Current Perspectives on Sleep-Related Injury, Its Updated Differential Diagnosis and Its Treatment

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This article provides an update on the differential diagnosis of sleep-related behavior disorders (viz. parasomnias) that cause nocturnal injury, including life-threatening injury to self or bedpartner. A catalog of nocturnal injuries is provided. Current treatments are discussed. Guidelines are given on the assessment of injurious nocturnal behaviors. Extensive polysomnographic (PSG) monitoring and comprehensive clinical evaluations are required for the proper diagnosis (and any comorbidity) to be identified and the appropriate treatment(s) to be initiated. In 1989, a report on a series of 100 adults with recurrent sleep-related injuries identified five disorders as being responsible for the nocturnal injuries: disorders of arousal (sleepwalking/sleep terrors [SW/ST]: NREM parasomnias); rapid-eye-movement (REM) sleep behavior disorder (RBD); nocturnal dissociative disorders; nocturnal seizures; and obstructive sleep apnea/periodic limb movements. Other disorders known to cause sleep-related injuries include nocturnal eating disorders; nocturnal scratching disorders; rhythmic movement disorders; bruxism; cerebral anoxic attacks; drug intoxication and withdrawal states; and Munchausen syndrome by proxy. Five types of nocturnal seizures can cause sleep-related injury: complex partial seizures; frontal lobe seizures; paroxysmal nocturnal dystonia; episodic nocturnal wandering; and paroxysmal periodic motor attacks. Malingering, which is not a psychiatric disorder, can also produce the complaint of sleep-related injury. Treatment of injurious parasomnias is usually effective and safe, even with long-term, nightly treatment. Benzodiazepines, particularly clonazepam, are the cornerstone of treating injurious SW/ST and RBD, and are effective adjuncts in the treatment of various other parasomnias. Parasomnias are rarely a direct manifestation of a psychiatric disorder, and when co-morbidity is present, treatment of the psychiatric disorder alone does not usually control the parasomnia. Conversely, pharmacotherapy of psychiatric disorders can induce or exacerbate parasomnias. Parasomnias represent striking examples of dissociated states of mind and behavior surrounding sleep, and their scientific understanding requires a close interlinking of clinical and basic research. Parasomnias inherently carry forensic implications, which are discussed in this article. (Sleep and Hypnosis 2000;1:8-21)

Key words: parasomnias, REM sleep behavior disorder, sleepwalking/sleep terrors/disorders of arousal, nocturnal seizure disorders, nocturnal eating disorders, nocturnal scratching disorders, forensic medicine, injury

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INTRODUCTION

In the 1994 movie WOLF (directed by Mike Nichols and played by Jack Nicholson), which told the story of a man who was episodically transformed into a wolf, the star of the movie was repeatedly surprised to find himself stained with mud and blood upon awakening from sleep in the morning. He noticed that his clothes and shoes were also dirty. So he surmised that he was in-

volved with something strange-and violent-during the night. He was unaware of the process responsible for what he observed in the morning, and therefore could not understand it, but started becoming afraid of it. Since he wanted to avoid having any more strange nocturnal escapades, once it became dark each night, he would handcuff himself to a radiator.

This impressive nocturnal transformation on the silver screen of the cinema finds a parallel-across a spectrum of severity-in the clinical histories of patients with sleep-related injury and violence caused by parasomnias, which encompass the behavioral disorders of sleep. With parasomnias, behavioral disturbances arise during the nocturnal sleep period, and then cease upon awakening in the morning. The affected person is often unaware of his actions while engaging in parasomnia behaviors that can become violent and result in self-injury or harm to others in the vicinity, and then have amnesia for the events upon awakening. On the other hand, many adults do have some awareness, or even complete awareness, of their parasomnia behavior, including dream-enactment, and then have partial or full recall of the events in the morning. The "parasomniac" may-after becoming injured during the night-resort to protecting himself and others at bedtime by locking doors and windows, getting rid of furniture in the bedroom, tethering himself to bed, making barricades with pillows or cushions around the bed, etc. Often, only an astonished witness can report on the exhibited nocturnal behavior.

Sleep is commonly regarded as a quiet resting time that lacks any behavior, other than minor movements, such as coughing, mumbling, yawning, stretching, rearranging bed covers, or changing body position. However, motor dyscontrol with complex and problematic behavior can emerge during the nocturnal sleep period (1-10). Violent behavior during sleep has been reported to affect 2% of the population (1). In 1989, our center published a clinical and polysomnographic study on 100 consecutive adult patients complaining of sleep-related injury; five disorders were identified as being responsible for causing nocturnal violence: Sleepwalking/sleep terrors (SW/ST), 54%; REM sleep behavior disorder (RBD), 36%; nocturnal psychogenic dissociative disorders, 7%; nocturnal seizures, 2%; and obstructive sleep apnea/periodic limb movement disorder of NREM sleep, 1% (2). Most patients in that study sustained repeated ecchymoses, and a minority had sustained lacerations and fractures. Since this article was published, additional causes of sleep-related injury have been identified, and other types of sleep-related injury have been recognized. Furthermore, the forensic implications of sleep violence have gained increasing attention in recent years (3). We will now endeavor to provide an update, with current perspectives, more than 10 years after our initial report on sleep-related injury.

GENERAL ASSESSMENT OF MOTOR DYSCONTROL, COMPLEX BEHAVIOR, VIOLENCE, AND INJURY DURING SLEEP

As with daytime violence, nocturnal violence has also been commonly ascribed to psychiatric disorders. However, the well-established myth concerning the direct association between abnormal sleep-related behavior-particularly if violent-and major psychopathology has now been debunked by several studies which have demonstrated that such behaviors can usually be explained by the presence of an intrinsic sleep disorder (chiefly parasomnias), or by nocturnal seizures (6-9). In other words, the abnormal nocturnal behaviors are usually not a manifestation of psychopathology, but rather are a direct manifestation of an organic condition (parasomnia, nocturnal seizure).

In principle, an abnormal behavior arising from sleep may be produced by three main groups of disorders: parasomnias, seizures, and psychiatric conditions (Table 1). The clinical picture of abnormal sleep-related behaviors can be so similar in all three groups that the clinician may have great difficulty in making a well-founded, accurate diagnostic judgement--without the crucial benefit of polysomnography (PSG). However, it should be emphasized that similarity does not mean isomorphism, since despite the apparent clinical overlap, the underlying neurophysiological processes are different across the three groups just mentioned.

Table 1. Differential diagnosis of sleep-related injury

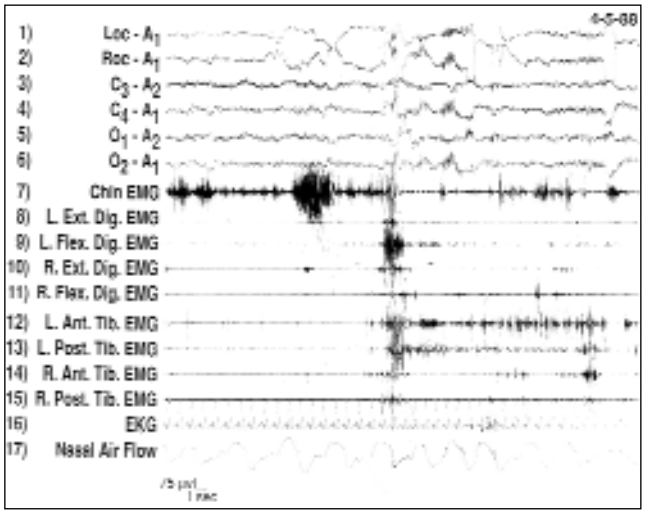
1. NEUROLOGIC
A. PARASOMNIAS
SW/ST
Sleep Drunkenness
RBD
Parasomnia Overlap Disorder
Status Dissociatus
Nocturnal Sleep-Related Eating Disorders
Bruxism
PLMD
Rhythmic Movement Disorders
Nocturnal Scratching Disorder
Obstructive Sleep Apnea
Cerebral Anoxic Attacks
B. NOCTURNAL SEIZURES
Complex Partial Seizures
Nocturnal Frontal Lobe Seizures
Paroxysmal Nocturnal Dystonia
Episodic Nocturnal Wandering
Paroxysmal Periodic Motor Attacks
C. DRUG INTOXICATION AND WITHDRAWAL STATES
2. PSYCHOGENIC
Nocturnal Dissociative Disorders
PTSD
Munchausen syndrome by Proxy
3. MALINGERING

A prime example of overlapping symptomatology across clinical disorders involves Automatism, which can emerge during the daytime and nighttime. Automatism can be a transitory or more prolonged (e.g. fugue state) complex behavior that occurs in the absence of conscious awareness or volitional intent. There is usually amnesia for the event. The automatic behavior emerges in the context of motor system activation and mental system deactivation. Various mechanisms can produce inactivation of consciousness, such as abnormal neurophysiologic activation (e.g. complex partial seizure), neuronal exhaustion in postictal states, sleep intrusion into wakefulness (e.g. narcolepsy), and psychopathologic activation (psychogenic dissociative disorders). Thus, similar and at times clinically indistinguishable behaviors can result from various different disorders, and so extensive clinical and PSG evaluations are necessary.

It is possible for similar clinical patterns to be generated by different causes, and that is precisely why PSG and video-PSG are critical in identifying the sleep or wake stage during which, or from which, the problematic behaviors arise, and the various possible precipitating circumstances, such as epileptiform activity. All-night, in-hospital (and not ambulatory), PSG monitoring (with extensive EEG and electromyographic [EMG] leads) can provide crucial neurophysiologic information on the recruitment of all the components that polygraphically define a state, and the recurrence or oscillation of such states throughout the sleep cycle (10). Also psychiatric and neurologic assessment, including psychometric tests, should be included in the protocol of the evaluation of complex and violent behavior during sleep. Thus, the combined clinical and PSG assessment of abnormal nocturnal behaviors is essential in the diagnostic and therapeutic process (11,12).

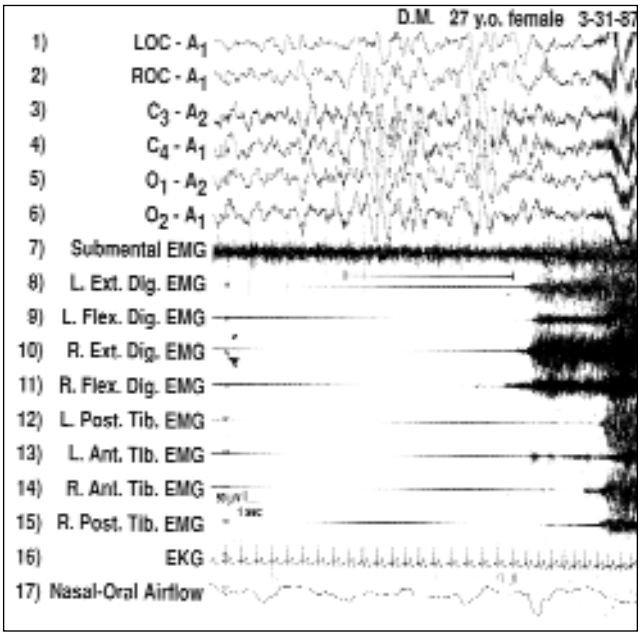
Multiple state-determining variables are recruited to occur in concert, resulting in the full declaration of a given state. Key to understanding parasomnias is the concept that sleep and wakefulness are not invariably mutually exclusive states, and that the various state-determining variables of wake, NREM sleep and REM sleep may occur simultaneously or oscillate rapidly (10). The admixture of wake and NREM sleep would explain several disorders of arousal and automatic behaviour. The tonic (i.e. continuous) and phasic (i.e. intermittent) components of REM sleep can become dissociated, intruding into, or persisting within, wakefulness, thus accounting for cataplexy, and wakeful dream-like states with automatic behavior, as found in narcolepsy. Also, the muscle tone and motor-behavioral activity of wakefulness can intrude into REM sleep, as found in REM sleep behavior disorder (RBD) (Figure 1).

Figure 1. Polysomnographic tracing demonstrating tonic and phasic motor dyscontrol with behavioral release during REM sleep in a 57 year old man with REM sleep behavior disorder (RBD). REMs are present in the electrooulogram (channels 1-2). The chin (i.e. submental) electromyogram (EMG) (channel 7) shows increased tone with prominent, phasic high-voltage twitching that subsides abruptly in the middle of the tracing, in conjunction with i) sudden onset of the normal REM atonia in the chin EMG; ii) sudden onset of abnormal EMG twitching of the extremity EMGs (channesls 8-15); and iii) behavioral emergence, with both legs jerking up, as noted by the technician. Excessive chin EMG twitching eventually reemerges in the right side of the tracing, followed by a brief return of REM-atonia before excessive twitching resumes. The EEG (channels 3-6) shows the typical low voltage, fast frequency activity of REM sleep. Despite EMG and behavioral activation, the EKG rate (channel 16) remains constant, which is typical of RBD.



Various associations of motor dyscontrol, autonomic activation and complex behavior with injuries during REM and NREM sleep have been reported in adult patients with RBD, SW-ST, Narcolepsy and Periodic Leg Movement Disorder (PLMD). Common to all these disorders is the appearence of motor activity occurring independently from waking consciousness. Dissociation between motor centres in the brainstem from the parent state of REM or NREM sleep could explain the complex motor behavior seen in RBD or SW-ST. The dissociation between behavior and conciousness may be related to sleep-related inactivation of attentional or memory systems. Therefore, as far as primary parasomnias are concerned, the underlying pathophysiologic mechanisms involve the dissociation (across a spectrum) among the components of the three basic states of being (wakefulness, REM sleep, NREM sleep), with rapid transitions also occurring among these states (Figure 2).

Figure 2. Polysomnographic tracing of an abrupt aroucal from slow wave sleep in a patient with longstanding, injurious sleepwalking (SW) and sleep terrors (ST). The EEG (channels 3-6) over the area covered by the bar reveals multi-channel, high-voltage delta ativity that immediately precedes an abrupt aroucal without behavioral activation. This EEG pattern (hypersynchronous delta) was previously considered to be a hallmark EEG finding preceding slow-wave sleep arousals in adults with SW/ST, but a recent study of 38 adults with SW/ST detected this EEG pattern in >2% of 252 slow-wave sleep arousalc (reference 16), so this pattern should now be considered to be a rare--and not a hallmark--finding. The arousal is marked by sudden, prominent EMG twitching in all EMG leads (channels 7-15), and by acceleration or EKG rate (channel 16). The electrooculogram (channels 1-2) does not reveal any conjugate eye movements.



CLINICAL CATEGORIES

1. NEUROLOGIC

A. PARASOMNIAS

Disorders of arousal. Normal sleep is frequently interrupted by intermittent brief arousals that usually produce either a transition to a lighter stage of sleep or -less frequently- a full awakening. In abnormal arousals the person seems to be trapped in a mixed state, unable to resume sleep and to awake in full. This impaired arousal process is named partial arousal (13) and probably represents an ambiguous or dissociated state as the subject displays behavior but is unaware of it. In this pathophysiological context, abnormal activation of the skeletal motor and/or autonomic nervous system emerges. The disorders of arousal comprise a spectrum ranging from confusional arousals (sleep drunkenness) to SW and ST (7,8,13). Some take the form of particular, rather "specialized" behaviors such as sleep-related eating or sleep-related sexual activity.

Sleepwalking-Sleep-terrors typically emerge, and spontaneously remit, during childhood but can persist beyond adolescence into adulthood, or may reappear in adulthood after a variable latency. In a series of 54 adults with SW-ST, 18 (33%) had their disorder of arousal begin after the age of 16 years (2). It has been estimated that 9-10% of adults may suffer from disorders of arousal and occurring weekly in 0.4% (14). Non-injurious SW has no gender preference (15) whereas injurious SW prevails in males (2,16). SW-ST are constitutional disorders, with genetic factors being relevant (14,17).

SW is characterized by complex, automatic behavior ranging from suddenly sitting up in bed with a glassy stare to automatic ambulation (13,15). During episodes, either meaningful or aany type of communication with a sleepwalker is often useless. There may be frenzied or aggressive behavior thus resulting in injury, but also quiet, calm behavior may result in serious injury as the subject may open the door and walk outside or he may go through a bedroom window (2,18). ST is characterized by abrupt onset of loud, unconsolable screaming with impressive autonomic accompaniments such as tachycardia, tachypnea, mydriasis and sweating (13,15). The subjects may sit up and engage in frenzied activity and become injured.

There is evidence strongly suggesting that the typical clear-cut distinction between SW and ST in childhood may become blurred in adulthood, as agitated SW and ambulatory ST have commonly been reported to occur-and documented during PSG studies-in the same adults (16). Moreover, the complex behaviors associated with SW-ST in adults presenting to a sleep disorders center tend to be violent. Nevertheless, active psychopathology is present in the minority of patients with active and injurious SW-ST (see below) (2,19). Although there is more often than not amnesia for the parasomnia event (15,20), vivid dream-like mentation (usually related to being threatened by imminent danger) can be experienced, with recall for the parasomnia event (2). Conversely, in childhood, there is complete amnesia of the SW-ST episodes and there is no dream recall.

SW-ST usually starts in the first third of the night but-in adults-it can occur throughout the entire sleep period. It has been documented that SW-ST episodes mostly arise during arousals from slow-wave (stages III and IV) sleep (1,13,21-23) which usually predominates in the first half of the night. PSG s may also show hyperabrupt arousals without behavior arising from slow-wave sleep. In 38 adults suffering from injurious SW-ST, three immediately post-arousal EEG patterns have beeen demonstrated: diffuse, rhythmic delta activity; diffuse delta and theta activity with some alpha-

beta activity; and prominent alpha and beta activity. In other words, the post-arousal EEG may show the persistence of sleep, the admixture of sleep and wakefulness, or complete wakefulness (16). Putative relationship of SW-ST with a "hypersynchronous delta pattern" (24) and "cyclic alternating pattern" (25) have been claimed. Otherwise, the sleep architecture is normal in SW-ST, as is the multiple sleep latency test (MSLT).

Injuries related to SW-ST range from lacerations or ecchymoses to criminal behavior including sexual abuse and even homicide or attempted homicide (26-28). The forensic implications of such behaviors are obvious (26-30). It merits mention that, on the one hand, a person involved in a criminal act during an episode of SW may qualify for legal acquittal, since SW involves a dissociated automatic state with impaired perception, cognition, judgement and memory. On the other hand, if a given person engages in a behavior known to precipate SW in that person (e.g. alcohol use or abuse, sleep deprivation, etc.), then that person should be considered legally responsible for any SW-related criminal behavior occurring in the context of willful engagement in the identified precipitating behavior (26,27,30).

Overall, many circumstances that increase slow-wave sleep, or produce arousals may ultimately trigger the SW-ST episodes. Namely, obstructive sleep apnea, periodic limb movements, nocturnal seizures, medical and neurological disorders, febrile illness, alcohol use or abuse, sleep deprivation, pregnancy, menstruation, and psychotropic medications-especially lithium carbonate and anticholinergic agents (7-10; 31-33). Also, cyclic physiologic hormonal changes can precipitate SW-ST episodes, as documented in two females suffering from premenstrual ST and injurious SW (34).

In regards to the presumed relationship of SW-ST to psychopathology (35-37), it has been demonstrated that there is no chronological linkage between psychiatric disorders and SW-ST, since neither the onset of SW-ST is related to the evolution of a given psychiatric condition nor does successful treatment and resolution of the psychopathological disorder result in improvement of SW-ST (2,36). Furthermore, psychometric data in PSG-monitored cases have not shown a consistent profile of abnormality, and in >50% of cases the profiles are unremarkable. Among all the psychiatric disorders, a past history of non-psychotic unipolar depression is most common in adults with SW-ST. (2). With these data at hand, psychopathology does not seem to play a decisive role in the development and persistence of SW-ST, although life events, and stress may act as precipitating mechanisms in predisposed persons (38).

The primary reason for obtaining PSG monitoring in the forensic setting of violent or otherwise injurious sleep-related behaviors is in ruling out causes other than the disorders of arousal. Short of capturing a SW or ST event during PSG monitoring, there are no PSG findings that will conclusively indicate that a given individual is prone to be a sleepwalker (39). The "hypersynchronous delta EEG pattern" (24), previously considered to be a marker for disorders of arousal, has recently been found to be extremely rare, and of no diagnostic relevance, in a large number of slow-wave sleep arousals in 38 adults with SW and ST (16).

Treatment is usually not necessary, and simple reassurance and sleep hygiene measures are often sufficient. However, in arousal disorders associated with either vigorous behavior and injury, or else with extremely frequent episodes, pharmacologic treatment is indicated. In such instances benzodiazepines, in particular clonazepam, provide extraordinary benefit in controlling such nocturnal behaviors. Alprazolam, diazepam, imipramine, paroxetine and carbamazepine can also be effective. It should be mentioned that longterm treatment with benzodiazepines in these conditions has proven to be safe, with no significant dosage tolerance, and with low risk (40). Self-hypnosis has been shown to be effective in milder cases of SW-ST (41), but treatment of any concurrent psychiatric disorder is mostly of no avail in controlling SW-ST (2,36).

Confusional arousals (sleep drunkenness) is a milder variant of SW-ST occurring during the transition between sleep and wakefulness and represents a disturbance of cognition and attention despite the motor behavior of wakefulness, resulting in complex behavior wihout conscious awareness (15, 42-44). This phenomenon may be precipitated by sleep deprivation, alcohol intake, sedatives or obstructive sleep apnea (OSA) (45).

REM Sleep Behavior Disorder (RBD) usually affects men above 50 years of age, but can affect either gender and can emerge at virtually any age, even in infancy. RBD is clinically characterized by the presence of complex and injurious behaviors during REM sleep, which usually represent attempted enactment of violent dreams (15).

PSG may show elevated muscle tone and/or excessive phasic submental and/or limb EMG twitching during REM sleep (46,47). In other words, there is an admixture of REM sleep and one component of wakefulness, as indicated by the typical PSG of REM sleep -and normal cycling among REM and NREM sleep stages-but with the intrusion of the muscle tone of wakefulness into REM sleep, which normally features

a protective muscle atonia (47). The nocturnal behavior is usually brief; complex and vigorous behaviors not infrequently lead to injuries involving the patient or the bed partner. Upon awakening from an episode there is ususally rapid return to alertness and the patient may report a vivid, violent dream which correlates with the displayed behavior. Of utmost clinical importance is understanding that the violence of the sleep-related behavior is usually discordant with the waking personality. This is a REM state-related disorder, so the abnormal behavior is expected to first emerge beginning approximately 90 minutes after sleep onset, at the time that REM sleep usually first appears during the nocturnal sleep cycle.

Although RBD is usually chronic, there are acute forms that are mostly induced by medication intake (tricyclic antidepressants, monoamine oxidase inhibitors, fluoxetine, venlafaxine, or selegiline) or medication withdrawal (alcohol, barbiturate, meprobamate) (48-52). Acute RBD is generally shortlived, but in the chronic form the episodes may emerge sporadically to several times nightly (52).

The chronic form of RBD is idiopathic in 25% to 60% of cases. (47,53). Virtually all females and nearly 50% of males with RBD have the onset of RBD linked with the onset of a neurological disorder, usually a degenerative condition (47,54-60) such as Parkinson s disease, olivopontocerebellar degeneration, Shy-Drager syndrome, corticobasal degeneration, Alzheimer disease, or dementia with Lewy body disease. It has been estimated that in particular 25% of patients with Parkinson s disease have behaviors suggestive of RBD or sleep-related injurious behaviors. RBD may also be the first manifestation of parkinson s disease, progressive supranuclear palsy or multisystem atrophy, and may antedate the onset of daytime symptoms in these disorders by more than 10 years (47,52,53,57,61,62). RBD may also be one of the manifestations of narcolepsy (63). Major stress events occasionally can trigger RBD (2,64). However, an association of RBD with psychopathology is quite rare.

Clonazepam is the drug of choice to treat RBD. Doses from 0.5-2 mg given at bed-time are effective. Other-far less effective-treatments include carbamazepine, melatonin, l-Dopa, imipramine. Although restoration of the normal atonia of REM sleep does not occur with treatment, suppression of excessive phasic EMG activity can occur (65). Clonazepam is very effective in controlling both the behavioral and the dream disturbances of RBD, as most RBD patients enact distinctly altered dreams that have become more vivid, action-filled, confrontative and violent.

A parasomnia overlap disorder with clinical and polysomnographic features of both RBD and SW-ST has been reported (66). In this syndrome the patients simultaneously fit the diagnostic criteria of SW-ST and RBD. This condition prevails in the males, mostly adults. Moreover, PSGs show evidence of motor dyscontrol both in REM and NREM sleep. Excessive twitching and lack of atonia in REM sleep with typical behaviors may be observed, as well as slow-wave sleep hyperabrupt arousals with/without SW-ST behavior and PLM s.

In most patients, the parasomnia overlap disorder is idiopathic, but a substantial number of patients (ca. 30%) have a symptomatic form, with various disorders being identified as emerging in tandem with the parasomnia. Such conditions included: narcolepsy, multiple sclerosis, brain tumor, brain trauma, M bius syndrome, indeterminate brain disorder (exaggerated startle response/atypical cataplexy), exclusivelly nocturnal atrial fibrillation, postraumatic stress disorder/major depression, chronic ethanol/amphetamine abuse and withdrawal, and mixed disorders (schizophrenia, brain trauma, substance abuse) (66).

A sub-group of adults with injurious SW-ST appears to also demonstrate excessive tonic and phasic motor activity during REM sleep, which can be regarded as a partial form of the "overlap SW-ST-RBD syndrome", but with the REM sleep abnormalities being restricted to the EMG, without involving behavioral release (67).

Status dissociatus represents the extreme dissociation of the three states of mammalian being: wakefulness, NREM and REM sleep. (10,68) In this condition, the polygraphic determinants of the three states may appear simultaneously or there are few, if any, features of either conventional REM or NREM sleep. Clinically, these patients exhibit a variety of vocalizations, movements and violent behavior during a state polygraphycally ambiguous (wake-like pattern), despite the patients claim of having been sleeping. The PSG mostly shows a fast desynchronized tracing with a mixture of rapid and slow eye movements as well as phasic and tonic muscle activity. Only short periods of the tracing can be scored as NREM sleep.

Conditions associated with status dissociatus include protracted withdrawal from alcohol abuse, narcolepsy, olivopontocerebellar degeneration, prior open heart surgery (68), and AIDS-related case with prominent brainstem involvement (CHS and MWM, personal observation). Similar signs and symptoms can be observed in fatal family insomnia (69,70). Clonazepam treatment may help control the abnormal motor activity (locomotor and verbal behavior) and may provide an increase in conventional sleep.

Nocturnal eating disorders can arise from any sleep stage with variable dissociation of conciousness

from the eating behavior, and is most frequently associated to SW but also with PLMD, restless legs syndrome, and other disorders (31,32,71). One third of these patients suffer injurries from their confused and impulsive cooking, food preparation and eating behavior. Injuries included scalding from consumig hot foods or liquids, and lacerations from collisions with furniture, doors or walls in the kitchen (31).

Rythmic movement disorders (RMD) include sleep-related stereotypic, repetitive movements of the head, neck, or large muscle groups, occurring with a frequency of 0.5-2 HZ. (hence rhythmic) which can persist from a few minutes to many hours, and may occur unpredictibly, even almost nightly. These disorders include headbanging (jactatio capitis nocturna), headrolling, and bodyrocking-even bodyrolling, legbanging, and legrolling (15). RMD occurs most frequently in infants, but adult persistence is not uncommon, and has unknown etiology, and may be familial in some cases. Rarely, late-onset forms have also been described (72).

Most children with RMD usually have no associated significant emotional or behavioral problems (73). However, it has been reported that older children and adolescents may have psychopathological problems (74). Injuries have been reported as often occuring in patients with associated severe mental retardation and psychopathology: soft tissue, eye, and skull injuries, tongue biting, hemoglobinuria with acute renal failure, internal carotid dissection and subdural haemorrhage (75-84) all have been regarded as a consequence of RMD. Although RMD may occur during all stages of sleep and wake-sleep transitions, they seem to appear most frequently during stage 2 sleep (85).

RMD are generally benign and spontaneously resolve with time. No effective pharmacologic or psychologic treatment has been found. Benzodiazepines and tricyclic antidepressant agents have been tried (86). Isolated cases have been reported as being responsive to imipramine (87) or hypnosis (88), or water bed therapy (89).

Periodic limb movements disorder (PLMD) is characterized by periodic contractions of the dorsiflexors of the foot occuring every 15-40 seconds (15). Associated flexion of the knee and hip may also occur. PLMD is often associated with arousals giving rise to disrupted sleep. Therefore, fatigue and excessive daytime sleepines are the chief complaints of these patients. However, PLMD is prevalent in other sleep disorders that have been involved in sleep injury such as RBD (47), Narcolepsy (63), OSA (15), nocturnal eating disorders (31,32), and nocturnal scratching (90). Otherwise, PLMD can be idiopathic although it can also be associated with restless legs syndrome, chronic uremia and other metabolic conditions (15). Tricyclic antidepressants and monoamine oxidase inhibitors and withdrawal from other drugs active on the CNS can also induce or aggravate the PLMD.

The treatment of choice for symptomatic PLMD is L-Dopa/carbidopa given at bedtime. Other effective drugs include clonazepam, codeine, and other dopaminergic agents.

Nocturnal scratching disorder. It is known that itching (pruritus) is a conscious sensation, but it has been documented that scratching-which is the behavioral response to the itching sensation-can occur during sleep (90-94). At least some patients have been found to repetitively scratch themselves during sleep, including perianal scratching. Scratching may even produce bleeding and deep severe skin ulcers (90) up to the point of exposing tendons. Nocturnal scratching may be predominant in stages 1 and 2 sleep, and is less frequently observed in REM and stages 3 and 4 sleep. This disorder may reflect a decreased threshold for nocturnal pruritus to trigger the scratching response, or it may reflect a dissociated and disinhibited scratching response in the absence of any itching perception (i.e. pruritus) during sleep. In some way, nocturnal scrtaching disorder parallels what is known in other rhythmic disorders (see above). Sleep of itchy patients tend to be superficial and disrupted (90). Whether scratching is related to an underlying dermatologic process, or is more likely related to the physiology of sleep itself, remains controversial (93). Even psychiatric factors being primarily responsible for such nocturnal behavior have been proposed (94). However, it is doubtful that psychogenic pruritus persists during sleep (94-96).

Bruxism is a stereotyped movement disorder occurring, during partial arousals from sleep, that is characterized by unconscious intermittent tight jaw closure with stereotyped rhythmic grinding of the teeth during all stages of sleep, mostly stage 2 sleep, without discernible EEG abnormalities (97). This parasomnia may result in damage to the teeth, to the periodontal structures, and to the temporomandibular joints (98). Although it may occur at any age, the disorder clearly prevails in children (99). A genetic tendency is noticeable (100) Recent studies indicate that bruxism may represent a manifestation of different disorders such as RBD (101), orofacial dyskinesia, mandibular dystonia and tremor (102). There is no definite, effective treatment, although promising results have been reported with local (on the lips) electrical stimulation techniques for com-

mon bruxism, and local injections of botulinum toxin (102) for dyskinetic bruxism. Other attempted treatments have included psychotherapy, hypnotherapy, physical therapy, muscle relaxants, and non steroidal antiinflammatory drugs (NSAIDs), all with a variable, generally poor response. It is noteworthy that malocclusion seems to play no role in bruxism, and therefore, there is no clear indication for malocclusion therapy in bruxism (103)

Obstructive sleep apnea syndrome (OSA), as already mentioned, can produce confusional arousals leading to automatic, complex or violent behavior (104). It is also possible that OSA induced arousals from REM sleep with dream-related complex or violent behaviors can result in injury (105) that is reminiscent of what is encountered in RBD (pseudo-RBD). NREM or REM sleep arousals in OSA may be associated with nocturnal eating disorder (31,32) Otherwise, nasal continuous positive airway pressure" (n-CPAP) treatment may produce a slow wave sleep rebound and emergent SW (106) or ST (107).

The consequences of OSA, such as desaturation and sleep deprivation, have been claimed to account for OSA induced epileptic seizures (108-110) with complex or violent behaviors.

Cerebral anoxic attacks produced by severe hypoxia caused by prolonged apneas in REM sleep have been described in a patient suffering from OSA syndrome (111). In this reported patient, anoxic attacks consisted of brief, violent leg and arm spasms. Finally, since agitation has been noted to occur after transitory cardiac arrest during wakefulness, it is possible that nocturnal, sleep-related agitation can emerge after nocturnal cardiac arrest.

B. NOCTURNAL SEIZURES

Nocturnal seizures originating in the orbital, mesial, or prefrontal regions may result in bizarre behaviors, aggression, violence and nocturnal wanderings. In the last decade there have been several descriptions on abnormal complex behaviors arising mostly from NREM sleep, such as episodic nocturnal wanderings (112-116), nocturnal (hypnogenic) paroxysmal dystonia (116-119), paroxysmal (arousals) awakenings, (120,121) and paroxysmal periodic motor attacks (122). The descriptions of all these "atypical" nocturnal phenomena are so similar that these reports probably refer to variations of the same syndrome. Increasing evidence on the epileptic nature of all these reported behaviors have finally clustered all under a nocturnal familial form of frontal lobe epilepsy: Autosomal dominant nocturnal frontal lobe epilepsy (ADNFLE) (123,124). The clinical spectrum of ADNFLE includes sudden awakenings with dystonic or dyskinetic movements, complex behaviors, and sleep-related violent behavior (123-126) that can be succesfully treated with antiepileptic drugs (mostly carbamazepine). Therapy is also effective to controlling daytime symptoms, such as seizures and hypersomnia (related to sleep disruption by excessive epileptic arousals). Extensive video-polysomnographical studies will generally show EEG epileptiform abnormalities over frontal areas.

Nocturnal complex partial seizures of the temporal lobe epilepsy are much less common than frontal lobe epilepsy. Somnambulism-like episodes, fear or panic, autoagressive behavior, and postictal nocturnal aggresiveness may be features of such epilepsy (127,128).

Postictal states of generalized epilepsy may result in epileptic fuga and prolonged automatic - putatively violent-behavior (poriomania) (129). Also, vigorous or violent behavior can emerge in the postictal state of either partial or generalized seizures (postictal psychosis) (130,131). It should be mentioned that during paranoid or schizophrenia-like psychotic post-ictal episodes, the EEG does not show seizure activity (131).

2. PSYCHOGENIC

Dissociative Disorders can occur during the nocturnal sleep period, with the typical EEG correlate consisting of a wakeful pattern (fast frequency, desynchronized, low voltage) lasting for 30-90 seconds while the patient appears to be behaviorally asleep, before the complex behaviors emerge. In other words, the affected person falls asleep and cycles through various sleep stages before there is an EEG awakening, and then shortly thereafter-but not immediately thereafter (as is seen with SW, ST, RBD)-there is behavioral activation (7,10,132,133). Documented injuries (132) include self-mutilation behaviors, such as genital cutting, self-burning, and punching through windows. Acting like a large jungle cat has also been reported in a male patient with exclusively nocturnal dissociative disorder. Sleep may facilitate the transition from one personality to the other, and two or more personalities may interact in the oneiric state. Transitions from one personality to another during sleep and the sudden shifts of states that induce the dissociative behavior, suggest a parasomnia phenomenon.

Nocturnal dissociative episodes may manifest as

either protracted wanderings or as attempted renactement of past physical and sexual abuse (132), with agitated and sexualized behaviors that occur during perceived "dreaming". These "dreams" are actually forms of dissociated wakeful mentation apparently related to past assaults (132). The patients usually have amnesia for the abnormal nocturnal episodes, which can resemble SW to an observor (132,133), thus reinforcing the previous statement that PSG is necessary to establish the diagnosis, in conjunction with a thorough clinical evaluation. Two crucial differences between nocturnal dissociative disorders and SW/ST (i.e. disorders of arousal) are the presence of diurnal dissociative episodes in most patients with the first disorder, and also the presence of established EEG wakefulness prior to nocturnal behavioral activation.

It should be noted that bedtime administration of benzodiazepine may aggravate a nocturnal dissociative disorder. Management of these conditions by experienced specialists is strongly recommended.

Posttraumatic stress disorder (PTSD). After a traumatic event beyond the normal limits of human experience some persons may develop PTSD. In such a case the clinical picture includes: intrusive thoughts (flash-back) that relive tragic past experience; living in isolation; reduced capacity to respond to environmental demands ("psychic anesthesia"), and a variety of autonomic, dysphoric and cognitive signs (134). In regards to sleep, these patients may experience Dissociative States with injury, and also injurious behaviors directly related to intense nightmares (135-136). Otherwise, the typical hypervigilance of these patients may produce insomnia.

The Limbic Psychotic Trigger Reaction in which motiveless unplanned homicidal acts occur is speculated to represent partial limbic seizures that are kindled by highly individualized and specific trigger stimuli, reviving past repetitive stress (137).

Munchausen syndrome by proxy involving children, may present as injurious sleep-related behaviors (138-146). These children are brought to a sleep center with histories of unusual nocturnal spells or "seizures", along with other complaints, such as sleep apnea and excessive daytime sleepiness. In fact, these problems originated with an adult, usually a caregiver, and often a parent. Secret video monitoring in a sleep laboratory during PSG-documented sleep, with the adult caregiver present, will usually provide the necessary documentation of the true cause for the reported problematic nocturnal behaviors (12). Child protection services should then be notified and psychiatric evaluation of the offending adult should also be initiated.

MALINGERING

Malingering, which is not a psychiatric disorder, involves conscious, willful behavior intended to mimic actual medical signs and symptoms for the purpose of achieving a specific gain. A case of sleep-related violence has been reported in which malingering was ultimately-after extensive clinical and PSG evaluations - considered to be the most likely explanation for a man repeatedly beating his wife at night (12).

FORENSIC IMPLICATIONS

In any desired and intentional behavior, the mind compares and weighs the desire to initiate a given act, and the moral duty to avoid that act if it is socially objectionable. Therefore, intra-psychic tensions can arise among what is desired, permitted, and prohibited. Consequently, the mission of a forensic expert involved in legal proceedings pertaining to a criminal act is precisely to decide on whether the central nervous system of the accused allows for appropriate judgement and volitional activity. In other words, does the state of brain and mind allow for proper determination of right from wrong in regards to interpersonal behavior?

Responsibility refers to the minimal mental conditions that makes a person both material and spiritual author of the act. The law is charged to consider whether a given action or act is psychically and ethically caused by the accused person. From the point of view of psychology, the crucial conditions are: a) that in the moment of the behavior the accused must have the intelligence and discernment of his acts; b) the subject must have free will (and not subject to automatism, etc.).

It has been demonstrated that complex, violent acts can arise from sleep without concious awareness, and therefore, without responsibility (Table 2).

Table 2. Catalog of injuries and violence during sleep

Ecchymoses
Abrasions
Lacerations
Fractures
Skin Ulcers
Burns
Scalding from hot foods
Choking on food
Subdural heamatomas,
Internal carotid artery dissection
Cranial trauma
Self mutilation
Sexual abuse
Assaults on others
Unexplained death, suicide mimic
Homicide, Filicide

A number of parasomnias, such as nocturnal panic disorder, are not included because they are not associated with injurious behavior. However, it is conceivable that these other parasomnias could eventually be included in this table if they induce a secondary frenzied or otherwise injurious behavior.

In other words, the subject is displaying an automatic behavior (146). Once it has been established that a given person is not reponsible for the behavior, a question remains to be answered. Should the law protect the subject and relatives from possible consequences of such a nocturnal behavior? In this context, it is worth mention that sleep related violence is a state dependent behavior. Thorough evaluation and effective treatment are mandatory before the patient can be regarded as no longer being a menace to society (27). In some cases, clear precipitating events can be identified and must be avoided to be exonerated from legal culpability (27). Thus, the court must be informed that recurrence of the criminal behavior is unlikely and that succesful treatment and prevention are possible in most cases.

CONCLUSION

Parasomnias comprise a wide array of injurious sleep behavior disorders that can usually be diagnosed with the discriminatory power of polysomnography-in conjunction with careful clinical evaluations-and can usually be satisfactorily and safely treated. Parasomnias are an uncommon manifestation of psychopathology, and are usually a manifestation of sleep-related physiologic dysfunction. Finally, knowledge on the parasomnias interlinks important findings from basic animal research with human clinical "experiments of nature".

REFERENCES

1. *Ohayon MM, Malija C, Priets RG. Violent behavior during sleep. J Clin Psychiatry 1997;58:369-376.*
2. *Schenck CH, Milner DM, Hurwitz TD, Bundlie SR, Mahowald MW. A polysomnographic and clinical report on sleep-related injury in 100 adult patients. Am J Psychiatry 1989;146:1166-1173.*
3. *Mahowald MW, Schenck CH. Sleep-related violence and forensic medicine issures. In: Chokreverty S, ed. Sleep Disorders Medicine: Basic Science, Technical Considerations and Clinical Aspects (2nd edn), Butterworth Heineman. Boston. 1999;729-739.*
4. *Moldofsky H, Gilbert R, Lue FA, MacLean AW. Sleep-related violence. Sleep 1995;18:731-739.*
5. *Raschka LB. Sleep and violence. Can J Psychiatry 1984;29:132-134.*
6. *Mahowald MW. Schenck CH. NREM sleep parasomnias. Neurologic clinics; Vol 14; 1996; 675-696.*
7. *Mahowald MW, Ettinger MG. Things that go bump in the night — the parasomnias revisited. J Clin Neurophysiol 1990;7:119-143.*
8. *Mahowald MW, Rosen GM. Parasomnias in children. Pediatrician 1990;17:21-*
9. *Mahowald MW, Schenck CH. Parasomnia purgatory. The epileptic/non epileptic interface. In: Rowan AJ, Gates JR, eds. Non-epileptic seizures. Boston. Butterworth-Heineman 1993;123-139.*
10. *Mahowald MW, Schenck CH. Dissociated states of wakefulness and sleep. Neurology 1992;42 (Suppl 6):44-52*
11. *Schenck CH, Mahowald MW. Injurious sleep behavior disorders (parasomnias) affecting patients on intensive care units. Intensive Care Med 1991;219-224.*
12. *Mahowald MW, Schenck CH, Rosen GM, Hurwitz TD. The role of a sleep disorder center in evaluating sleep violence. Arch Neurol 1992;49:604-607*
13. *Broughton RJ. Sleep disorders: disorders of arousal? Science 1968; 159:1070-1078.*
14. *Hublin C, Kaprio J, Partinen M, et al. Prevalence and genetics of sleepwalking. A population-based twin study. Neurology 1997; 48:177-181*
15. *Thorpy MJ, Chairman. Diagnostic Classification Steering Committee. ICSD. International Classification of Sleep Disorders: Diagnostic and coding manual. Rochester, MN, American Sleep Disorders Association, 1990.*
16. *Schenck CH, Pareja JA, Patterson AL, Mahowald MW. Analysis of polysomnographic events surrounding 252 slow-wave sleep arousals in thirty-eight adults with injurious sleepwalking and sleep terrors. J Clin Neurophysiol 1998;15:159-156.*
17. *Crisp AH. The sleepwalking/night terrors syndrome in adults. Postgraduate Medical Journal. 1996;72:599-604.*
18. *Millier N, Ummenhofer W. Somnambulism and trauma: case report and short review of the literature. The Journal of Trauma: Injury, Infection and Critical Care 1999;4:420-422.*
19. *Hartmann E, Greenwald D, Brune P. Night terrors and sleepwalking. Sleep Res 1982;11:121.*
20. *Fisher C, Kahn E, Edwards A, et al. A psychophysiological logical study of nightmares and night terrors. III. Mental content and recall of stage 4 night terrors. J Nerv Ment Dis 1974;158:174-188.*
21. *Gastaut H, Broughton R. A clinical and polygraphic study of episodic phenomena during sleep. Recent adavances in Biological Psychiatry 1965;7:197-222.*
22. *Kales A, Jacobson A, Paulson J, Kales JK, Walter R. Somnambulism: psychophysiological correlates. I. All-night EEG studies. Archives of General Psychiatry 1966;14:586-594.*
23. *Fisher C, Kahn E, Edwards A, Davis D. A psychophysiological study of nightmares and night terrors. I physiological aspects of the stage 4 night terror. Journal of Nervous and Mental Disease 1973; 157:75-98.*
24. *Blatt I, Peled R, Gadoth N, Lavie P. The value of sleep recording in evaluating somnambulism in young adults. Electroencephalogr Clin Neurophyhsiol 1991;78:407-412.*

25. *Zucconi M, Oldani A, Ferrini-Strambi L, Smirne S. Arousal fluctuations in non-rapid eye movements parasomnia: the role of cycling alternating pattern as a measure of sleep instability. J Clin Neurophysiol 1995;12:147-154.*

26. *Schenck CH, Mahowald MW. A Polysomnographic documented case of adult somnambulism with long-distance automobile driving and frequent nocturnal violence: parasomnia with continuing danger as a noninsane automatism?. Sleep 1995;765-772.*

27. *Schenck CH, Mahowald MW. An analysis of a recent criminal trial involving sexual misconduct with a child, alcohol abuse and a succesful sleepwalking defence: arguments supporting two proposed new forensic categories. Med Sc Law 1998;38:147-152.*

28. *Broughton R, Billings R, Cartwright R, Doucette D, Edmeads J, Edwardh M, Ervin F, Orchard B, Hill R, Turrell G. Homicidal somnambulism: a case report. Sleep 1994;17:253-264.*

29. *Kavey NB, Whyte AB, Resor Jr. SR, Gidro-Frank S. Somnambulism in adults. Neurology 1990; 40:749-752.*

30. *Mahowald MW, Schenck CH. Medical-legal aspects of sleep medicine. In: Neurologic Clinics. Medical-legal issues facing neurologists. Vol 17; number 2;1999;215-233.*

31. *Schenck CH, Hurwitz TD, Bundlie SR, Mahowald MW. Sleep-related eating disorders: polysomnographic correlates of a heterogeneous syndrome distinct from daytime eating disorders. Sleep 1991;14:419-431.*

32. *Schenck CH, Hurwitz TD, O Connor KA, Mahowald MW. Additional categories of sleep-related eating disorders and the current status of treatment. Sleep 1993;16:457-466.*

33. *Mahowald MW, Schenck CH. Parasomnias including the restless legs syndrome. Clinics in Chest Medicine 1998;19:183-202.*

34. *Schenck CH, Mahowald MW. Two cases of premenstrual sleep terrors and injurious sleep-walking. J Psychosom Obstet Gynecol 1995;16:79-84.*

35. *Hublin C, Kaprio J, Partinen M, Koskenvuo M. Limits of self-report in assessing sleep terrors in a population survey. Sleep 1999; 22:89-93.*

36. *Llorente MD, Currier MB, Norman SE, Mellman TA. Night terrors in adults: phenomenology and relationship to psychopathology. J Clin Psychiatry 1992;53:392-394.*

37. *Kales JD, Kales A, Soldatos CR, et al. Night terrors: clinical characteristics and personality patterns. Arch Gen Psychiatry 1980; 37:1413-1417.*

38. *Schenck CH, Mahowald MW. On the reported association of psychopathology with sleep terrors in adults. Sleep (in press).*

39. *Mahowald MW, Schenck CH. Parasomnias and the law. Sleep Medicine Reviews. (In press).*

40. *Schenck CH, Mahowald MW. Long-term, nightly benzodiazepine treatment of injurious parasomnias and other disorders of disrupted nocturnal sleep in 170 adults. American Journal of Medicine 1996;100:333-337.*

41. *Hurwitz TD, Mahowald, MW, Schenck CH, Schlutter JL, Bundlie SR. A retrospective outcome study and review of hypnosis as treatment of adults with sleepwalking and sleep terror. Journal of Nervous and Mental Disease 1991;179:228-233.*

42. *Guilleminault C, Moscovitch A, Leger D. Forensic sleep medicine. Nocturnal wandering and violence. Sleep 1995;18:740-748.*

43. *Roth B, Nevsimalova S, Sagova V, et al. Neurological, psychological and polygraphic findings in sleep drunkenness. Arch Suisses Neurol, Neurochir, Psychiatr 1981;129:209-222.*

44. *Roth B, Nevsimalova S, Rechtschaffen A. Hypersomnia with "sleep-drunkenness". Arch Gen Psychiatry 1972;26:456-462.*

45. *Pressman MR, Meyer TJ, Kendrick-Mohammed J, Figueroa WG, Greenspon LW, Peterson DD. Night terrors in an adult precipitated by sleep apnea. Sleep 1995;18:757-64.*

46. *Schenck CH, Bundlie SR, Ettinger MG, et al. Chronic behavioral disorders of human REM sleep: a new category of parasomnia. Sleep 1986;9:293-308.*

47. *Schenck CH, Hurwitz TD, Mahowald MW. REM sleep behavior disorder: an update on a series of 96 consecutive cases and a review of the literature. Journal Sleep Research 1993;2:224-231.*

48. *Louden MB, Morehead MA, Schmidt HS. Activation by selegiline (Eldepryle) of REM sleep behavior disorder in parkinsonism. West Virginia Medical Journal 1995;91:101.*

49. *Schenck CH, Mahowald MW, Kim SW, et al. Prominent eye movements during NREM sleep and REM sleep behavior disorder associated with fluoxetine treatment of depression and obsessive-compulsive disorder. Sleep 1992;15:226-235.*

50. *Schutte S, Doghramji K. REM behavior disorder seen with venlafaxine (Effexor). Sleep Research 1996;25:364.*

51. *Silber MH. REM sleep behavior disorder associated with barbiturate withdrawal. Sleep Research 1996;25:371.*

52. *Mahowald MW, Schenck CH. REM sleep behavior disorder. In: Kryger MH, Dement W, Roth T (eds). Principles and Practice of Sleep Medicine, Second Edition. Philadelphia: W.B. Saunders Company 1994;574-578.*

53. *Sforza E, Krieger J, Petiau C. REM sleep behavior disorder: clinical and physiopathological findings. Sleep Medicine Reviews 1997; 1:57-69.*

54. *Schenck CH, Hurwitz TD, Mahowald MW. A report of 7 cases females with the REM sleep behavior disorder (RBD). Sleep Res 1990;19:282.*

55. *Boeve BF, Silber MH, Petersen RC, et al. REM sleep behavior disorder and degenerative dementia with or without Parkinsonism: a syndrome predictive of Lewy body disease. Neurology 1998; 49:a358-359 (abstract).*

56. *Kimura K, Tachibana N, Aso T, et al. Subclinical REM sleep behavior disorder in a patient with corticobasal degeneration. Sleep 1997;20:891-894.*

57. *Schenck CH, Bundlie SR, Mahowald MW. Delayed emergence of a parkinsonian disorder in 38% of 29 older men initially diagnosed with idiopathic rapid eye movement sleep behavior disorder. Neurology 1996;46:388-393.*

58. *Tachibana N, Kimura K, Kitajima K, et al. Rem sleep without atonia at early stage of sporadic olivopontocerebellar atrophy. J Neurol Sc 1995;132:28-34.*

59. *Tison F, Wenning GK, Quinn NP, et al. REM sleep behavior disorder as the presenting symptom of multiple system atrophy. J Neurol Neurosurg Psychiatry 1995;58:379-380.*

60. *Turner RS, Chervin RD, Frey KA, et al. Probable Lewy body disease in a patient with REM sleep behavior disorder. Neurology 1995;45:709-712.*

61. *Montplaisir J, Petit D, Decary A., et al. Sleep and quantitative EEG in patients with progressive supranuclear palsy. Neurology 1997;49:999-1003*

62. *Pareja JA, Caminero AB, Masa JF, et al. A first case of progressive supranuclear palsy and pre-clinical REM sleep behavior disorder presenting as inhibition of speech during wakefulness and somniloquy with phasic muscle twitching during REM sleep. Neurolog a 1996;11:304-306.*

63. *Schenck CH, Mahowald MW. Motor dyscontrol in narcolepsy: rapid-eye-movement (REM) sleep without atonia and REM sleep behavior disorder. Annals Neurology 1992;32:3-10.*

64. *Schenck CH, Mahowald MW. REM sleep parasomnias. Neurologic Clinics 1996;14:697-720*

65. *Lapierre O, Montplaisir J. Polysomnographic features of REM sleep behavior disorder: development of a scoring method. Neurology 1992;42:1371-1374.*

66. *Schenck CH, Boyd JL, Mahowald MW. A parasomnia overlap disorder involving sleepwalking, sleep terrors, and REM sleep behavior disorder in 33 polysomnographycally confirmed cases. Sleep 1997;20:972-981.*

67. *Vachatimanont P, Pareja JA, Mahowald M, Schenck CH. Tonic and phasic electromyographic activity during REM sleep in adults with injurious sleepwalking and complex behaviors arising from slow-wave sleep. Sleep Research 1994; 23:337.*

68. *Mahowald MW, Schenck CH. Status dissociatus — a perspective on states of being. Sleep 1991;14:69-79.*

69. *Medori R, Tritschler H-J, LeBlanc A, et al. Fatal familial insomnia, a prion disease with a mutation at codon 178 of the prion protein gene. N Engl J Med 1992;326:444-449.*

70. *Tinuper P, Montagna P, Medori R. The thalamus participates in the regulation of the sleep-waking cycle: A clinico-pathological study in fatal familial thalamic degeneration. Electroencephalogr Clin Neurophysiol 1989;73:117-123.*

71. *Menkes DB. Triazolam-induced nocturnal bingeing with amnesia. Australian and New Zealand Journal of Psychiatry 1992;26:320-321.*

72. *Bramble D. Two cases of severe head-banging parasomnias in peripuberal males resulting from otitis media in toddlerhood. Child: care, health and development 1995;21:247-253.*

73. *Mitchell R, Etches P. Rhythmic habit patterns (stereotypes). Developmental Medicine and Child Neurology 1977;19:545-550.*

74. *Thorpy M, Spielman A. Persistent jactatio nocturna. Neurology 1984;34:208-209.*

75. *Brody S. Self-rocking in infancy. J Am Psychoanal Assoc 1960; 8:464-491.*

76. *Bemporad JR. Cataracts following head banging: a report of two cases. Am J Psychiatry 1968;125:245-249.*

77. *Stuck KJ, Hern ndez RJ. Large skull defect in a head banger. Pediatr Radiol 1979;8:257-258.*

78. *Vasiknanonte P, Kuasirkul S, Vasiknanonte S. Two faces of nocturnal tongue biting. J Med Assoc Thai 1997;80:500-506.*

79. *Robertson MM, Trimble MR, Less AJ. Self-injurious behavior and the Gilles de la Tourette syndrome: A clinical study and review of the literature. Psychol Med 1989;19:611-625.*

80. *Spalter HF, Bemporad JR, Sours JA. Cataracts following chronic headbanging. Arch Ophthalmol 1970;83:182-186.*

81. *Blaser S, Macknin ML. Head-banging with subsequent hemoglobinuria and acute renal failure. Cleve Clin Q 1983;50:347-350.*

82. *Sormann GW. The headbangers tumour. Br J Plast Surg 1982; 35:72-74.*

83. *Jackson MA, Hughes RC, Ward SP. "Headbanging" and carotid dissection (Abstract) Br Med J 1983; 287:1262.*

84. *Mackenzie JM. "Headbanging" and fatal subdural haemorrhage. Lancet 1991;338:1457.*

85. *Dyken ME, Lin-Dyken DC, Yamada T. Diagnosing rhythmic movement disorder with video-polysomnography. Pediatr Neurol 1997;16:37-41.*

86. *Thorpy MJ, Glovinsky PB. Parasomnias. Psychiatr Clin North Am 1985;69:1289-1315.*

87. *Drake MEJ. Jactatio nocturna after head injury. Neurology 1986; 36:867-868.*

88. *Rosenberg C. Elimination of a rhythmic movement disorder with hypnosis: A case report. Sleep 1995;18:608-609.*

89. *Garc a J. Rosen G, Mahowald M. Waterbeds in treatment of rhythmic movement disorders:experience with two cases. Sleep Research 1996;25:243.*

90. *Brodland DG, Staats BA, Peters MS. Factitial leg ulcers associated with an unusual sleep disorder. Arch Dermatol 1989;125:1115-1118.*

91. *Savin JA, Paterson WD, Oswald I. Scratching during sleep. Lancet 1973;296-297.*

92. *Aoki T, Kushimoto H, Hishikawa Y, Savin JA. Nocturnal scratching and its relationship to the disturbed sleep of itchy subjects. Clin Exp Dermat 1991;16:268-272.*

93. *Savin JA, Paterson WD, Oswald I, Adam K. Further studies of scrtaching during sleep. Br J Dermatol 1975;93:297-302.*

94. *Gupta MA, Gupta AK, Kirkby S, Schork NJ, Weiner HK, Eellis CN, Voorhees JJ. Pruritus associated with nocturnal wakenings: organic or psychogenic? J Am Acad Dermatol 1989;21:479-484.*

95. *Bernhard JD. Nocturnal wakening caused by pruritus: organic or psychogenic? Amer Acad Dermatol 1990;723-767.*

96. Savin, Adam K, Oswald I, Paterson WD. Pruritus and nocturnal wakenings. Amer Acad Dermatol 1990;23:767-768.

97. Satoh T, Harada Y. Electrophysiological study on tooth-grinding during sleep. Electroencephalography Clin Neurophysiol 1973; 35:267-275.

98. Ware JC, Rugh JD. Destructive bruxism: sleep stage relationship. Sleep 1988;11:172-181.

99. Attanasio R. Nocturnal bruxism and its clinical management. Dent Clin North Am 1991;35:245-252.

100. Abe K, Shimakawa M. Genetic and developmental aspects of sleeptalking and teethgrinding. Acta Paedopsychiatrica 1966; 33:339-344.

101. Tachibana N, Yamanaka K, et al. Sleep bruxism as a manifestation of subclinical rapid eye movement sleep behavior disorder. Sleep 1994;17:555-558.

102. Clarke GT, Koyano K, et al. Oral motor disorders in humans. California Dental Association Journal 1993;21:19-30.

103. Vanderas A. Relationship between malocclusion and bruxism in children and adolescents: A review. Pediatr Dent 1995;17:7-12.

104. Guilleminault C, Silvestri R. Disorders of arousal and epilepsy during sleep. In Sterman MB, Shouse MN, Passouant PP, eds. Sleep and Epilepsy. New York, Academic Press 1982;513-531.

105. Nalamalapu U, Goldberg R, DePhillipo M, et al. Behaviors simulating REM behavior disorder in patients with severe obstructive sleep apnea. Sleep Research 1996; 311.

106. Millman RP, Kipp GR, Carskadon MA. Sleep-walking precipitated by treatment of sleep apnea with nasal CPAP. Chest 1991;99:750-751.

107. Pressman MR, Meyer TJ, Kendrick-Mohamed J, et al. Night terrors in an adult precipitated by sleep apnea. Sleep 1995;18:773-775.

108. Guilleminault C. Natural history, cardiac impact and long-term follow-up of sleep apnea syndrome. In Guilleminault C, Lugaresi E, eds. Sleep/Wake Disorders: Natural history, epidemiology, and long-term evolution. New York, Raven Press, 1983;107-125.

109. Houdart R, Mamo H, Tomkiewicz H. La forme epileptogene du syndrome de Pickwick. Revue Neurologique 1960;103:466-468.

110. Kryger M, Quesney LF, Holder D, et al. The sleep deprivation syndrome of the obese patient. Am J Med 1974;56:531-539.

111. Cirignotta F, Zucconi M, Mondini S, et al. Cerebral anoxic attacks in sleep apnea syndrome. Sleep 1989;12:400-404.

112. Pedley TA, Guilleminault C. Episodic nocturnal wanderings responsive to anticonvulsant drug therapy. Ann Neurol 1977;2:30-35.

113. Guilleminault C, Leger D, Phillip P, Ohayon MM. Nocturnal wandering and violence: review of a sleep clinic population. J Forensic Sci 1998;43:158-163.

114. Hijang Y-Z, Chu N-S. Episodic nocturnal wandering and complex hallucination. A case with long-term follow-up. Seizure 1998; 7:67-71.

115. Maselli RA, Rosenberg RS, Spire JP. Episodic nocturnal wanderins in non-epileptic young patients. Sleep 1988;11:156-161.

116. Montagna P. Nocturnal paroxysmal dystonia and nocturnal wandering. Neurology 1992;42(Suppl 6):61-67.

117. Maccario M, Paroxysmal nocturnal dystonia presenting as excessive daytime somnolence. Arch Neurol 1990;47:291-294.

118. Lugaresi E, Cirignotta F. Hynogenic paroxysmal dystonia: epileptic seizure or a new syndrome? Sleep 1981;4:129-138.

119. Lugaresi E, Cirignotta F, Montagna P. Nocturnal paroxysmal dystonia. J Neurol Neurosurg Psychiatry 1988;49:375-380.

120. Peled R, Lavie P. Paroxysmal awakenings from sleep associated with excessive daytime somnolence. A form of nocturnal epilepsy. Neurology 1986;36:95-98.

121. Montagna P. Sforza E, Tinuper P, Cirignotta F, Lugaresi E. Paroxysmal arousals during sleep. Neurology 1990;40:1063-1066.

122. Sforza E, Montagna P, Rinaldi R, Tinuper P, Cerullo A, Cirignotta F, et al. Paroxysmal periodic motor attacks during sleep: clinical and polygraphic features. Electroencephalogr Clin Neurophysiol 1993;86:161-166.

123. Scheffer IE, Bhatia KP, Lopes-Cendes I, Fish DR, Marsden CD, Andermann F, et al. Autosomal dominant nocturnal frontal lobe epilepsy: a distinctive clinical disorder. Brain 1995;118:61-73.

124. Scheffer IE, Bhatia KP, Lopes-Cendes I, Fish DR, Marsden CD, Andermann F, et al. Autosomal dominant frontal lobe epilepsy misdiagnosed as sleep disorder. Lancet 1996;347:1191-1192.

125. Oldani A, Zucconi M, Ferini-Strambi L, Bizzozero D, Smirne S. Autosomal dominant nocturnal frontal lobe epilepsy: electroclinical picture. Epilepsia 1996;37:964-976.

126. Oldani A, Zucconi M, Asselta R, Modugno M, Bonati MT, Dalpr L, Malcovati M, Tenchini ML, Smirne S, Ferini-Strambi L. Autosomal dominant nocturnal frontal lobe epilepsy. A video-polysomnographic and genetic appraisal of 40 patients and delineation of the epileptic syndrome. Brain 1998;121:205-223.

127. Bernasconi A, Andermann F, Cendes F, Dubeau F, Andermann E, Olivier A. Nocturnal temporal lobe epilepsy. Neurology 1998; 50:1772-1777.

128. Silvestri R, De Domenico P, Musolino R, Mento G, Marabello L, Longo M, Di Perri R. Nocturnal complex partial seizures precipitated by REM sleep: a case report. Eur Neurol 1989;29:80-85.

129. Mayeux R, Alexander MP, Benson DF, et al. Poriomania. Neurology 1979;29:1616-1619.

130. Manchanda R, Miller H, McLachlan RS. Post-ictal psychosis after right temporal lobectomy. J Neurol Neurosurg Psychiatry 1993; 56:277-299.

131. Logsdail SJ, Toone BK. Post-ictal psychosis; a clinical and phenomenological description. Br J Psychiatry 1988;152:246-252.

132. Schenck CH, Milner DM, Hurwitz TD, Bundlie SR, Mahowald MW. Dissociative disorders presenting as somnambulism: polysomnographic, video and clinical documentation (8 cases). Dissociation 1989;11:19-24.

133. *Schenck CH, Mahowald MW. Update on nocturnal psychogenic dissociative disorders: data from a series of 25 patients undergoing polysomnographic monitoring. Bloomington, MN: Abstracts of the Annual Midwest Sleep Society Meeting, 1996;7:23.*

134. *American Psychiatric Association. Diagnostic and statistical manual of mental disorders. (3rd ed). Washington DC: American Psychiatric Association, 1987.*

135. *Bisson JI. Automatism and post-traumatic stress disorder. Br J Psychiatry 1993;163:830-832.*

136. *Coy JD. Letter to the editor. J Emerg Med 1996;14:760.*

137. *Pontius AA. Homicide linked to moderate repetitive stresses kindling limbic seizures in 14 cases of limbic psychotic trigger reaction. Aggression and Violent Behavior 1997;2:125.*

138. *Bryk M, Siegel PT. My mother caused my illness: The story of a survivor of Munchausen by proxy syndrome. Pediatrics 1997; 100:1.*

139. *Byard RW, Beal SM. Munchausen syndrome by proxy: Repetitive infantile apnoea and homicide. J Paediatr Child Health 1993; 29:77-79.*

140. *Griffith JC, Slovik LS. Munchausen by proxy and sleep disorders medicine. Sleep 1989;12:178-183.*

141. *Light MJ, Sheridan MS. Munchausen syndrome by proxy and sleep apnea. Clin Pediatr 1990;29:162-168.*

142. *My JH, Maccia RJ, Kanter JL. Munchausen s syndrome. A medico-legal dilemma. Med Sci Law 1997;37:198.*

143. *Rosenberg DA. Web of deceit: A literature review of Munchausen syndrome by proxy. Child Abuse Negl 1987;11:547-563.*

144. *Samuels MP, McClaughlin W, Jacobson RR, et al. Fourteen cases of imposed upper airway obstruction. Arch Dis Child 1992;67:162-170.*

145. *Skau K, Mouridsen SE. Munchausen syndrome by proxy: A review. Acta Paediatr 1995;84:977-982.*

146. *Mahowald MW, Schenck CH. Complex motor behavior arising during the sleep period: forensic science implications. Sleep 1995; 18:724-727.*