

REVIEW ARTICLE

Violence in sleep

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Although generally considered as mutually exclusive, violence and sleep can coexist. Violence related to the sleep period is probably more frequent than generally assumed and can be observed in various conditions including parasomnias (such as arousal disorders and rapid eye movement sleep behaviour disorder), epilepsy (in particular nocturnal frontal lobe epilepsy) and psychiatric diseases (including delirium and dissociative states). Important advances in the fields of genetics, neuroimaging and behavioural neurology have expanded the understanding of the mechanisms underlying violence and its particular relation to sleep. The present review outlines the different sleep disorders associated with violence and aims at providing information on diagnosis, therapy and forensic issues. It also discusses current pathophysiological models, establishing a link between sleep-related violence and violence observed in other settings.

Keywords: sleep; behavioural neurology; sleep in movement disorders; seizures

Abbreviations: RBD = rapid eye movement sleep behaviour disorder; REM = rapid eye movement

Introduction

Although generally considered as mutually exclusive, sleep and violence can coexist. One of the first reports dates back to medieval times and relates to a Silesian woodcutter, who after a few hours of sleep woke up abruptly, aimed his axe at an imaginary intruder and killed his wife instead (Gastaut and Broughton, 1965). Another early case was reported by Yellowless in 1878

and describes a young man with a history of sleep terrors who killed his 18-month-old son by smashing him against the wall during the night, taking him for a wild beast that was about to attack his family. In 1893, Charcot was asked to pronounce himself on a case of attempted murder that occurred during an apparent episode of somnambulism, in which a servant, shortly after falling asleep, injured his landlady and another employee with a gun (Brouardel *et al.*, 1893). Nowadays, dramatic reports of

somnambulistic homicide still gain considerable attention in the media (Broughton *et al.*, 1994; Cartwright, 2004).

The most widely known condition in this respect is probably sleepwalking, but numerous other disorders with a potential for sleep-related violence exist, including other parasomnias, epilepsy and psychiatric diseases. Important advances in the fields of genetics, neuroimaging and behavioural neurology have expanded the understanding of the mechanisms underlying violence and its particular relation to sleep. Along with this increasing knowledge, neurologists, psychiatrists and sleep specialists assume an increasing role in legal issues related to violent acts committed during sleep. They should be familiar with these conditions as most sleep disorders associated with violence are treatable, thus making the correct diagnosis constitutes the critical first step in the prevention of further violence and ensuring personal and public safety.

The present review will focus on violent behaviour emerging with different sleep disorders, and aims at providing information on diagnosis, therapy and forensic issues. It also discusses current pathophysiological models, establishing a link between sleep-related violence and violence observed in other settings.

Definitions

For the purpose of this review, 'violence' is defined as an aggressive act that inflicts unwarranted physical harm on others (Filley *et al.*, 2001). It is a subset of 'aggression', a broader term encompassing both mental and physical damage. 'Premeditated aggression' (also referred to as instrumental, predatory or proactive aggression) is purposeful and goal-directed. 'Impulsive aggression' (also termed affective, reactive or hostile aggression) constitutes a response to a frustrating or threatening event that induces anger and occurs without regard for any potential goal. It has a particular relevance with regard to sleep-related violence (Volavka, 1999; Blair, 2004; Siever, 2008).

Epidemiology

A study evaluating the frequency of sleep-related violence in the general population by means of telephone interviews reported a prevalence of 2% (Ohayon *et al.*, 1997). However, this percentage might be overestimated, as the study was based on a standardized questionnaire and did not involve assessment by a sleep specialist. Harmful behaviour has been reported in 59% of patients with sleep terrors and sleepwalking that were consecutively recruited at a sleep clinic (Moldofsky *et al.*, 1995) and in 70% of patients with nocturnal wanderings of different aetiologies (Guilleminault *et al.*, 1995), but again these proportions may be overestimated, as patients with sleep-related violent behaviour are more likely to consult sleep clinics.

Epidemiologic studies indicate that gender is the most consistent risk factor for violence in general (Stanton *et al.*, 1997) and this holds true for sleep-related violence. Violent behaviour and injury in arousal disorders is 1.6–2.8 times more common in males (Schenck *et al.*, 1989a; Moldofsky *et al.*, 1995; Guilleminault *et al.*, 1998). About 97% of injuries and 80% of potentially lethal behaviours in rapid eye movement sleep behaviour disorder (RBD) occur in males (Schenck *et al.*, 1989a, 2009). Ictal and

peri-ictal aggression is also over-represented in males (Rodin, 1973; Delgado-Escueta *et al.*, 1981; Marsh and Krauss, 2000; Tassinari *et al.*, 2005a).

Disorders underlying sleep-related violence

True sleep-related violence occurs in the course of parasomnias and nocturnal seizures. In nocturnal dissociative disorders (Schenck *et al.*, 1989b), factitious disorder, malingering (Griffith and Slovik, 1989; Mahowald *et al.*, 1992) and dementia, violence may be related exclusively to the nocturnal period, but occurs in a state of wakefulness (Table 1).

Parasomnias

Disorders of arousal

Disorders of arousal consist of incomplete awakenings from non-rapid eye movement (non-REM) sleep characterized by reduced vigilance, impaired cognition, retrograde amnesia for the event and variable motor activity, ranging from repetitive and purposeless movements to more complex behaviours such as eating, drinking, driving, sexual intercourse and aggression (American Academy of Sleep Medicine, 2005). Confusional arousals consist of confusional behaviour upon awaking from sleep, most often from slow-wave sleep in the first part of the night. If an individual leaves the bed and starts walking, the disorder is referred to as sleepwalking. In sleep terrors, the arousal is characterized by intense autonomic activation and typical behavioural features such as sitting up in bed and screaming, while the person remains unresponsive to external stimuli. Different arousal disorders may coexist in the same individual and an episode may start as one arousal disorder and evolve into another (i.e. sleep terror evolving into agitated sleepwalking). Arousal disorders preferentially occur in the first half of the night and tend not to recur during the same night or to arise from daytime naps. Dreamlike mentation associated with episodes of sleepwalking or sleep terrors are mostly unpleasant, involving aggression on the part of the sleeper (24%), misfortune (54%) and apprehension (84%) (Oudiette *et al.*, 2009).

Disorders of arousal are common during childhood but may persist (18–25%) or arise *de novo* during adulthood (0.6%) (Hublin *et al.*, 1997). Genetic predisposition in the case of sleepwalking is suggested by a 10-fold increased prevalence among first-degree relatives of sleepwalkers (Kales *et al.*, 1980). The genes that confer the risk of sleepwalking remain essentially unknown. The only established marker is the presence of the HLA DQB1 allele found in 35% of sleepwalkers, compared with only 13% of normal subjects (Lecendreau *et al.*, 2003).

It is generally assumed that episodes of arousal disorders are more likely to occur in genetically predisposed individuals in the presence of an increased pressure for slow-wave sleep and factors favouring arousals or fragmenting sleep. Increased slow-wave sleep pressure occurs after sleep deprivation and with a large variety of psychotropic medications, as reviewed elsewhere

Table 1 Characteristics of disorders with potential for sleep-related violence

Disorder	State of occurrence	Clinical features	Preferential occurrence of violence
Confusional arousals	Dissociation wake/non-REM sleep	Incomplete awakening, reduced vigilance, impaired cognition and amnesia for the event	When being forced to awaken from sleep
Sleepwalking	Dissociation wake/non-REM sleep	Like confusional arousals but with perambulation	On incidental encounter or when approached by another person
Sleep terror	Dissociation wake/non-REM sleep	Incomplete awakening from non-REM-sleep with manifestations of fear	Linked to a frightening dream image
RBD	Dissociation wake/REM sleep	Acting out of dreams	In relation to a dream that is being acted out
Nocturnal paroxysmal dystonia	Possible in all sleep stages, preferentially stage non-REM 2	Bipedal automatisms, twisting of trunk and pelvis, vocalizations, dystonic posturing of head/limbs	Accidental or in relation to hyperkinetic features of seizures
Epileptic nocturnal wandering	Possible in all sleep stages, preferentially stage non-REM 2	Like sleepwalking, more directed violence possible	Accidental or when approached or restrained by another person
Confusional states	Wake	Variable	Variable
Psychiatric dissociative states	Wake or wake/sleep transition	Variable, most frequent manifestation is wandering, generally amnesia for the event	Often automutilation, thrashing movements, assaults
Malingering	Wake	Variable; associated with primary or secondary gain	Variable

(Pressman, 2007b). Among these, sedative–hypnotic medications, particularly zolpidem, are associated with complex and aggressive sleep-related behaviours (Dolder and Nelson, 2008; Lam *et al.*, 2008; Hwang *et al.*, 2010). Based on the fact that a few early studies showed an increase in slow-wave sleep after alcohol ingestion (Prinz *et al.*, 1980; MacLean and Cairns, 1982; Dijk *et al.*, 1992; Feige *et al.*, 2006; Van Reen *et al.*, 2006), alcohol has been claimed to induce sleepwalking episodes by defendants in criminal cases. A recent review of the literature on the subject, however, revealed that low levels of alcohol in social drinkers resulted in an increase in slow-wave sleep in only 6 of 19 published studies. Studies in which alcohol was administered to a clinically diagnosed sleepwalker, or providing evidence for increases in slow-wave sleep in severely intoxicated individuals were not identified (Pressman *et al.*, 2007). Conditions known for increasing the likelihood of arousals from sleep include sleep disordered breathing, periodic leg movements in sleep, fever (Karacan *et al.*, 1968), stress and provocation by noise and touch (Pressman, 2007a). Among these, obstructive sleep apnoea is increasingly recognized as a trigger for violent disorders of arousal (Baron and Auckley, 2004; Lateef *et al.*, 2005; Schenck and Mahowald, 2008). Experimental sleep deprivation (Joncas *et al.*, 2002; Zadra *et al.*, 2008) and forced arousals (Pilon *et al.*, 2008) have been shown to increase the number and complexity of somnambulistic episodes in sleepwalkers.

Violence in arousal disorders

Disorders of arousal have a potential for sleep-related violence. Homicide and attempted homicide (Yellowless, 1878; Podolsky, 1959, 1961; Bonkalo, 1974; Chuaqui, 1975; Hartmann, 1983; Oswald and Evans, 1985; Tarsh, 1986; Howard and D'Orban, 1987; Scott, 1988; Ovuga, 1992; Broughton *et al.*, 1994; Nofzinger and Wettstein, 1995), filicide (Luchins *et al.*, 1978),

along with completed and attempted 'pseudo-suicide' (Chuaqui, 1975; Mahowald *et al.*, 2003) have been reported in the setting of arousal disorders in the medical literature (Table 2). Inappropriate sexual behaviours (Wong, 1986; Hurwitz *et al.*, 1989; Buchanan, 1991; Fenwick, 1996; Shapiro *et al.*, 1996; Rosenfeld and Elhajjar, 1998; Guillemainault *et al.*, 2002; Ebrahim, 2006; Bejot *et al.*, 2010) occurring during arousal disorders are frequently associated with violence. Among the 31 patients with parasomnia-related sleepsex reviewed by Schenck and coworkers, 45% displayed assaultive behaviour, 29% had sex with minors and 36% sustained legal consequences from their sexual sleep-related behaviour.

Pressman (2007a) reviewed 32 legal and medical case reports of violence associated with disorders of arousal and found that aggressive behaviour occurred in different ways across confusional arousals, sleepwalking and sleep terrors. In confusional arousals, violence was preferentially elicited when individuals were awakened from sleep by someone else. Violent behaviour during sleepwalking, in contrast, tended to occur when the sleepwalking episode was already underway and the individual was approached by another person or incidentally encountered someone else. Violence related to sleep terrors appears to be a reaction to a (dreamed or hallucinated) concrete, frightening image that the individual can subsequently describe. In the same series of cases, Pressman (2007a) examined the role of physical contact and proximity as triggering factors of sleep-related violence and found that 100% of confusional arousals, 81% of sleep terrors and 40–90% of sleepwalking cases were associated with provocations including noise, touch and/or close proximity. The victims were rarely sought by sleepwalkers.

Two further studies tried to identify risk factors for the occurrence of violence in disorders of arousal. Moldofsky *et al.* (1995) retrospectively compared clinical and polysomnographic

Table 2 Case reports of sleep-related murder and legal cases during which sleep-related murder was used as a defence (irrespective of the final verdict)

Case name (reference)	Murder	Nature of episode	Verdict of court
Bonkalo (Bonkalo, 1974)	Defendant awakened by noise around midnight. Grabbed axe and attacked 'stranger in room', killing his wife instead	Confusional arousal	Not reported
Bonkalo (Bonkalo, 1974)	Night shift supervisor fell asleep in office, ~30 min later employee entered office and awakened him, supervisor pulled gun in confusion and fired, killing employee	Confusional arousal	Not reported
Bonkalo (Bonkalo, 1974)	Boy tried to pick up something next to sleeping defendant, who was aroused by disturbance, grabbed knife and stabbed boy	Confusional arousal	Not reported
Bonkalo (Bonkalo, 1974)	Knight stabbed friend to death (was asleep when friend tried to awaken him)	Confusional arousal	Not reported
Fain versus Commonwealth as cited by Pressman (2007)	Porter entered darkened hotel room unannounced and awakened defendant who shot hotel porter three times	Confusional arousal	Convicted and reversed on appeal
Tibbs versus Commonwealth as cited by Pressman (2007)	Victim attempted to arouse defendant from sleep, was stabbed to death with knife	Confusional arousal	Convicted and reversed on appeal
Bradely versus State as cited by Pressman (2007)	Disturbed by noise while asleep, jumped up with gun and started firing, found girlfriend dead on bed	Confusional arousal	Acquitted
Podolsky (Podolsky, 1959)	Awakened to find hands around neck of prostitute he had slept with	Confusional arousal	Not reported
HMS advocate versus Fraser as cited by Pressman (2007)	Reported image of wild animal rising from floor and attacking child. Tried to defend child from beast, grabbing child instead and smashing him against the wall, killing him	Sleep terror followed by sleepwalking	Acquitted
R. versus Nhete as cited by Pressman (2007)	While sleeping around campfire, dreamt that he was being burnt. Jumped up and grabbed axe, killing a man close to him	Sleep terror followed by sleepwalking	Acquitted
R. versus Cogden as cited by Pressman (2007)	Vivid image of soldiers attacking daughter. Left house, grabbed axe, entered daughter's room and 'defended her' by hitting twice with axe, killing her	Sleep terror followed by sleepwalking	Acquitted
R. versus Dhalami as cited by Pressman (2007)	Dreamt he was being attacked, 'defended himself', stabbing man to death	Sleep terror followed by sleepwalking	Not reported
Podolsky (Podolsky, 1959)	Dreamed that burglars had entered home and were killing family. Grabbed two guns and fired 10 shots, killing father and brother, injuring mother	Sleep terror followed by sleepwalking	Acquitted
Howard and D'Orban (Howard and D'Orban, 1987)	Image of two Japanese soldiers chasing him and wife through jungle. In his dream, he strangled one soldier and kicked another, killing his wife by strangling her instead	Sleep terror followed by sleepwalking	Acquitted
Bonkalo (Bonkalo, 1974)	Woken up by wife who was shouting incorrectly that there were burglars in house. Grabbed gun, went to window and shot night watchman instead	Sleepwalking	Not reported
Pennsylvania versus Ricksgers as cited by Pressman (2007) and Nofzinger and Wettstein (Nofzinger and Wettstein, 1995)	Shot wife in hip. Bullet reflected upward resulting in her death	Sleepwalking	Convicted
Nofzinger (Nofzinger and Wettstein, 1995)	Shot his wife with gun ~1 h after falling asleep. Was known for sleep apnoea and chronic respiratory failure	Sleepwalking	Convicted
R. versus Parks (Broughton, 1994)	Stabbed mother-in-law to death and injured father-in-law after a 20 min ride in car	Sleepwalking	Acquitted
Arizona versus Falater (Cartwright, 2004)	Stabbed wife 44 times, 45 min later found her apparently alive, dragged her to swimming pool and held head under water	Sleepwalking	Convicted
California versus Reitz as cited by Pressman (2007)	Smashed flowerpot against girlfriend's head, dislocated elbow and wrist, stabbed in back of neck with pocketknife, killing her	Sleepwalking	Convicted
R. versus Catling as cited by Pressman (2007)	Stabbed girlfriend nine times and cut throat while she was asleep. Involvement of zopiclone	Sleepwalking	Withdrew sleepwalking defense and pleaded guilty

Adapted from Pressman (2007a), with permission; R. = Rex/Regina.

characteristics of subjects with sleepwalking and sleep terrors and found that violent behaviour directed towards other people occurred preferentially in males and was significantly associated with more stressors, a disturbed sleep schedule, excessive use of caffeinated beverages, drug abuse and less stage 4 non-REM sleep. The retrospective nature of this study and the fact that occurrence of violence and associated factors was based uniquely on the individual's subjective recall represents major limitations of this study. Guilleminault *et al.* (1995) retrospectively reviewed a series of 41 adult individuals with nocturnal wandering of different aetiologies. Compared with non-violent individuals, the 29 violent patients were predominantly male (65 versus 42%) and included the only two subjects with temporal lobe epilepsy.

Functional neuroimaging studies performed on sleeping subjects reveal that during non-REM sleep, the prefrontal cortex is hypoactive in comparison with the resting wakeful state (Maquet *et al.*, 1996, 1997; Braun *et al.*, 1997; Andersson *et al.*, 1998; Kaufmann *et al.*, 2006). The prefrontal associative cortices are functionally related to executive functions including planning, attention and judgement (Cummings, 1993). A single photon emission computed tomography study performed during an episode of sleepwalking documented hyperperfusion of the posterior cingulate cortex and cerebellar vermis in addition to decreased cerebral blood flow in the frontal and parietal association cortices (Bassetti *et al.*, 2000). In line with this observation, a recent intracerebral electroencephalogram (EEG) study showed that during confusional arousals, the motor and cingulate cortices are activated and display the same activity as during wakefulness, whereas the frontoparietal associative cortices exhibit an enhancement of delta activity characteristic of sleep (Terzaghi *et al.*, 2009). These findings suggest that disorders of arousal share characteristics of both sleeping and waking states, resulting from the selective activation of thalamo-cingulate pathways implicated in the control of complex motor and emotional behaviour, and from hypoactivation of other thalamocortical pathways, including those projecting to the frontal lobes. This constellation of regional brain activity during non-REM sleep shares a number of similarities with the presumed anatomical substrates of violence occurring during wakefulness. Dysfunction in the medial prefrontal and orbitofrontal region areas is indicated by impaired emotional recognition of faces, errors in odour identification and disadvantageous decisions in gambling tests, all of which have been observed in patients with impulsive aggressive disorder (Best *et al.*, 2002; Dileo *et al.*, 2008). Further evidence for the involvement of these areas is suggested by the finding that traumatic brain injuries are associated with violence if localized to the frontal ventromedial or orbitofrontal regions (Grafman *et al.*, 1996). Individuals with intermittent explosive disorder exhibit exaggerated amygdala activity and diminished activation of the orbitofrontal cortex in response to faces expressing anger, and fail to demonstrate coupling between these two structures (Coccaro *et al.*, 2007). According to functional models (Blair, 2004), the amygdala and the orbitofrontal lobe act on subcortical systems mediating reactive aggression. Although the role of the amygdala is to up- or down-regulate their response to threat, the orbitofrontal cortex exerts its modulating activity in response to social cues. It is thus conceivable that unwarranted aggressive behaviours occurring during parasomnias

are favoured by the unrestrained influence of the amygdala that is no longer kept in balance by proper introspection of executive function normally maintained by the dorsolateral prefrontal cortex, since it remains hypoactive.

In summary, arousal disorders represent a dissociated state with features of both wakefulness and sleep. Sleep violence in this setting seems to be favoured by hypoactivity of prefrontal associative cortices that are functionally related to planning, attention and judgement. It is further modulated at different levels—the predisposition for arousal disorders is mainly influenced by genetic and maturational factors. The occurrence of an arousal disorder on a particular night depends on a delicate balance between slow-wave sleep pressure and arousing factors. Violent acts occur more frequently in males, can be elicited by provocation (noise, touch) and are probably influenced by dream imagery and mental content.

Rapid eye movement sleep behaviour disorder

RBD is characterized by the loss of normal muscle atonia and an increase of phasic muscle activity during REM sleep, and is associated with altered dream content and acting out of dreams. It is more prevalent in males after the age of 50. In at least 50% of cases, it represents the first manifestation of a neurodegenerative disorder including Parkinson's disease, multisystem atrophy and dementia with Lewy bodies (Iranzo *et al.*, 2006b). RBD may also occur in the course of narcolepsy (Schenck and Mahowald, 1992; Nishino and Kanbayashi, 2005), secondary to brainstem lesions (Kimura *et al.*, 2000; Plazzi and Montagna, 2002; Zambelis *et al.*, 2002; Provini *et al.*, 2004; Tippmann-Peikert *et al.*, 2006; Mathis *et al.*, 2007) or limbic encephalitis (Iranzo *et al.*, 2006a; Lin *et al.*, 2009). Dreams of patients with idiopathic and secondary RBD have a more aggressive content compared with dreams of normal subjects, despite non-elevated or even lower levels of daytime waking aggressiveness (Fantini *et al.*, 2005). Likewise, among patients with Parkinson's disease, those affected by RBD have more aggressive dreams compared with patients without RBD, irrespective of gender (Borek *et al.*, 2007).

Violence in rapid eye movement sleep behaviour disorder

Violence with RBD results in injury to the patient or to the bed partner in 32–69% of cases (Comella *et al.*, 1998; Olson *et al.*, 2000; Scaglione *et al.*, 2005). Harm to the patient himself generally occurs when the patient hits the furniture or wall, or falls out of bed, and can result in serious injuries, including subdural haematomas, high cervical fractures and other fractures (Dyken *et al.*, 1995; Comella *et al.*, 1998; Olson *et al.*, 2000). Attempted assault of sleep partners has been reported to occur in 64% of cases, with injuries in 3% (Olson *et al.*, 2000). It may be potentially lethal to the affected person and to the bed partner (Schenck *et al.*, 2009). Unlike violence related to disorders of arousal, in RBD the individual is readily oriented upon awakening and can generally recall vivid dream imagery related to the violent act. Figure 1 shows a practical example of sleep violence in the context of RBD.

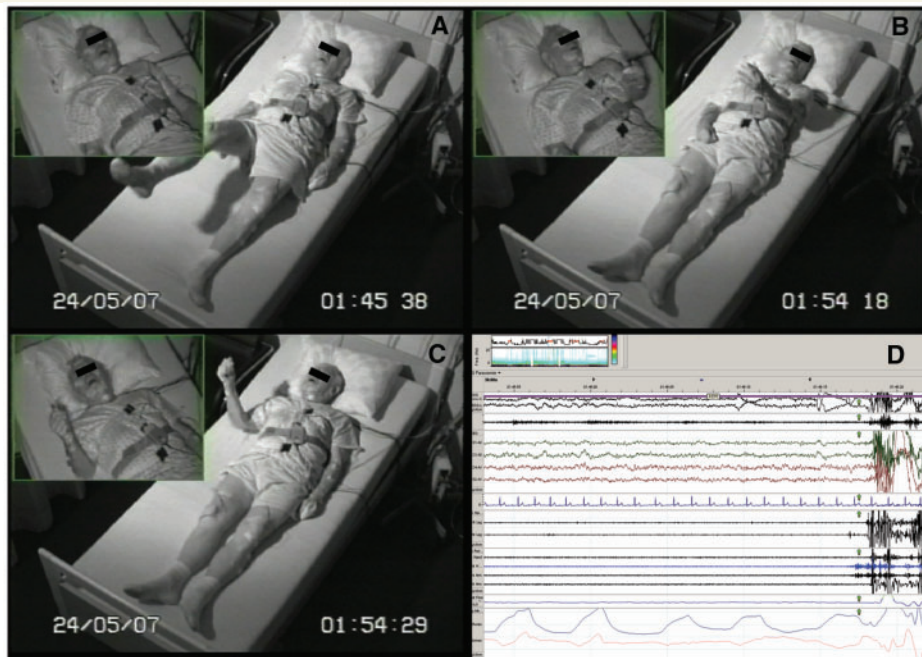


Figure 1 An 82-year-old male with a 2-year history of symmetrical extrapyramidal symptoms and cognitive dysfunction, attributed to vascular leucoencephalopathy. According to his wife, he displayed frequent vocalizations and violent behaviour during sleep. Polysomnographic recordings (D) show loss of normal muscle atonia during REM sleep and increased phasic muscle activity, as well as acting out of dreams with kicking (A), hitting (B) and menacing gestures (C). Based on these features, the diagnosis of RBD was established.

Lesion studies performed on cats (Jouvet and Delorme, 1965; Hendricks *et al.*, 1982; Lai and Siegel, 1988, 1990, 1997; Shouse and Siegel, 1992; Morrison, 1998) have suggested that absence of motor activity during normal REM sleep relies on two main mechanisms: (i) active inhibition of spinal motor neurons (i.e. REM atonia), and (ii) reduced drive of locomotor centres (i.e. leading to reduced phasic muscle activity). In the rat, potential neuroanatomical substrates for the control of REM sleep have been identified (Boissard *et al.*, 2002, 2003; Lu *et al.*, 2006): 'REM off' regions localized in the ventrolateral part of the periaqueductal grey matter and the lateral pontine tegmentum would interact in a mutually inhibitory way with 'REM on' regions (i.e. the sublaterodorsal nucleus and precoeruleus region) that project to the medulla and spinal cord, with a net inhibitory effect on motor neurons. Based on lesion and neuropathological studies as well as pharmacological effects, similar mechanisms have been postulated to be operative in humans (Boeve *et al.*, 2007). The precise nature and localization of the locomotor generators, which are presumed to project to the spinal motoneurons, either directly or indirectly via other brainstem nuclei, has yet to be defined. The observation that limbic encephalitis in humans, without evidence for brainstem dysfunction, is associated with RBD (Iranzo *et al.*, 2006a) and the altered dream content in RBD (Fantini *et al.*, 2005; Borek *et al.*, 2007) suggest a role of the limbic system in the pathogenesis of RBD, possibly by modulation of connections between the limbic system and the brainstem regions responsible for REM sleep atonia. Studies using single photon emission computed tomography imaging showed hypoperfusion in frontal

and temporo-parietal cortical areas in patients with RBD compared with control subjects, providing further evidence for involvement of supratentorial structures in the pathophysiology of RBD (Shirakawa *et al.*, 2002; Mazza *et al.*, 2006).

In summary, disorders causing RBD affect brainstem structures or supratentorial limbic regions. Dream enactment occurs in the presence of increased activity of locomotor centres and incomplete REM sleep associated muscle atonia. Violence in this setting is related to enactment of aggressive dream content and shows a strong male predominance.

Parasomnia overlap disorder and status dissociatus

Parasomnia overlap disorder is diagnosed when patients fulfil criteria for both RBD and disorders of arousal (American Academy of Sleep Medicine, 2005). In status dissociatus, features of each one of the three states (REM, non-REM and wakefulness) are simultaneously present, in the absence of identifiable conventional sleep stages (American Academy of Sleep Medicine, 2005). Both conditions can be associated with violent behaviour (Mahowald and Schenck, 1991; Schenck *et al.*, 1997).

Epilepsy

In nocturnal frontal lobe epilepsy, seizures can occur exclusively during sleep and can be associated with violence. Paroxysmal arousals consist of abrupt arousals from sleep with stereotyped

motor phenomena, including head movements, frightened expression and dystonic limb posturing. Episodes of the so-called nocturnal paroxysmal dystonia involve more complex motor activity such as bipedal automatisms (kicking, cycling), rhythmic movements of the trunk (sometimes with semi-purposeful repetitive movements mimicking sexual activity) and limbs, as well as tonic and dystonic posturing. Autonomic activation with tachycardia and hypertension generally accompanies the episodes. Epileptic nocturnal wanderings generally start with an abrupt arousal, proceed through the stage of paroxysmal dystonia and eventually culminate in perambulation. Alerting stimuli have been shown to trigger seizures in patients with autosomal dominant nocturnal frontal lobe epilepsy (El Helou *et al.*, 2008). On awakening, individuals

are generally rapidly oriented, unlike in other seizure types, and there is no prolonged post-ictal confusion (Provini *et al.*, 1999). Clinical manifestations of seizures are often very similar to the motor activity observed during disorders of arousal, making a distinction between the two conditions difficult (distinguishing features are summarized in Table 3). Nocturnal hypermotor seizures (Nobili *et al.*, 2004; Ryvlin, 2006) including epileptic nocturnal wanderings (Nobili *et al.*, 2002) can occasionally originate from the temporal lobe.

Violence in epilepsy

Injury resulting from nocturnal seizures can be accidental and related to hyperkinetic features (Schenck *et al.*, 1989a; Fig. 2), although

Table 3 Distinguishing features between nocturnal seizures in nocturnal frontal lobe epilepsy and arousal disorders

Features	Disorders of arousal	Nocturnal frontal lobe epilepsy
Age of onset	Childhood	Childhood or adolescence
Persistence into adulthood	Uncommon	Frequent
Motor features	Variable, not highly stereotyped, no dystonic posturing	Highly stereotyped, often hyperkinetic
Amnesia for event	Generally present	Generally present
Post-ictal confusion	Frequent	Generally absent
Duration	Generally >30 seconds	Seconds to 3 minutes
Same-night recurrence	Low	High
Timing	First-third of the night	Any time of the night, often in clusters
Ictal EEG	Slow waves	Clear-cut epileptiform discharges in <10%

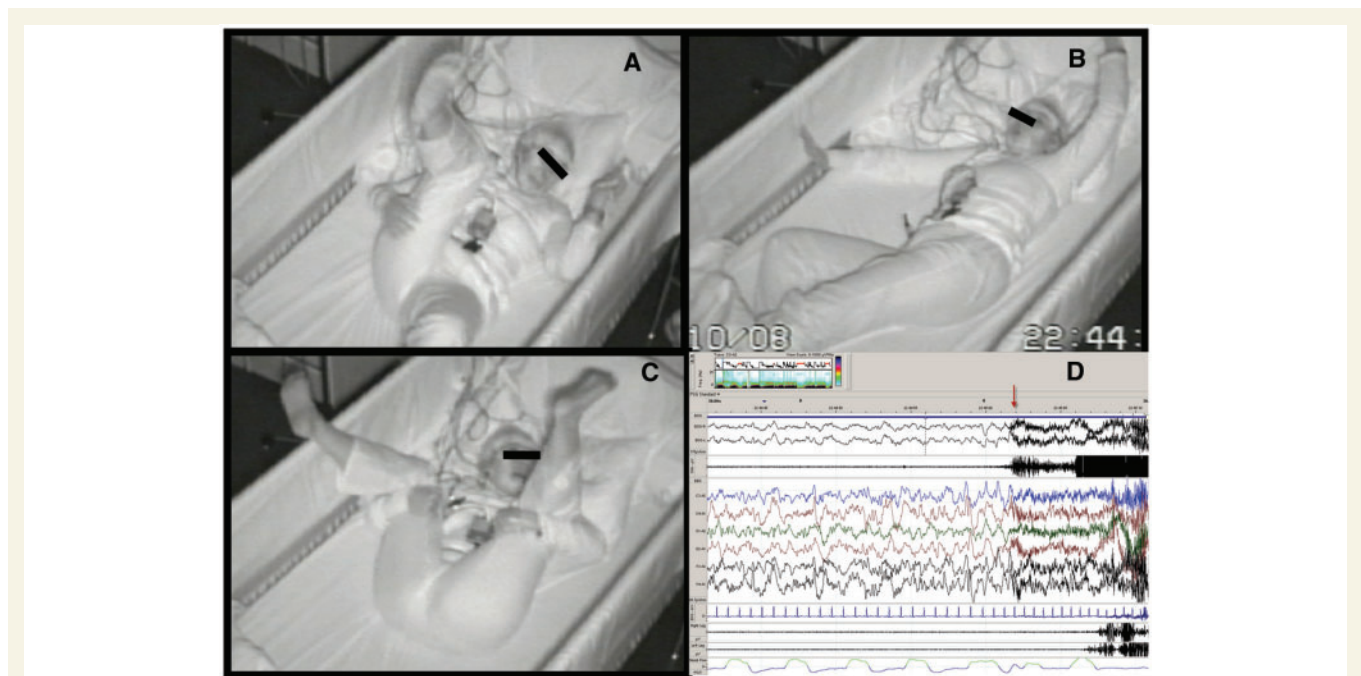


Figure 2 A 36-year-old female with nocturnal frontal lobe epilepsy since the age of 13. Seizures occurred up to 10 times a night and were characterized by an abrupt awakening from sleep with a feeling of chest tightness and dyspnoea, and progressed to tonic posturing of the limbs and hyperkinetic features, including elevation and extension of the left arm (B) and pedalling (A and C). The patient was fully aware during the seizure but was unable to speak. She had sustained several injuries including fractures of her fingers when hitting objects during the seizures. The red arrow on the polysomnographic recording (D) shows the beginning of a seizure arising from slow wave sleep. Intracranial EEG recordings indicated that seizures originated from a cortical dysplasia in the left frontal cingulum.

compared with seizures during wakefulness, the injury potential of seizures occurring solely during sleep is probably lower, as the bed represents a relatively safe environment (Wirrel, 2006). Ictal aggression is exceptional (Rodin, 1973; Delgado-Escueta *et al.*, 1981; Ramani and Gumnit, 1981; Marsh and Krauss, 2000; Tassinari *et al.*, 2005a; Reuber and Mackay, 2008). It can take the form of biting, grasping, hitting, threatening, screaming, facial expressions of anger, pushing, shoving and spitting (Marsh and Krauss, 2000; Tassinari *et al.*, 2005a) and has been reported to occur during episodes of nocturnal epileptic wandering (Maselli *et al.*, 1988; Schenck *et al.*, 1989a; Guillemainault *et al.*, 1995; Plazzi *et al.*, 1995). The violent act generally starts abruptly, is of short duration, is not intentionally directed towards others and does not involve intricate skills or purposeful movements. Some behavioural patterns, such as ictal grasping or repetitive limb movements, although not directed towards others, can be misinterpreted by bystanders as threatening, intentional gestures. Ictal aggression is recurrent, out of character and stereotyped for a given patient, and is frequently associated with amnesia (Marsh and Krauss, 2000). These kinds of epileptic manifestations seem to be

more common in males (Rodin, 1973; Delgado-Escueta *et al.*, 1981; Marsh and Krauss, 2000; Tassinari *et al.*, 2005a) and are more frequently observed in seizures originating from the non-dominant hemisphere (Tassinari *et al.*, 2005a). Peri-ictal aggression occurs in the pre-ictal, or more commonly in the post-ictal period, in a setting of confusion and abnormal mood (depression, psychosis or delirium) (Fig. 3). More directed violence (towards others) can occur as a reaction to stimuli of the patient's environment; such as the act of restraining the patient (Delgado-Escueta *et al.*, 1981; Tassinari *et al.*, 2005a).

Intracranial EEG investigations have demonstrated that seizures characterized by violent complex behaviours and intense fear are associated with a predominantly bilateral dysfunction of a network involving the cingulate, orbitofrontal and temporal regions (the amygdala, hippocampus and lateral temporal cortex) (Tassinari *et al.*, 2005a). As with disorders of arousal, these potential anatomical substrates are similar to those postulated to underlie violence during wakefulness. Additional observations indicate that arousal disorders and seizures in nocturnal frontal lobe epilepsy might share a final common pathway. Both conditions have a



Figure 3 A 32-year-old male with frontal lobe epilepsy since the age of 18 years. Seizures started with an aura characterized by cephalic tingling and fear followed by left head deviation associated with clonic jerks (first in the left arm, then rapidly bilaterally). Consciousness was preserved. Nocturnal seizures were occasionally followed by a secondary generalization with a post-ictal phase characterized by violent movements and auto-aggressive behaviour. The patient could throw himself against the wall (first line of photogram), fall out of bed (second line) or leave the room (third line). Intracerebral EEG recordings showed that seizures originated from a cortical dysplasia in the right frontal superior gyrus.

similar clinical semiology. Patients with nocturnal frontal lobe epilepsy have a high prevalence of parasomnias in the personal or family history compared with large control populations (Provini *et al.*, 1999; Bisulli *et al.*, 2010). Finally, single photon emission computed tomography imaging findings documented cingular and cerebellar hyperperfusion in both sleepwalking and epileptic paroxysmal arousals (Bassetti *et al.*, 2000; Vetrugno *et al.*, 2005). It has been postulated that the final common pathway between arousal disorders and seizures in nocturnal frontal lobe epilepsy might imply activation of so-called central pattern generators in the brainstem that subserve stereotyped innate motor behaviour necessary for survival, such as locomotion, swimming, sexual activity, other rhythmic motor sequences and aggressive behaviour (as an innate action pattern aimed at the defence of the personal space) (Tassinari *et al.*, 2003, 2005b, 2009). However, solid evidence for the existence of central pattern generators in humans is still lacking.

In summary, ictal sleep-related violence results from direct activation of specific brain regions in the frontal and temporal lobes. Alternatively, violence can result from hyperkinetic features or occur in the post-ictal period, in a setting of confusion or abnormal

mood. Nocturnal frontal lobe epilepsy and arousal disorders have similar patterns of brain activation and share common favouring factors (sleep deprivation, provocation), suggesting a common final pathway.

Nocturnal dissociative disorders

Dissociative disorders are defined as a disruption of the usually integrated functions of consciousness, memory, identity or perception of the environment that occur without the conscious awareness of the part of the individual (American Psychiatric Association, 2000). They are sometimes associated with violence (McCaldon, 1964) and may arise exclusively or predominantly from the sleep period (Schenck *et al.*, 1989b; Fig. 4) during well-established EEG wakefulness, either at the transition from wakefulness to sleep or within several minutes after awakening from stages 1 or 2 of non-REM or from REM sleep (American Academy of Sleep Medicine, 2005). Patients are generally female, have a history of sexual abuse and post-traumatic stress disorder and often have additional dissociative episodes during daytime (Schenck *et al.*, 1989b).

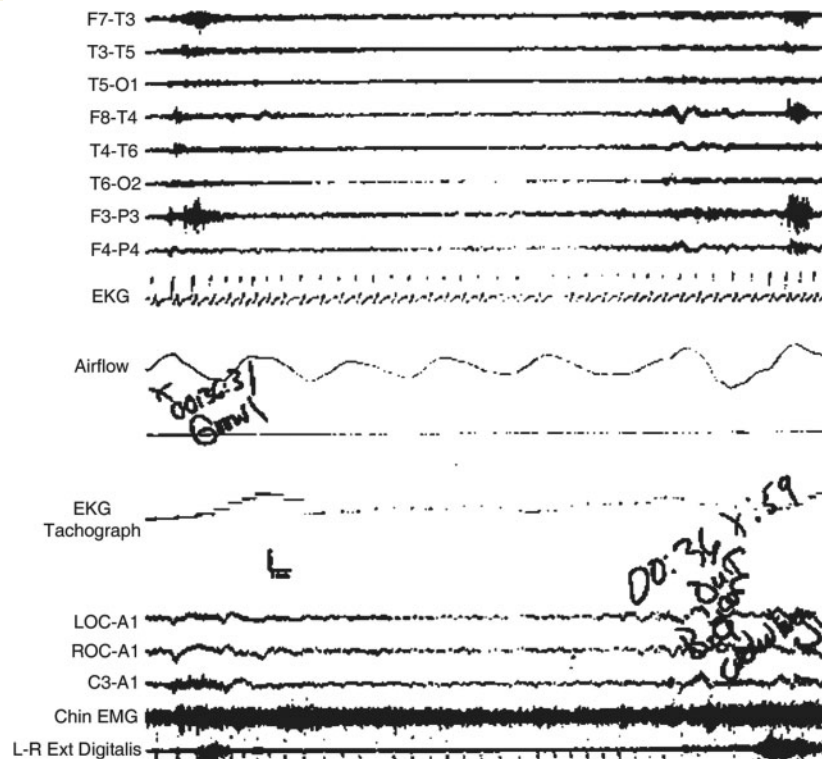


Figure 4 Fifty-three minutes after sleep onset in the sleep lab, a 19-year-old male suddenly begins to growl and then 28 s later he leaves the bed and crawls away in the manner of a large jungle cat, as noted by the sleep technologist. A nine channel EEG indicates a corresponding wakeful state. For 4 years, once or twice weekly, he had acted like a large jungle cat (with deep, persistent growling) for 30–60 min during the nocturnal sleep period at home. His parents commented on his ‘super-human strength’ during these episodes, such as leaping far from his bed, lifting a marble table with his jaws, lifting a mattress with his jaws and then dragging it across a room. He frequently left imprints of his teeth on wooden furniture. He often bruised and lacerated himself all over his body from aggressive and violent behaviour during these episodes, and also repeatedly injured his lips and gums from biting sharp objects. He has never had a dissociative episode during the daytime, so his case represents an exclusively nocturnal, sleep-related animalistic dissociative (multiple personality) disorder. Reprinted from Schenck *et al.* (1989b), with permission.

Factitious disorder

In factitious disorder (also named Münchhausen syndrome), an individual presents with an illness that is deliberately produced or falsified for the purpose of assuming the sick role or produces symptoms in another person (factitious syndrome by proxy) (American Psychiatric Association, 2000). Both disorders may occur during the sleep period and can be associated with violence (Griffith and Slovik, 1989; Mahowald *et al.*, 1992).

Malingering

In contrast to factitious disorder, malingering is not a mental illness and intentionally produced symptoms or signs are motivated by external incentives for the behaviour (i.e. economic gain, avoiding legal responsibility, improving physical well-being) (American Psychiatric Association, 2000). Again, symptoms can be referred to the sleep period, but when observed, always occur during well-established EEG-wakefulness.

Pathophysiological considerations

In light of current knowledge about sleep disorders outlined in the previous sections, it becomes apparent that the occurrence of sleep-related violence is modulated at two different levels: (i) dissociation of states; and (ii) factors specifically linked to sleep violence.

Dissociation of states

Parasomnias underlying sleep violence represent a 'dissociation' of the normally well-defined three states of being, sharing features of both wakefulness and either non-REM or REM sleep (Mahowald, 2008). State dissociation results from simultaneous and selective activation of some brain regions [e.g. thalamocingulate pathways in arousal disorders (Bassetti *et al.*, 2000; Terzaghi *et al.*, 2009); and probably limbic regions in RBD (Iranzo *et al.*, 2006a)] and deactivation of others [i.e. the frontal and parietal associative cortex in arousal disorders (Bassetti *et al.*, 2000; Terzaghi *et al.*, 2009) and pathways regulating muscle atonia in RBD (Boeve *et al.*, 2007)]. In some conditions such as epilepsy, activation of specific brain regions alone seems to be sufficient to induce the disorder. Several factors modulating state dissociation have been identified: genetic predisposition [as indicated by the presence of genetic markers and familial clustering of arousal disorders (Kales *et al.*, 1980; Hublin *et al.*, 1997; Lecendreux *et al.*, 2003)], maturational factors (accounting for the strong prevalence of arousal disorders in childhood compared with adulthood), neurodegeneration [as demonstrated by the strong association between RBD and degenerative disorders (Iranzo *et al.*, 2006b)] and other cerebral disorders [such as limbic encephalitis resulting in RBD (Iranzo *et al.*, 2006a; Lin *et al.*, 2009)].

Factors specifically linked to sleep violence

Most complex behaviours observed in the course of parasomnias and seizures are benign. Why and in what circumstances violent behaviour emerges has yet to be defined. The most consistent risk factor identified in this respect is male gender, which is strongly associated with violence in general, including sleep violence. The emotional state and dream content, which have been shown to be predominantly unpleasant and aggressive in RBD and arousal disorders (Fantini *et al.*, 2005; Borek *et al.*, 2006; Oudiette *et al.*, 2009), most likely play a role in the occurrence of sleep violence. Interaction with the sleeper, i.e. provocation by noise and touch (Pressman, 2007a) are additional factors that have been shown to be related to violence in arousal disorders and sleep-related seizures.

Diagnostic procedures

History and physical examination

The first step in diagnosing a sleep disorder associated with violence is obtaining a complete history, preferably from both the patient and the bed partner. Particular emphasis should be placed on identifying the key features that allow a distinction among the different sleep disorders that have been discussed earlier (Table 1). Information on sleep and wake habits, comorbidities (particularly neurological and psychiatric conditions) and drug intake should also be obtained. Depending on the suspected diagnosis, the history might be complemented by clinical scales, such as the REM sleep behaviour disorder screening questionnaire (Stiasny-Kolster *et al.*, 2007) or the nocturnal frontal lobe epilepsy and parasomnia scale (Derry *et al.*, 2006b). The clinical history should be followed by general physical, neurological and psychiatric examinations, and in cases with suspected cerebral lesions, by imaging studies of the brain and neuropsychological testing.

Home videos

Documentation of nocturnal episodes with home videos using a camera with infrared night vision function is useful for differential diagnosis (Derry *et al.*, 2009; Nobili, 2009), as various sleep disorders such as sleepwalking often fail to occur in a laboratory setting.

Polysomnography

An extensive polygraphic study with a multichannel scalp EEG, electromyographic monitoring of all four extremities and continuous, time-synchronized audiovisual recording is essential. Even if they fail to capture the event, long-term recordings are useful to identify typical or specific inter-ictal EEG markers. Video-EEG-polysomnography is superior to standard polysomnography for the evaluation of parasomnias because of the increased capability to identify and localize EEG abnormalities and to correlate behaviour with EEG and polysomnography (Aldrich and Jahnke, 1991).

Technicians are often of help, as they can give information on possible precipitating factors of events (e.g. noise) and can interact with the patient during and after the event in order to assess the level of consciousness, the ability to speak and the presence of post-ictal confusion or dream recall.

Arousal disorders

Sleep macro-architecture is essentially normal in patients with arousal disorders (Schenck *et al.*, 1998; Pressman, 2004; Zadra *et al.*, 2004), but may show increased numbers of arousals or fragmentation of slow-wave sleep, in particular in the first non-REM sleep episode (Espa *et al.*, 2000; Guilleminault *et al.*, 2001). Hypersynchronous delta activity, consisting of bilateral rhythmic delta-waves occurring for 10–20 s during slow-wave sleep, is considered as typical for arousal disorders, but is non-specific, given that it occurs in up to 66% of normal arousals (Pressman, 2004; Zadra *et al.*, 2004). Abnormalities of the cyclic alternating pattern, representing infra-slow oscillations observed on EEG between phases of sleep promoting slow oscillations and wake-promoting cortical arousals have been documented in chronic sleepwalkers. These include a decrease in phase A1 and an increase in phases A2 and A3 (Guilleminault *et al.*, 2006). Even if polysomnographic recordings fail to capture an event, they are useful in identifying a coexistent trigger for parasomnias that requires specific treatment (i.e. sleep apnoea as a trigger for sleepwalking). Prior sleep deprivation has been shown to increase the diagnostic yield of these studies (Joncas *et al.*, 2002; Pilon *et al.*, 2008).

Epilepsy

Documentation of epileptic potentials is diagnostic and specific in patients with suspected nocturnal seizures. Unfortunately, certain areas of the frontal lobe (e.g. the medial and orbital cortex) are not accessible to surface EEG recordings, so that inter-ictal epileptic potentials cannot be documented in up to 60% of cases (Bautista *et al.*, 1998; Kellinghaus and Luders, 2004). Even ictal recordings remain inconclusive in 20–40% of cases due to overlapping motor artefacts (Williamson and Spencer, 1986; Morris *et al.*, 1988; Laskowitz *et al.*, 1995). Additional daytime EEG, preferentially after sleep deprivation, should be carried out in patients with suspected seizures. Distinguishing features between nocturnal seizures and disorders of arousal have been reviewed (Derry *et al.*, 2006a; Nobili, 2007; Tinuper *et al.*, 2007) and are summarized in Table 3. A clinical scale has been developed with the aim to distinguish between these conditions (Derry *et al.*, 2006b) although some items seem to limit its sensitivity (Manni *et al.*, 2008).

Rapid eye movement sleep behaviour disorder

The polygraphic documentation of increased phasic muscle activity and loss of normal muscle atonia during REM sleep is currently necessary to establish the diagnosis of RBD (American Academy of Sleep Medicine, 2005). A recent controlled study of patients with

RBD has provided cut-off scores for tonic and phasic EMG activity during REM sleep, with excellent specificity, to assist in objectively confirming the diagnosis of RBD (Montplaisir *et al.*, 2010). Nevertheless, certain caveats need to be considered in this diagnostic process (Mahowald *et al.*, 2010). Video-polysomnography typically shows rapid twitches of the extremities or more complex movements representing dream enacting behaviour occurring during REM sleep. Although on awakening, the individual can frequently recall a vivid dream corresponding to the behaviour displayed, the presence or absence of remembered dream imagery does not reliably differentiate between disorders of arousal and RBD (Schenck, 2005; Oudiette *et al.*, 2009). Also, a history of dream enactment is not required by the international classification of sleep disorders for diagnosing RBD, since this is not a universal phenomenon in RBD (American Academy of Sleep Medicine, 2005).

Treatment

General principles

Treatment is aimed at the specific aetiology. In all cases, the environment of the sleeping patient should be made safe, which can be accomplished by advising the patient to sleep on the first floor or in the basement and away from windows, to secure doors and windows and remove potentially dangerous objects from the bedrooms. In the USA, where the Second Amendment to the Constitution maintains an individual and collective 'right to keep and bear arms', patients and family members should be questioned concerning the presence of firearms in the home. When indicated, appropriate safety measures (ideally removing the firearms, or at the very least disarming, securing and locking them away) should be undertaken. Patients and close relatives should be informed as to the nature of the events and the fact that violent behaviour in the course of parasomnias or seizures is not intentional.

Disorders of arousal

In disorders of arousal, any underlying trigger or precipitating factor should be treated or avoided (i.e. sleep deprivation; coexisting disorders such as sleep apnoea, but also touching or awakening the patient). In fact, successful treatment of sleep disordered breathing has been shown to allow control of sleepwalking episodes (Guilleminault *et al.*, 2005).

The drug of choice for the treatment of arousal disorders is clonazepam, which should be started at a dosage of 0.5 mg at bedtime and progressively increased up to 2–3 mg if necessary and well tolerated. Other benzodiazepines (including diazepam, alprazolam, triazolam and flurazepam), antiepileptics (e.g. carbamazepine, phenytoin and gabapentin) and antidepressants (e.g. imipramine, paroxetine, trazodone), as well as melatonin have been shown to be effective, although reported data rely on small case series or single cases (Cooper, 1987; Kavey *et al.*, 1990; Lillywhite *et al.*, 1994; Wilson *et al.*, 1997; Guilleminault *et al.*, 2005; Hughes, 2007). There are no randomized controlled

studies of any pharmacological treatment of disorders of arousal (Harris and Grunstein, 2009). Nevertheless, there is a consensus among experienced sleep physicians who have managed many patients with disorders of arousal that bedtime therapy with benzodiazepines is usually highly effective. Behavioural treatments such as hypnosis can also be helpful (Mahowald and Schenck, 2005a).

Rapid eye movement sleep behaviour disorder

Controlled, randomized, double blind studies evaluating the efficacy of pharmacotherapy in RBD are lacking. Clonazepam is considered as the treatment of choice for RBD and has been shown to be effective, with reported success rates between 87% and 90% (Schenck *et al.*, 1993; Olson *et al.*, 2000). Its mechanism of action in this disorder is not completely understood. Polysomnographic recordings show that it diminishes phasic muscle activity during REM sleep without re-establishing normal muscle atonia. Clonazepam should be given with an initial dosage of 0.5 mg, and can be increased to 1 or 2 mg progressively if necessary (Gagnon *et al.*, 2006). Although it is generally regarded as safe, this drug should be used with caution in elderly patients because of the risk of falling and developing a confusional state. It can also be associated with worsening of obstructive sleep apnoea.

Alternatively, melatonin is an option, especially in patients with multiple comorbidities (Kunz and Bes, 1997, 1999; Takeuchi *et al.*, 2001; Boeve *et al.*, 2003; Anderson and Shneerson, 2009). Given at bedtime, a dose range of 3–9 mg has been shown to reduce RBD symptoms and to partially restore REM sleep atonia (Kunz and Bes, 1997, 1999; Boeve *et al.*, 2003). The mechanisms underlying its efficacy are not known.

Drugs with conflicting effects on RBD include acetylcholinesterase inhibitors (Grace *et al.*, 2000; Maclean *et al.*, 2001; Boeve *et al.*, 2003) and dopaminergic drugs (Sharf *et al.*, 1978; Loudon *et al.*, 1995; Tan *et al.*, 1996; Garcia-Borreguero *et al.*, 2002; Fantini *et al.*, 2003; Yamauchi *et al.*, 2003; Ozekmekci *et al.*, 2005; Schmidt *et al.*, 2006; Kumru *et al.*, 2008).

Epilepsy

Nocturnal seizures require specific antiepileptic treatment. In cases of nocturnal frontal lobe epilepsy, carbamazepine is the treatment of choice. It completely abolishes seizures in 20% of patients and reduces seizures in nearly half of patients (Provini *et al.*, 1999). Approximately 20% of patients have pharmacoresistant seizures (Provini *et al.*, 1999). Patients with drug resistant, disabling seizures should be considered for resective surgery, which may provide excellent results in selected cases (Nobili *et al.*, 2007).

Forensic issues

Sleep-related automatism that occur in the course of parasomnias or seizures usually qualify for diminished responsibility because the person is not conscious of his act, its consequences and of the fact that it is wrong. A criminal conviction in many western

countries is secured upon proving two essential elements: *mens rea* (guilty mind) and *actus reus* (the accomplished act). In alleged violent behaviour arising from sleep, *actus reus* is usually never in doubt, whereas the medical expert will need to provide compelling arguments related to *mens rea*, or the claimant's degree of consciousness. Applying this concept to a particular act can be problematic for a variety of reasons. First, there are several degrees of consciousness, with numerous transitions between normal and pathological sleep (Broughton and Shimizu, 1995). Second, there is currently no diagnostic tool that enables the diagnosis of an underlying sleep disorder with absolute certainty, and even if there was one, the presence of a sleep disorder does not necessarily establish causal link to committed act and, more importantly, cannot replicate the element of *mens rea* associated with the alleged criminal act. The judicial concept of automatism is considerably different from the medical concept; in common law countries, a distinction is made between insane automatism, which are caused by intrinsic causes, and sane automatism that result from extrinsic factors. According to this classification, automatism in the context of a seizure or parasomnia would be considered as insane. For the accused, sane and insane automatism have very different legal consequences. If considered insane, the person is committed to a medical institution for an indefinite period of time; if deemed sane, the accused is acquitted without any mandated medical consultation or follow-up (Mahowald and Schenck, 2005b).

A sleep-related violent offence must be distinguished from a simulation. Criteria for establishing the putative role of an underlying sleep disorder in a specific violent act have been proposed by Mahowald and coworkers (1990, Table 4). In addition to a medical evaluation, an extensive forensic-psychiatric evaluation is required in this setting, aimed at identifying a plausible offense mechanism based on the offender's behavioural pattern and personality profile. In the presence of a plausible wakeful offense mechanism, the assumption of a sleep-related violent offence becomes more unlikely.

Table 4 Criteria for establishing the role of an underlying sleep disorder in a violent act (Mahowald *et al.*, 1990)

Presence of an underlying sleep disorder
Presence of solid evidence supporting the diagnosis
Previous occurrence of similar episodes
Characteristics of the act
Occurs on awakening or immediately after falling asleep
Abrupt onset and brief duration
Impulsive, senseless, without apparent motivation
Lack of awareness of individual during event
Victim: coincidentally present, possible arousal stimulus
On return to consciousness
Perplexity, horror, no attempt to escape
Amnesia for event
Presence of precipitating factors
Attempts to awaken the subject
Intake of sedative/hypnotic drugs
Prior sleep deprivation
Alcohol or drug intoxication precludes the use of disorder of arousal in forensic cases

Summary

Bona fide sleep-related violence occurs in a heterogeneous group of disorders. Violence may represent enactment of aggressive dreams as in RBD, emerge as a primitive behaviour in a setting of hypoactivity of the prefrontal associative cortices subserving complex cognitive functions, as occurs in arousal disorders, or result from direct activation of specific frontal and temporal networks as with nocturnal epileptic seizures. Although the precise mechanisms leading to sleep violence are not fully understood, a number of potential anatomical substrates and risk factors have been identified. Knowledge of disorders underlying sleep violence and how to differentiate and diagnose them is of major importance. Not only are most of them treatable but the consequences of sleep violence are also often devastating for the affected individual and represent a significant concern for public health.

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