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Sleep Violence—Forensic Science Implications: Polygraphic and Video Documentation

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ABSTRACT: During the past century, infrequent, anecdotal reports of sleep-related violence with forensic science implications have appeared. Recent rapid developments in the field of sleep-disorders medicine have resulted in greater understanding of a variety of sleep-related behaviors, and formal sleep-behavior monitoring techniques have permitted their documentation and classification. Sleep-related violence can be associated with a number of diagnosable and treatable sleep disorders, including (1) night terrors/sleepwalking, (2) nocturnal seizures, (3) rapid eye movement (REM) sleep-behavior disorder, (4) sleep drunkenness, and (5) psychogenic dissociative states occurring during the sleep period. Potentially violent automatized behavior, without consciousness, can and does occur during sleep. The violence resulting from these disorders may be misinterpreted as purposeful suicide, assault, or even homicide. Sleep-related violence must be added to the list of automatisms. A classification system of both waking and sleep-related automatic behavior is proposed, with recommendations for assessment of such behavior.

KEYWORDS: forensic science, sleep disorders, violence

> In all of us, even in good men,
> there is a lawless, wild-beast nature
> which peers out in sleep.
>
> PLATO, *The Republic*

> Acts done by a person asleep cannot be criminal,
> there being no consciousness.
>
> FITZGERALD, "Voluntary and Involuntary Acts"

One definition of automatism is: "an act which appeared purposive, executive, and at times extraordinarily elaborate, but occurring without the person knowing what he was doing, and the memory of which, in most instances, did not persist" [*1*]. Should violence or injury result from such automatic behavior, difficult forensic science issues may arise,

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as in cases of seizure-related incidents. As a result of routine clinical studies at our center, there has been firm documentation that sleep-related behaviors of diverse etiologies may result in dangerous automatic behavior with forensic science implications.

Methods

All subjects were referred to the Minnesota Regional Sleep Disorders Center for evaluation of bothersome or injurious behaviors occurring during the sleep period. All underwent formal psychiatric and neurologic evaluations and extensive polysomnographic (PSG) study. Clinical evaluation consisted of gathering a lifetime sleep/wake history, medical, neurologic, and psychiatric history and review of systems. PSG data were obtained by protocol, using standard methods for recording and scoring [*2*]. PSG monitoring included an electrooculogram (EOG), electroencephalogram (EEG), electromyogram (EMG: chin, bilateral anterior tibialis and bilateral extensor digitorum muscles), electrocardiogram (EKG), and oro-nasal airflow or more extensive respiratory monitoring. A nine-channel scalp EEG montage was used with a paper speed of 15 mm/s. The patients were continuously video-taped.

Results

Our findings have been published during various stages of our ongoing study on sleep-related injury [*3–7*]. We have been able to identify, name, and describe a new category of dangerous sleep disorder, the Rapid Eye Movement (REM) Behavior Disorder (RBD), thus underscoring the importance and use of conducting formal, extensive PSG studies of nocturnal behavior disorders. The following case vignettes illustrate the various types of primary sleep disorders which can result in accidental injury or death.

Case 1: Night Terrors/Sleepwalking (NT/SW)

M. O., a 67-year-old self-referred man presented with a 40-year history of frequently leaving bed during sleep, screaming in terror, and often running into walls and furniture while experiencing "fearful dreams" of being in a frightening situation requiring rapid flight. He had jumped through his double-paned, second-floor bedroom window on 4 occasions and once landed headfirst, sustaining a fractured C3 spinous process and multiple lacerations requiring hospitalization. He recalled "feeling as if some great force threw me from the bed and out the window." In fact, he had had to hurdle 5 ft (1.5 m) over a file cabinet and a desk to go through the window. Although he was referred to a psychiatrist because a suicide attempt was suspected, a psychiatric disorder was not identified. For the next year he attempted to confine himself to bed at night with a restraint jacket (Fig. 1). He ultimately came to our center because of recurrent sleep violence.

Two consecutive PSG studies did not detect seizures, sleep apnea, or any REM sleep abnormality, but did reveal an excessive number of spontaneous abrupt arousals exclusively from delta wave (Stage 3/4) sleep and also an elevated percentage of Stage 3/4 sleep for age. Thus, the PSG data in conjunction with the history established the diagnosis of night terror/sleepwalking.

This case calls attention to the dangerous but not rare persistence throughout the adult life cycle of a phenomenon typically considered to be a childhood condition, and graphically depicts how death might result from night terrors/sleepwalking: had this man jumped from a higher floor, or landed differently, or crashed on a surface harder than dirt, then the outcome probably would have been fatal.

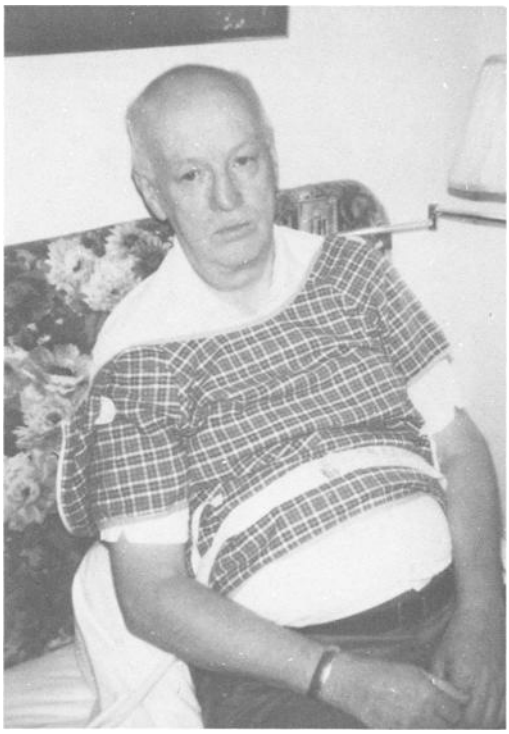

FIG. 1—*A 67-year-old man with severe NT/SW demonstrates use of a hospital restraint jacket which he had attached to his bed nightly for a year to prevent a recurrence of jumping through a second-floor window while asleep. During a prior episode he had sustained a cervical fracture and multiple lacerations.*

Case 2: RBD

G. M., a 73-year-old man, married for 46 years, had a 5-year history of progressively dangerous dream-enacting behaviors during sleep, beginning more than 2 h after sleep onset. He would repeatedly kick his wife or grab her arms tightly, and once her ankle remained injured for 2 months. She commented that "he becomes so terribly strong in his sleep." He frequently fell out of bed during sleep and had sustained a 4-in. (10-cm) head laceration after jumping into furniture while dreaming of being tackled by a dog. His wife noticed that on one occasion, "his arm shot straight out very rapidly,"—he awakened to report a dream of reaching for his gun and shooting an attacker in self-defense.

His most severe injury occurred four months before referral when he dove off his bed into a dresser and lost consciousness for 45 min. He was then hospitalized for twelve days and underwent neurosurgical repair of a fracture through the base of the odontoid process of C2 (Fig. 2). Postoperatively, a nurse in the hospital found him during the night yelling while suspended by his restraints over the edge of his bed; he was apparently asleep.

His wife reported that his premorbid sleep had been unremarkable, and that his violent sleep behaviors were completely unlike his pleasant waking personality. There was neither a psychiatric history nor a history of childhood sleepwalking or night terrors.

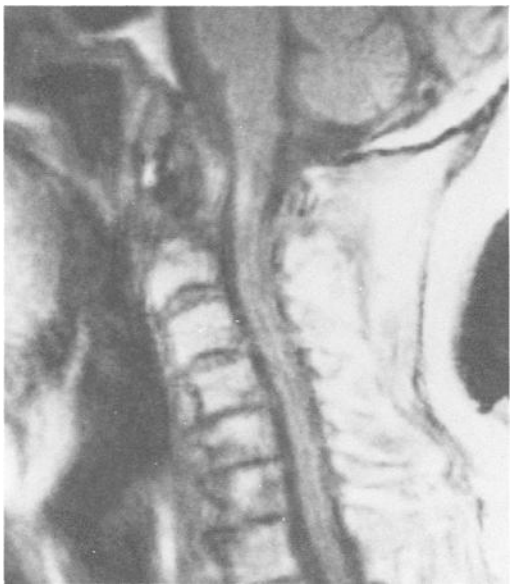

FIG. 2*a*—*Cervical MRI scan on a 73-year-old man who had leaped into a dresser while dreaming that he was jumping away from an attacker. Abnormalities include: (1) fracture through base of odontoid process, with odontoid process displaced posteriorly with respect to anterior arch of C1 and body of C2 and (2) prominent soft tissue masses along anterior-lateral spinal canal bilaterally adjacent to fracture, question whether posttraumatic hematoma. Overnight polysomnographic studies established diagnosis of RBD.*

Two consecutive overnight PSG studies did not detect seizures or sleep apnea. However, REM sleep was abnormal on both nights, with loss of the customary atonia and emergence of kicking and arm flailing, one sequence of which was a verified dream enactment (Fig. 3). These findings confirmed the diagnosis of RBD [5], and treatment with clonazepam, 0.5 mg just before sleep (hs), resulted in prompt control of abnormal sleep behaviors.

Case 3: Nocturnal Seizure

K. W., an 18-year-old high-school student was referred for evaluation of violent nocturnal behavior, occurring up to 6 times nightly. His father described these spells as "like sleepwalking." These were characterized by screaming, jumping up and out of the bed, and running around the house. During these spells, he had fallen on the stairs and broken closet doors, injuring himself in the process. These were associated with complete amnesia. Multiple all-night PSG recordings using extensive scalp-EEG montages were performed, and multiple spells were recorded. The EEG during most spells was obscured by movement artifact; however, between spells, isolated left temporal spikes and frequent bilaterally synchronous rhythmic paroxysmal activity were present. Immediately before 2 spells, there was atypical, but definite, left hemisphere electrical seizure activity (Fig. 4).

Before the sleep laboratory evaluation, extensive neurologic evaluation had been performed, and no evidence for seizures was found. An erroneous psychiatric diagnosis had

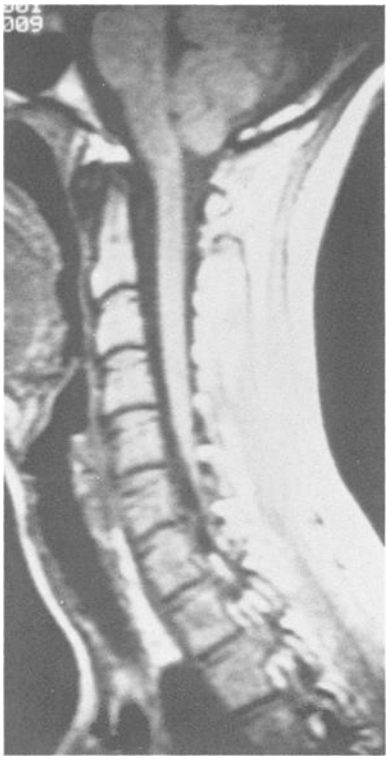

FIG. 2*b*—*Cervical MRI scan showing a normal odontoid process, in contrast to fracture seen in Fig. 2*a.

been made, and the patient and the entire family had been participating in ongoing psychiatric evaluation and counseling. Treatment with anticonvulsant medication alleviated the spells.

Case 4: NT/SW

P. A., a 16-year-old white male high-school student was referred for evaluation of possible "sleep driving" which had resulted in a serious motor vehicle accident. He had no history of psychiatric disease. Three months before the accident, he had an episode of complex sleepwalking.

On the night of the event, he had been sleep deprived, having stayed up later than usual the preceding night studying for a test. He arrived home after a party, which did not involve drugs or alcohol, at 1:00 a.m. and 15 min later, fell asleep in a chair watching television. He awakened at 1:45 a.m. after having been involved in a two-car accident 1.5 miles (2.4 km) from home on a road he never routinely drove. He was amnestic for the interval between having fallen asleep and the accident and could conceive of no reason for his having been driving on that road at that (or any) time. An all-night sleep study revealed unusually frequent arousals from slow-wave sleep with no evidence of seizure activity. This case underscores the fact that sleepwalking behavior may be extremely complex.

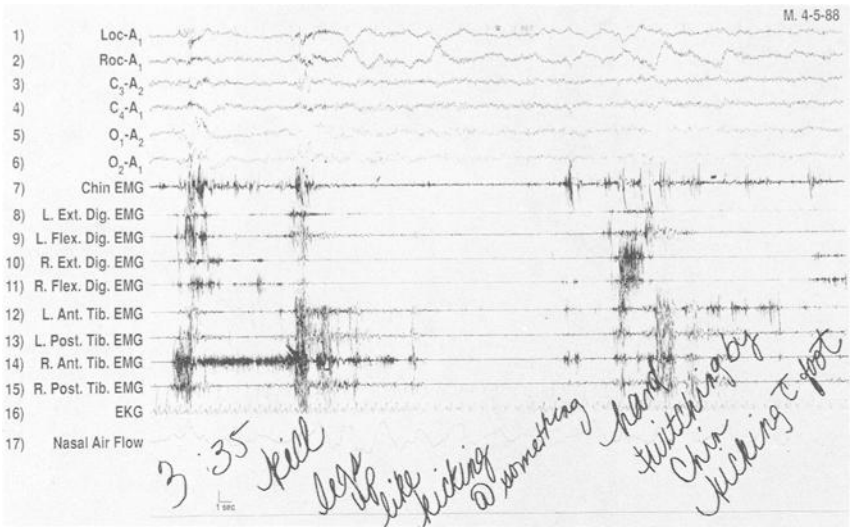

FIG. 3—*PSG of a 73-year-old man with RBD. REM stage of sleep is identified by presence of REMs (1–2), an activated low-voltage EEG (3–6), and a substantially atonic chin EMG (7). However, there is clearly excessive, intermittent loss of chin EMG atonia, with emergence of high voltage, phasic twitching. Limb EMGs (8–15) also have excessive twitching, and the technician observes vigorous behaviors.*

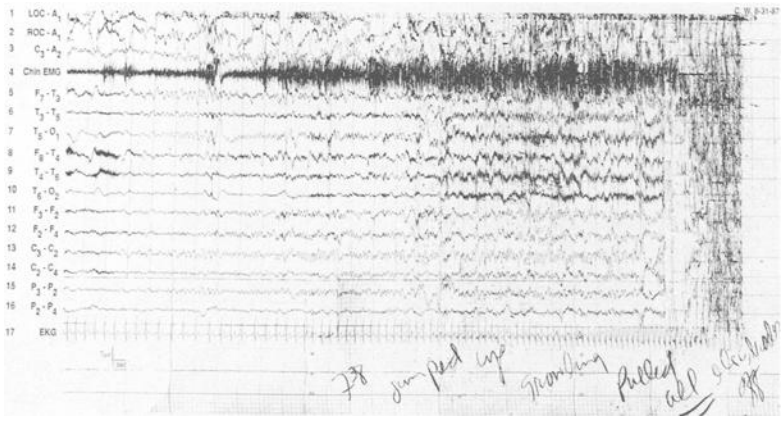

FIG. 4—*PSG of an adolescent with violent nocturnal seizures (Case 3). Initially there is generalized EEG suppression, followed by left-sided rhythmic activity (Channels 5,6,7,13,15) culminating in his jumping out of the bed, growling, and ripping off the electrodes.*

Discussion

Automatized behavior may arise from both wakefulness and sleep. We have reviewed the literature and propose a useful clinical classification (Table 1).

Review of Violent Wakeful Automatized Behavior

Automatic behavior may be seen in a wide variety of central nervous system (CNS) disorders including vascular, neoplastic, toxic/metabolic, infectious, degenerative, and traumatic conditions. The common thread among most of these disorders is amnesia—loss of short-term memory, possibly as a result of dysfunction within the limbic system. These conditions have been discussed elsewhere [*8*] and will be mentioned only briefly.

Transient global amnesia (TGA), which is generally felt to represent transient ischemia of the posterior circulation, but has also been associated with migraine headaches [*9*] or seizures [*10*], has resulted in illegal activity [*11,12*]. More persistent amnestic states may occur with limbic lesions such as tumors, infections (particularly limbic encephalitis as with H. Simplex infections or paraneoplastic states), trauma, or the Wernicke-Korsakoff syndrome induced by thiamine deficiency [*8*]. The medicolegal aspects of waking amnesia and automatisms have been well reviewed [*13,14*].

A number of toxic/metabolic states have been associated with automatic and sometimes violent behavior. These include hypoglycemia (murder, fatal motor vehicle accident, assault) [*1,15–17*], hypoxia/carbon monoxide poisoning [*8*], and alcohol intoxication or withdrawal [*18–20*]. Phencyclidine ingestion may also be associated with violent behavior [*21*].

The role of seizures in automatic behavior associated with aggression and violent or criminal actions has been extensively reviewed [*22–38*]. Although said to be controversial, our Case 3 confirms that seizures may indeed result in violence. TGA may also be the manifestation of seizures [*10*]. "Absence spells" induced by prolonged (lasting for

TABLE 1—*Conditions associated with automatic behavior.*

I. Organic neurologic disorders
  A. Vascular—transient global amnesia (TGA) (including migraine)
  B. Mass lesions
    1. increased intracranial pressure
    2. deep midline lesions
  C. Toxic/metabolic
    1. endocrine
    2. hypoxia/carbon monoxide poisoning
    3. drugs/alcohol (intoxication/withdrawal)
    4. thiamine deficiency (Wernicke-Korsakoff)
  D. Infectious (limbic encephalitis)
  E. Trauma
  F. Seizures
  G. Sleep disorders
    1. night terrors/sleepwalking (NT/SW)
    2. REM sleep behavior disorder (RBD)
    3. nocturnal seizures
    4. sleep drunkenness (SD)
      A. narcolepsy and idiopathic CNS hypersomnolence
      B. sleep apnea
      C. sleep deprivation
II. Psychogenic dissociative states (may occur exclusively from sleep)
  A. Fugues
  B. Multiple personality disorder
  C. Psychogenic amnesia

hours) periods of hyperventilation may masquerade as either intermittent amnesia or seizures [*39*].

Review of Violent Sleep-Related Automatized Behavior

Scattered reports and infrequent reviews of sleep-related violence have appeared in the medical and legal literature and in the lay press. Reported events have included

- somnambulistic homicide, attempted homicide [*40–52*];
- murders and other crimes with sleep drunkenness [*53*], including sleep apnea [*54*] and narcolepsy [*55*];
- suicide [*48, 56, 57*]; and
- NT/SW with potential violence/injury [*58–60*].

It is clear that during both wakefulness and sleep, the dissociation of motor activity and consciousness may occur in a number of situations, best classified as neurologic or psychiatric in their etiology.

Differential Diagnosis of Sleep-Related Violent Behavior

Disorders of Arousal: NT/SW—These are impressive sleep phenomena which tend to arise from Stages 3 and 4 slow-wave sleep, the deepest stages of non-rapid eye movement (NREM) sleep, usually occurring in the first third of the sleep cycle and rarely during naps. There is frequently a positive family history, suggesting a genetic component; they are common in childhood, usually decreasing in frequency with advancing age [*61–66*]. The night terror is also termed pavor nocturnus. *Sleepwalking* or *somnambulism* is prevalent in childhood (up to 17%) with a peak incidence at eleven to twelve years of age and is more common in adults (2.5%) than generally acknowledged [*7,67,68*]. The *night terror* is the most dramatic disorder of arousal. It is frequently initiated by a loud, blood-curdling scream associated with cataclysmic panic, and may be followed by impressive motor activity such as hitting the wall, running around or out of the bedroom—resulting in bodily injury or property damage. A common feature is inconsolability during the activity, with complete amnesia for the event [*65,66,69*]. However, our own experience runs counter to the dictum that there is never remembered imagery [*7*]. The individual appears to be awake, but is unable to perceive properly the environment; attempts at consolation are often fruitless and may serve only to prolong the confusional state. The commonly held belief that somnambulism and night terrors are benign and do not result in injury is clearly erroneous. The accompanying behaviors may be violent, resulting in considerable injury to the individual, others, or damage to the environment (Figs. 5 through 7) [*7,47–50,53,54,58–60*].

Precipitants of NT/SW include febrile illness, alcohol, prior sleep deprivation, and emotional stress [*53,54,70*]. Medication-induced cases have been reported with sedative/hypnotics, neuroleptics, minor tranquilizers, stimulants, and antihistamines, often in combination with each other or with alcohol [*50,71,72*].

Although it is commonly felt that persistence of these behaviors beyond childhood or that their development in adulthood is an indication of significant psychopathology [*73,74*], this may not necessarily be the case [*75*]. Our own experiences suggest that it is not [*7*].

The mechanism of these disorders is not clearly understood, but both genetic and environmental factors are operant. An underlying physiologic process is suggested by: (1) the atypical arousal/EEG pattern recorded during the episodes, suggesting the simultaneous occurrence of wakefulness and sleep; (2) the difference of visual evoked potentials during arousal from slow wave sleep as compared to those during arousal from

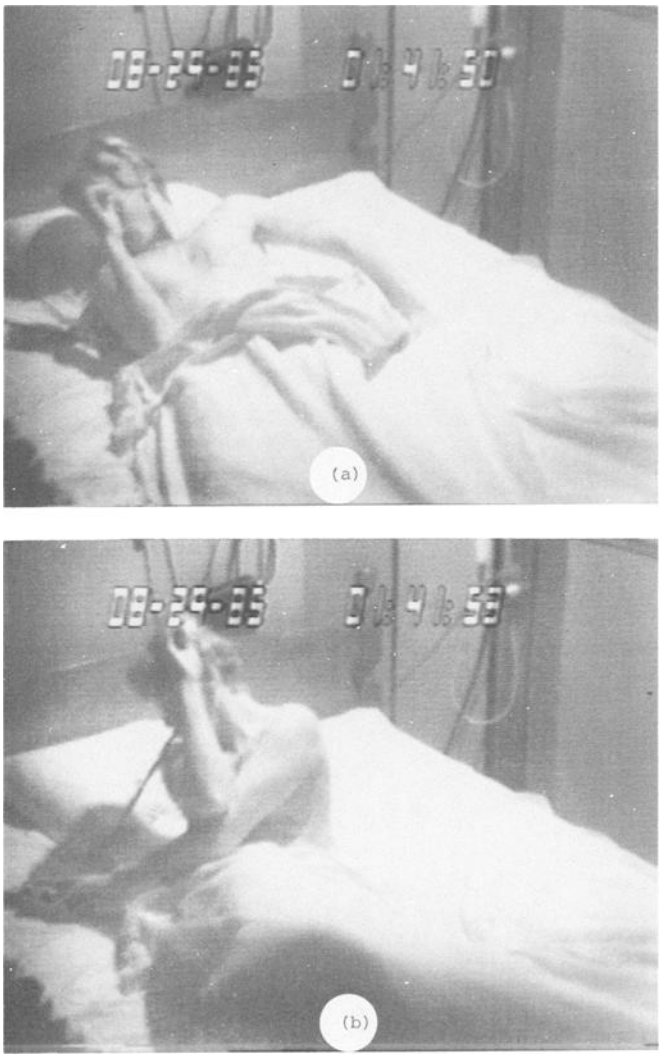

FIG. 5—*Physical appearance of a subject during a typical night terror. Photographs from a sleep lab videotape illustrating the abrupt onset from Stage 3/4 sleep of a violent NT episode. This 24-year-old man reported feeling sudden terror as he perceived a threatening object.* (a). *He then becomes poised to attack in self-defense* (b). *This sequence culminates with several hard punches and yelling.*

REM sleep or during wakefulness, suggesting an alteration of consciousness [*76*]; and (3) the unusually intense and persistent orienting response to auditory stimuli in subjects suffering from night terrors (as measured by somatic, autonomic, EEG activity, and responsiveness), indicating a heightened intrinsic excitability of the nervous system [*77*]. That somnambulism can be induced in normal children by standing them up during slow-wave sleep [*78,79*] and that night terrors can be precipitously triggered in susceptible individuals by sounding a buzzer during slow-wave sleep [*65,79*] speak against these behaviors being the culmination of complex, ongoing sleep mentation. In addition, a sudden event is again suggested by precipitous acceleration of heart rate and respiration—rather than the gradual acceleration seen before a REM dream anxiety attack [*66*].

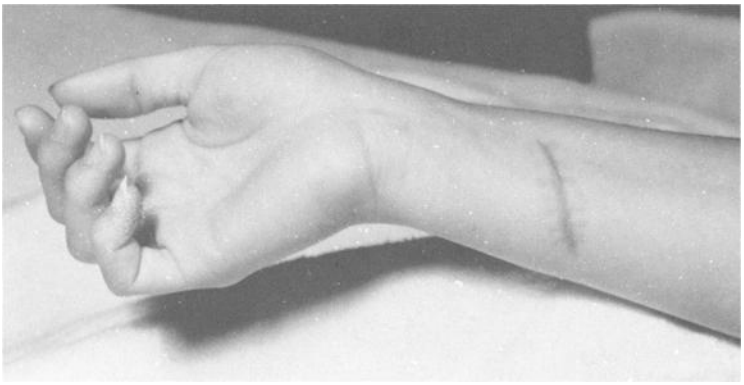

FIG. 6—*Forearm laceration sustained during a SW episode which resulted in subject thrusting arm through a window.*

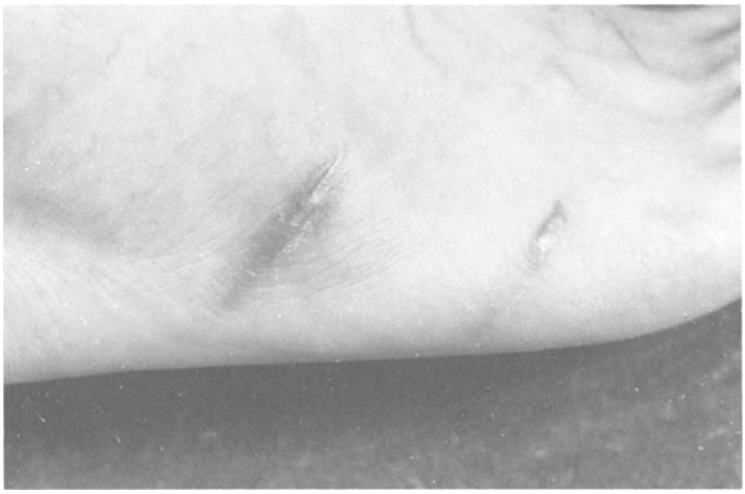

FIG. 7—*Foot lacerations sustained by subject while kicking and breaking a window during frenzied SW.*

Superficially, the night terrors are similar to the REM dream anxiety attacks and to the posttraumatic stress disorder. However, NT/SW are associated with more impressive autonomic activity (diaphoresis, tachycardia, hypertension, tachypnea), and preceding complex mental imagery is less frequently recalled. Simple frightening images (a face, animal, fire, and so forth) may be remembered following a night terror, but usually not the intricate plot or series of events experienced in the REM sleep dream anxiety attack [*80*]. Except in cases of the REM behavior disorder (discussed below), there is generalized somatic muscle atonia (paralysis) during REM sleep dreams, which would preclude such motor activity [*5*]. The potentially violent sleep-related behavior associated with the posttraumatic stress disorder arises from all stages of sleep, indicating that it is neither simple REM sleep anxiety nor NT/SW [*81*].

Treatment of uncomplicated NT/SW is often not necessary. Provision of reassurance and the understanding that the symptoms may diminish with time is often sufficient. The tricyclic antidepressants (amitriptyline [*82*] and imipramine [*83–85*]) and benzodiazepines (alprazolam [*86,87*], diazepam [*85,88–90*], midazolam [*91*], temazepam [*92*], and clonazepam [*7*]) may be effective, and should be administered if the behaviors are dangerous to person or property or extremely disruptive to family members. Nonpharmacologic treatments such as psychotherapy [*93*], progressive relaxation [*94*], and hypnosis [*90,95,96*] are recommended for long-term management. The avoidance of drugs, alcohol, and sleep deprivation are also important. Finally, note that "*all the characteristics of somnambulism underline the difference between wakefulness and consciousness*" [*56*]. Shakespeare's understanding of this concept was demonstrated in *Macbeth*: during Lady Macbeth's sleepwalking, the Doctor of Physic says, "'You see, her eyes are open,'" the Waiting-Gentlewoman responding, "'Ay, but their sense is shut'" (v,i).

RBD—Normally, REM sleep is associated with generalized somatic muscle paralysis (atonia), which may act as a protective measure by preventing the physical enactment of dreams. Recently, our center identified a disorder in humans (predicted in 1965 by animal experiments [*97*]) in which the REM-related atonia is absent, resulting in dramatic and frequently injurious behavior during dreams—the chronic RBD. The extensive oneiric (dream) behaviors displayed by these patients is frequently misdiagnosed as manifestations of a seizure or psychiatric disorder. Although usually idiopathic and tending to affect older males, it has been associated with a variety of primary neurological diseases in approximately one third of cases [*6*]. The overwhelming male predominance (95%) of RBD may support animal and human data relating sexual hormones to aggression and violence [*98,99*]. Eighty–six percent of our 55 RBD patients presented with the chief complaint of sleep injury (fractures, lacerations, repeated ecchymoses) [*6*]. The following methods used by the RBD patients to minimize sleep-related injury attest to the severity of this pathological condition: restraining devices (Posey jacket, rope around the waist, tethered to the bed [Fig. 8]), covering windows with plastic, forming pillow barricades, using sleeping bags (two were destroyed), padded waterbeds, mattress on the floor, sleeping in an empty room. Several wives have been nearly strangulated by their husbands who had grabbed their necks very tightly during attempted dream enactment. Others sustained severe headaches lasting days from blows to the head. Treatment with clonazepam is very effective [*5*] for RBD patients.

A similar, but transient, form is seen in toxic/metabolic conditions, induced by medications such as tricyclic antidepressants, monoamine oxidase inhibitors, or biperiden [*100–105*]. It may also be seen during withdrawal states, most notably ethanol withdrawal [*106*], and may account for some of the bizarre behavior characteristic of delirium tremens and other withdrawal or toxic states [*107–111*].

Nocturnal Seizures—Approximately 10% of patients with seizures experience spells exclusively or predominantly during sleep [*112*]. Frantic and elaborate nocturnal motor activity is well documented in seizures originating in the orbital, mesial, or prefrontal region [*113–118*]. A condition termed "episodic nocturnal wanderings," clinically indistinguishable from other forms of sleep-related motor activity such as sleepwalking, but which is responsive to anticonvulsant therapy, has also been described [*119,120*]. Clinical seizure-like activity without identifiable epileptiform activity in the scalp-EEG is characteristic of the condition termed "hypnogenic paroxysmal dystonia," which responds effectively to treatment with the anticonvulsant carbamazepine [*121,122*]. Other unusual seizures which may present as automatic behavior include "paroxysmal nightmares" [*123*] and (debatably) "episodic dyscontrol" [*124,125*].

Sleep Drunkenness (SD)—Delirium (which has been dubbed "everyman's psychosis," because everyone is susceptible to it) [*126*] occurring during the transition between sleep and wakefulness may result in sleep drunkenness (SD), a disturbance of cognition and

FIG. 8—*An elderly man with RBD wears a restraining device he used nightly for over five years to confine himself to bed during sleep. A subarachnoid hemorrhage, from which the subject had otherwise recovered, previously had triggered the onset of a disorder of violent, dream-enacting behaviors which was confirmed to be RBD by PSG study. Treatment with nightly clonazepam was successful; he has not worn this device for the past six years.*

attention coincident with the motor behavior of wakefulness, resulting in complex behavior without conscious awareness [*127–129*]. SD is potentiated by prior sleep deprivation or the ingestion of alcohol or sedative/hypnotics before sleep onset [*130*] and could be expected to occur more frequently in the setting of chronic sleep fragmentation as seen in obstructive sleep apnea or in conditions of impaired wake-sleep "boundary control" such as narcolepsy. Shoplifting has been reported during a period of automatic behavior in a narcoleptic [*55*]. Such behavior may be explained by the simultaneous occurrence of or rapid oscillation between waking and sleep states [*5,131*].

Patients suffering from obstructive sleep apnea or periodic movements of sleep experience frequent arousals which may serve to trigger arousal-induced precipitous motor activity.

Psychogenic Dissociative States—Waking dissociative states may have forensic-science implication [*132*]. Recently, violent or injurious psychogenic dissociative states arising from EEG wakefulness during the sleep period have been described [*7,133,134*].

Clinical and Laboratory Evaluation of Waking, Sleep/Violence

Violent, automatic behavior arising during wakefulness should be evaluated by a detailed medical history and physical examination, specifically addressing amnestic, toxic/metabolic, or epileptiform conditions.

The history of prominent, violent, or potentially injurious motor behavior arising from sleep should invoke the possibility of one of the aforementioned organic conditions. Our experience with over 100 adult cases of sleep-related injury/violence has repeatedly indicated that clinical differentiation, without PSG study, among RBD, night terror/som-

nambulism, nocturnal seizures, sleep apnea, and sleep-related psychogenic dissociative states may be impossible [*5–7*].

Evaluation must include a complete review of sleep/wake complaints from patient or bedpartner or both and detailed neurologic and psychiatric histories and examinations. The diagnosis may only be suspected clinically. Extensive polygraphic study employing an extensive scalp EEG at a paper speed of 15 mm/s, electromyographic monitoring of all four extremities, and continuous audiovisual recording are mandatory for correct diagnosis [*119,135–141*]. Detailed technician observations, attempts at precipitating arousals (such as by sudden noise during slow wave sleep), and the patient's response to attempted awakenings during an episode are invaluable (Fig. 9).

Establishing the diagnosis of nocturnal seizures may be extremely difficult, as the motor activity associated with the spell often obscures the EEG pattern. Further, there may be no scalp-EEG manifestation of the seizure activity. Numerous well-documented cases of scalp-electrode EEG-negative but depth-electrode EEG-positive electrical seizure activity [*142,143*] or video-documented clinical seizure activity [*144*] have been reported. Another possible explanation for "scalp-electrode EEG-negative" seizures is that some seizures are manifest electrically with only generalized low-voltage fast activity, not followed by post-ictal slowing [*145*]. Such activity arising from EEG-recorded sleep may be misinterpreted as an "arousal," rather than as electrical seizure activity. The aforementioned difficulties in evaluating nocturnal seizures (obscuring of the record by movement artifact, absence of surface electroencephalographic abnormality or electrical seizure activity, lack of post-ictal slowing, misinterpretation of electrical seizure activity as an "arousal") emphasize the necessity of extensive, in-person laboratory monitoring. (Scantily-channeled "ambulatory" EEG monitoring has led to the misdiagnosis of functional psychiatric

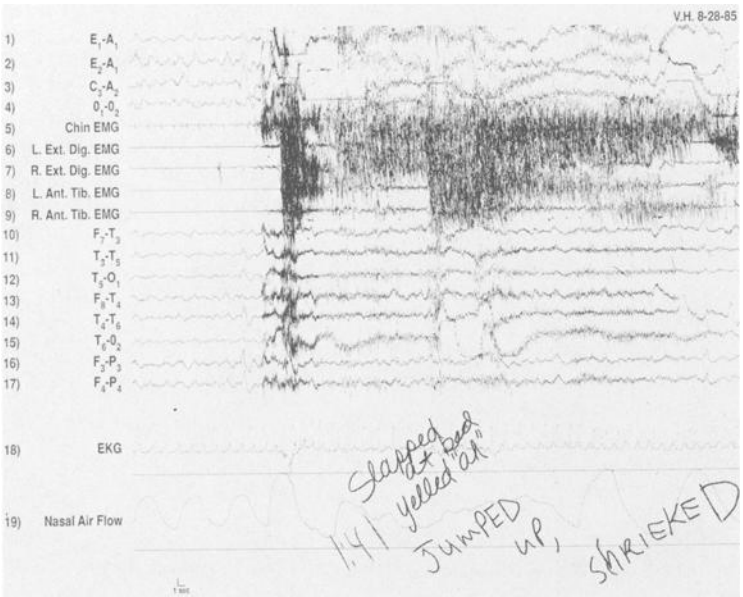

FIG. 9—*PSG of a 24-year-old man with lifelong severe NT/SW depicting a typical episode of NT. An unremarkable sequence of delta-wave (Stage 3/4) sleep (EEG: 3–4, 10–17) is precipitously terminated by a pathological arousal in which subject is seen to slap at bed, yell out, and jump up. Chin EMG: 5; limb EMGs: 6–9.*

disease in a number of our patients subsequently demonstrated to have bona fide nocturnal seizures.) Such studies are best performed in experienced sleep disorders centers with interpretation by a veteran clinical polysomnographer. If the history or physical examination suggests underlying neurologic disease, further studies such as magnetic resonance imaging (MRI) or computerized tomographic (CT) scanning of the brain, multimodal (visual, auditory and somatosensory) evoked potentials, or formal neuropsychometric evaluation are indicated.

Legal Evaluation of Sleep Violence

The legal implications of automatic behavior have been discussed and debated in both the medical and legal literature [*146–151*]. As with non-sleep automatisms, the identification of a specific underlying organic or psychiatric sleep/violence condition does not establish causality for any given deed.

To assist in the determination of the putative role of an underlying sleep disorder in a specific violent act, we offer these guidelines, modified from Bonkalo (sleepwalking) [*53*], Walker (epilepsy) [*38*], and Glasgow (automatism in general) [*152*], and formulated from our clinical experience:

1. There should be reason (by history or formal sleep laboratory evaluation) to suspect a bona fide sleep disorder. Similar episodes, with benign or morbid outcome, should have occurred previously.
2. The duration of the action is usually brief (minutes).
3. The behavior is usually abrupt, immediate, impulsive, and senseless—without apparent motivation. Although ostensibly purposeful, it is inappropriate to the total situation, out of (waking) character for the individual, and without evidence of premeditation.
4. The victim is someone who merely happened to be present and who may have been the stimulus for the arousal.
5. Immediately following return of consciousness, there is perplexity or horror, without attempt to escape or to conceal or cover up the action. There is evidence of lack of awareness on the part of the individual during the event.
6. There is usually some degree of amnesia for the event; however, this amnesia need not be complete.
7. In the case of NT/SW or sleep drunkenness, the act may:
   (a) occur upon awakening (rarely immediately upon falling asleep)—usually at least one hour after sleep onset,
   (b) occur upon attempts to awaken the subject, or
   (c) have been potentiated by alcohol ingestion, sedative/hypnotic administration, or prior sleep deprivation.

Summary

Violent behavior during sleep may result in events which have forensic science implications. The apparent suicide (for example, a leap to death from a second-story window), assault, or murder (for example, strangulation, stabbing, shooting) may be the unintentional, nonculpable but catastrophic result of NT/SW, nocturnal seizures, RBD, or psychogenic dissociative states. These frequently treatable sleep-related conditions should be considered as causes of injury or death, particularly in cases of apparently motiveless and otherwise inexplicable violence occurring during the sleep period.

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