the skin, mydriasis, tachycardia, rapid breathing), inconsolability, and overwhelming fear or acute panic. On rarer occasions, this initial terror can be followed by complex behavioral manifestations such as leaving the bed, fleeing the room or

thrashing around. Behaviors can also be violent, result in self-injury or property damage (Rauch & Stern, 1986; Schenck et al., 1989). These more extreme episodes can result in considerable distress in family members of affected patients. The duration of sleep terrors is usually relatively short, often from under a minute to a few minutes. However, sleep terrors can be accompanied or followed by a sleepwalking episode thereby extending the episode's duration and rendering ambiguous the distinction between sleep terrors and somnambulism (Broughton, 2000; Fisher, Kahn, Edwards, & Davis, 1973). Attempting to console or awaken an individual during a sleep terror can prolong or intensify the episode. Once the sleep terror episode has subsided, the person usually does not fully awaken, returns to sleep, and remains partially or completely amnesic for the event the following day.

If questioned immediately following an episode, patients may report indistinct recollections of immediate threats from which they were trying to escape. Precipitating imagery, ranging from a brief frightening image or thought to more elaborate dreamlike mentation has been noted, particularly in adults (Fisher, Kahn, Edwards, Davis, & Fine, 1974; Kahn, Fisher, & Edwards, 1991; Pressman, 2007a; Schenck et al., 1989). Although some of this mental content may be related to "post-arousal" states (e.g., fear of dying associated with autonomic activation), there exist numerous examples of imagery occurring during "pre-arousal" events (Fisher et al., 1974).

Etiology

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 only presented summarily below.

Sleep terrors that occur in childhood are usually not associated with a neurological condition, whereas onset in adulthood could indicate a neurological disease including thalamic or brainstem lesions (Di Gennaro et al., 2004; Mendez, 1992). Although many studies have shown that NREM parasomnias can occur in otherwise mentally healthy individuals (Espa et al., 2000; Schenck & Mahowald, 2000; Schenck et al., 1989), sleep terrors in adulthood have been described in relation to psychopathology and personality disorders (e.g., Kales et al., 1980). Epidemiological findings indicate that individuals reporting sleep terrors are twice as likely as sleepwalkers and 5 times more likely than controls to meet diagnostic criteria for a DSM-IV mood disorder, and about 3.5 times more likely than sleepwalkers or controls to report a history of mental disorders, including mood and anxiety disorders (Ohayon et al., 1999).

As is true for somnambulism and confusional arousals, genetic factors play a major role. MZ twins are more concordant than DZ twins for sleep terrors (Abe, Oda, Ikenaga, & Yamada, 1993; Nguyen et al., 2008) and terrors are twice as frequent in children for whom one or both parents have a sleepwalking history than in children with non-affected parents (Abe, Amatori, & Oda, 1984). These data and the clinical similarities between these two parasomnias suggest a common genetic predisposition and similar pathophysiological mechanisms.

In individuals with sleep terrors, the orienting response to auditory stimuli has been reported to be more intense and persistent than in normal subjects, suggesting a hyperexcitability of the nervous system in these individuals (Rogozea & Florea-Ciociu,

1985). Furthermore, there appears to be an association between the severity of sleep terrors and the intensity of the responsiveness change.

EEG findings prior to and during episodes

When compared to controls, children and adults with sleep terrors or a mixture of disorders of arousal (e.g., sleep terror and sleepwalking) show an increased number of arousals during SWS (Espa et al., 2002; Espa et al., 2000), more SWS-to-wake transitions (Broughton, 1991), of brief microarousals preceded by EEG slow wave synchronization (Halasz, Ujjaszsi, & Gadoros, 1985), and in the number of CAP cycles and rates (Bruni et al., 2008; Zucconi et al., 1995). The resulting sleep fragmentation is viewed by several researchers as interfering with normal buildup of SWA and one study showed that when compared to controls, SWA in patients with disorders of arousal is significantly decreased and shows a slower rate of decay across NREM cycles (Espa et al., 2000). As previously discussed, 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 co-existence in patients with disorders of arousal of pressure for deep sleep and of a process resulting in repeated arousals during SWS. Finally, based on their analysis of CAP parameters in children with sleep terrors, Bruni et al (2008) suggest that the abnormal alteration of EEG amplitude that characterizes parasomniacs' SWS might be a neurophysiological marker of disorders of arousal.

As with sleepwalkers, frequent occurrence of HSD has been reported in the sleep EEG of sleep terror patients (Espa et al., 2000; Halasz et al., 1985). As reviewed in detailed in the preceding section on sleepwalking, HSD activity has a low specificity for the diagnosis of NREM parasomnias.

Duration of stage 3-4 sleep preceding episode onset is positively correlated with episode intensity as assessed by heart rate increase and maximum heart rate after arousal (Fisher et al., 1973). Similarly, one investigation (Zadra & Nielsen, 1998) of a patient with chronic sleep terrors found that pre-arousal delta power in central and frontal regions was proportional to the sleep terror's intensity. A subsequent study (Espa et al., 2000) of patients with sleepwalking, sleep terrors or both arousal disorders found that behavioral episodes were preceded by an increase in SWA with the main increase occurring immediately prior to the episode's onset.

During the sleep terror itself, the EEG activity demonstrates that the subject is neither fully asleep nor fully awake (Fisher et al., 1973). 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.

Differential diagnosis

Table 2 presents the diagnostic criteria for sleep terrors in the American Psychiatric Association's DSM-IV (APA, 1994). Diagnosis of sleep terrors can often be made based on a detailed clinical history, including complete description of the time course and content of sleep-related behaviors. As with sleepwalking, descriptive information from family members, a bed partner and the use of home video recording can be particularly valuable in characterizing behavioral manifestations.

Insert Table 3 about here

Sleep terrors need to be distinguished from nightmare disorder, complex partial seizures, and nocturnal panic attacks. See corresponding section on sleepwalking for the differentiation between NREM parasomnias and RBD.

Nightmares are vivid and disturbing mental experiences that generally occur during REM sleep and often result in awakening (AASM, 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 which can characterize sleep terrors are rare during nightmares.

As previously mentioned for somnambulism, disorders of arousal and complex partial epileptic seizures share several clinical similarities and precipitating factors. Complex partial seizures may occur in any sleep stage throughout the sleep period and usually involve repetitive stereotypic behaviours. Sleep terrors and seizures may coexist in the same person.

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 (APA, 1994). These attacks are comparable to panic attacks experienced in the daytime and they may sometimes be clinically similar to sleep terrors. Nocturnal panic attacks usually occur in late stage 2 or early stage 3 sleep and unlike many sleep terrors, patients do not become physically agitated or aggressive during the panic attack (Craske & Tsao, 2005). 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.

Assessment

Given the similarities in pathophysiological processes believed to underlie disorders of arousal, most of the assessment considerations described in relation to sleepwalking also apply for sleep terrors. The incidence of sleep terrors in the sleep laboratory is lower than in the patient's normal environment (Broughton, 2000; Fisher et al., 1973). As with somnambulism, repeated brief microarousals and arousals from SWS that do not progress into actual episodes are common in sleep terror patients, especially in the second half of the first two SWS sleep episodes (Broughton, 1991; Espa et al., 2000). A normal polysomnogram, however, does not rule out a diagnosis of sleep terrors. Rarely, sleep terrors may arise from stage 2 sleep. It is also possible to induce sleep terrors in predisposed individuals by sounding a loud buzzer (Fisher, Byrne, Edwards, & Kahn, 1970; Fisher et al., 1973)

As has been shown with sleepwalking, the occurrence of sleep terrors in both children and adult populations can be secondary to sleep respiratory events, including obstructive sleep apnea and upper airway resistance syndrome or other sleep disorders. Treatment of the precipitating sleep disorder may result in a disappearance of sleep terror episodes.

De novo sleep terrors have been reported in association with right thalamic lesion (Di Gennaro et al., 2004) and brainstem lesion (Mendez, 1992).

Prevalence and developmental aspects

Reported incidence estimates for sleep terrors are wide-ranging, partly due to important variations in the age range studied, sampling methods, and definitions used. Further, parents and patients may not differentiate between nightmares and sleep terrors. The overall prevalence of sleep terror in children range from 1% to 18% with a peak prevalence between ages 5 to 7 (DiMario & Emery, 1987; Klackenberg, 1982; Laberge et al., 2000; Schredl, 2001). When an operational definition is supplied to parents, a high overall prevalence (40%) was found for preschoolers aged 2.5 to 6 years and sleep terrors were more frequent between the ages 2.5 to 4 than at ages 5 and 6 (Petit et al., 2007). Persistent sleep terrors were related to separation anxiety and to having one's parents recently divorce. Another parent-based investigation (Nguyen et al., 2008) of 390 pairs of MZ and DZ twins assessed at 10 months and 30 months revealed a prevalence of sleep terrors of 36.9% at 18 months and 19.7% at 30 months with no notable gender differences. Children with sleep terrors may also experience somnambulism at a later age (Klackenberg, 1987) and by age 11, approximately 35% of children with sleep terrors will also experience episodes of somnambulism (Laberge et al., 2000). As is the case with somnambulism and confusional arousals, sleep terrors tend to resolve during adolescence and do not display a gender difference (Laberge et al., 2000; Petit et al., 2007). Sleep terrors can also emerge in adulthood and may persist longer in life than does sleepwalking (Kales et al., 1980). The prevalence in the general adult population is about 2.2%, shows no significant gender differences, and declines gradually with age to attain about 1% at 65 years of age and older (Ohayon et al., 1999). A high degree of overlap exists in adult populations among the three principal disorders of arousal.

Treatment options

The scheduled awakening technique, previously described for the management of sleepwalking, has also been shown to be effective in the treatment of sleep terrors in children (Durand, 2002; Durand & Mindell, 1999; Frank et al., 1997; Lask, 1988, 1993). This technique involves briefly awakening the patient approximately 15-20 minutes prior to the expected time of occurrence. One report of three young children suffering from chronic sleep terrors found that the intervention reduced the frequency of sleep terrors in all three children and that these improvements were maintained after 12 months (Durand & Mindell, 1999). Results of a randomized study in 45 children with sleep terrors assigned to a treatment or a non-treatment group showed satisfactory treatment with L-5-hydroxytryptan administered at bedtime for 20 consecutive days (Bruni, Ferri, Miano, & Verrillo, 2004). Follow-up visits at 1 and 6 months post-treatment found that a significantly higher proportion of children in the treated group showed a positive response (93.5% responders at 1 month and 83.9% at 6 months) as compared to the non-treated group (28.6% responders at 1 and 6 months). In adults, when the episodes are not associated with injury potential, treatment is often unnecessary. If a treatment is needed, the same pharmacological and non-pharmacological approaches as for somnambulism can be tried.

Conclusions

Despite numerous clinical and empirical investigations, the exact mechanisms which give rise to NREM parasomnias remain unclear. Considerable progress has been made documenting the importance of associated factors and in identifying key variables

involved in their etiology. Although the precise mode of inheritance for disorders of arousal remains to be specified, a genetic contribution has been well documented and strong support also exists for the idea that sleepwalkers experience difficulties in maintaining stable and consolidated NREM sleep. Continued work in the fields of genetics and polysomnography (including spectral analyses) likely represent the most promising venues in order to elucidate our understanding of these sleep disorders. There is also a growing literature on developmental aspects associated with parasomnias, from young preschoolers to older adolescents, which also helps us understand how and when NREM parasomnias emerge and why these conditions persist and even worsen in some individual but not others. The limited data collected on neuroimaging and transcranial magnetic stimulation in relation to NREM parasomnias suggest that, although methodologically challenging, these investigative tools may pave the way towards a better understanding of disorders of arousal. Finally, preliminary assessments of phenomenological dimensions associated with somnambulism and sleep terrors indicate that perceptual, cognitive and affective dimensions can play an important role in the subjective experience of these two parasomnias.

Unlike most sleep disorders, the diagnosis of sleepwalking and sleep terrors is still primarily based on the patient's clinical history. The development of an investigative protocol that would allow researchers and clinicians to establish a polysomnographically-based diagnosis for disorders of arousal remains central. This issue is especially important given the growing number of medico-legal cases of sleep-related violence. A recent series of studies have shown that sleep deprivation significantly increases the frequency of somnambulistic events recorded in the laboratory. Moreover, the

probability of recording an episode in predisposed individuals can be further increased by combining sleep deprivation with experimental forced awakenings. Although the results obtained thus far appear promising, additional work on larger and more varied clinical samples is needed.

A variety of nonpharmacological as well as pharmacological treatments have been recommended for long-term management of somnambulism and sleep terrors. However, well-designed controlled clinical trials are needed to guide and support treatment decisions.

Future directions:

1. What role does NREM instability play in the pathophysiology of NREM parasomnias?
2. Why do common genetic and pathophysiological factors give rise to sleepwalking in some individuals and sleep terrors in others?
3. Can a validated investigative protocol be developed that will allow researchers and clinicians to establish a PSG-based diagnosis for sleepwalking and sleep terrors?
4. What are the neurobiological predictors of pharmacological and non-pharmacological treatment response in patients suffering from disorders of arousal?
5. To what extent can brain imaging and clinical trials help us understand possible neurotransmitter dysfunctions in NREM parasomnias?

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Table 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 the sleep period	First third to half of the sleep period	First third to half of the sleep period	Last half of the sleep period	Last half of the 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	Behaviour that correlates with dream content.
Duration	1-15min	1-30min	1-10min	3-20min	1-10min
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

Table 2. Clinical criteria for Sleepwalking and Sleep Terror Disorder**DSM-IV Diagnostic criteria for Sleepwalking Disorder (307.46)**

- A. Repeated episodes of rising from bed during sleep and walking about, usually occurring during the first third of the **major sleep episode**.
- 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.
- C. On awakening (either from the sleepwalking episode or the next morning), the person has **amnesia** for the episode.
- 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).
- E. The sleepwalking causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- 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.

DSM-IV Diagnostic criteria for Sleep Terror Disorder (307.46)

- 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.
- B. Intense fear and signs of autonomic arousal, such as tachycardia, rapid breathing, and sweating, during each episode.
- C. Relative unresponsiveness to efforts of others to comfort the person during the episode.
- D. No detailed dream is recalled and there is **amnesia** for the episode.
- E. The episodes cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- 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.

Table 3. Key assessment features of a clinical interview for NREM parasomnias

- Age of onset
- Timing of the episode during the sleep period
- Episode frequency and duration
- Detailed description of the episode including behavioral manifestations, affect, and mentation during and after the event
- Responsiveness to external stimuli during the event
- Level of consciousness or awareness when awakened from an episode
- Memory for the event
- Precipitating or triggering factors
- Sleep wake-pattern and sleep environment
- Symptoms of daytime sleepiness
- Symptoms of other sleep disorders
- Familial history for NREM parasomnias and other sleep disorders
- Medical, psychiatric and neurological history
- Medication and substance use

Figure Legends:**Figure 1.**

Example of post-arousal EEG pattern III during a somnambulistic episode recorded from slow wave sleep in a 22 year old man. The EEG shows relatively low voltage, fast frequencies, intermixed with theta activity. Alpha activity is predominant in the posterior regions.

Figure 2.

Example of a somnambulistic episode recorded during daytime recovery slow wave sleep following 25h of sleep deprivation in an 18-year-old man. At the beginning of the episode, the EEG shows diffuse delta activity. Later, there is a fluttering of the EEG showing theta activity intermixed with faster activity.

Figure 3.

Mean frequency (and SEM) of somnambulistic events recorded in sleepwalkers across sleep periods with and without auditory stimuli (AS). Differences are significant at a level of $p < 0.05$. (From Pilon et al., 2008). Results show that the number of somnambulistic episodes significantly increases during recovery sleep with AS when compared to baseline and to normal sleep with AS. Recovery sleep without AS also significantly increases the mean frequency of episodes when compared to baseline recordings.





