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ORIGINAL PAPER

Sleepwalking, A Possible Side Effect of Antipsychotic Medication

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Abstract Two case examples and a review of the sleep literature illustrate the potential of antipsychotic medication to trigger sleepwalking episodes in the context of schizophrenia. Causative hypotheses are briefly reviewed, as well as risk factors, differential diagnosis, and management. Sleepwalking may contribute to delusions, aggression, and accidental suicide. It is important to investigate sleep disorders in schizophrenia. They are not rare and may contribute to behavior that increases the stigma and isolation of individuals with schizophrenia.

Keywords Schizophrenia · Sleepwalking · Antipsychotic side-effects · Suicide · Violence

In 2002, I read that a 12-year-old adolescent had seriously injured herself during the night [1]. The injury was initially considered to be the result of a suicide attempt but, on investigation, the underlying cause proved to be a sleepwalking disorder. This led me to reconsider the cause of death of an adult patient with a diagnosis of schizophrenia who had been found in the buff, frozen to death on the street in the middle of a Canadian winter night. Our team had called it suicide but, in retrospect, he had probably been sleepwalking. He had previously demonstrated somnambulistic-like behavior when hospitalized, although it was not interpreted as such; it was seen as willful mischief making. He was taking antipsychotic medication and he was obese. Both obesity and antipsychotics can increase the risk for sleepwalking [2]. Another patient diagnosed with schizophrenia and treated with antipsychotics threw herself out the window of a several storey building in the middle of the night. Again, this was seen as a suicide, and perhaps it was, since she had attempted suicide previously. But she was not alone at the time of the incident, she was not considered depressed, and she had been drinking and using drugs—a frequent trigger for a sleepwalking episode. She communicated no wish to end her life and she left no note. There was no prior

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Table 1 DSM-IV-TR criteria for sleepwalking disorder

Arousals in slow-wave sleep
Unresponsiveness during episode
Amnesia for episode
Confusion upon awakening

history of sleepwalking or night terrors whereas most adult sleepwalkers do have a childhood history of such behaviors [3] but, then again, she was never asked.

Schizophrenia patients show disturbed sleep patterns from 30 to 80% of the time when studied with polysomnography [2]. Among 1235 psychiatric outpatients (all diagnoses) in Hong Kong, the prevalence of sleepwalking was recently found to be 8.5% (compared to the general adult population prevalence of 2.6%) [4]. Sleepwalking disorder refers to complex motor behaviors, usually initiated during deep (stages 3–4) non-rapid-eye-movement sleep and often familial in occurrence. The disorder is characterized by a dissociated state of partial awakening; the motor system is awake but consciousness remains clouded. The patient is often frightened and defensive during episodes and, upon full wakening, has partial or complete amnesia for what took place (Table 1).

Most psychotropic drugs, not only antipsychotics, are associated with parasomnias, but this side-effect is little discussed in the psychiatric literature, an unfortunate omission since sleepwalking can bring with it serious repercussions. As many as 24% of adult sleepwalkers report self-injury during episodes [5]. Sleepwalking is associated with violence, even homicide [6]. In individuals diagnosed with psychotic illness, nighttime behaviors that appear odd can add to stigma and isolation. The effects of sleepwalking can be mistaken for symptoms of relapse and paradoxically treated with increased doses of antipsychotics, which may exacerbate the problem.

Case Example Jenna (Not Her Real Name)

Jenna was a 50-year-old woman who lived alone and attended the Women's Psychosis Clinic at the Centre for Addiction and Mental Health. She had a diagnosis of schizophrenia since her early 20s and was being treated with phenytoin for epilepsy. Jena's distressing auditory hallucinations (voices of terrorized children) had been very difficult to treat. She developed side-effects to all drugs tried on an outpatient basis and had to be hospitalized in order to achieve a quetiapine dose that worked (800 mg/day). This was accomplished by increasing the dose slowly in hospital, in very small gradients. Jenna also took sertraline 200 mg/day for co-morbid depression. On this combination of medications, Jenna recovered and actively re-engaged with her family and with two support and activity groups. Occasionally, however, she came to her therapy sessions distressed, claiming that someone was breaking into her apartment at night. Her evidence was that household items were being displaced; she awoke in the morning to find that books she had placed on her night table were in the living room or dishes left in the sink were in the dishwasher. Nothing was ever stolen. She attributed the night intrusions to malevolence on the part of her neighbors, attempts to persuade her to move so that their relatives could occupy her apartment. The care team took these to be delusional thoughts and her quetiapine dose was increased to 900 mg/day. Her distress continued, but her complaints changed from break-ins to reports of sleepwalking. These reports were vague because her memory of them was vague. She had imprecise memories of going to the kitchen and eating at night. One day she said that she had gone out at night and had taken a taxi somewhere, she did not know where. She

was quite sure of this, but could remember no further details. She was referred to a sleep clinic. The somnography supported a diagnosis of sleepwalking and she was prescribed clonazepam 2 mg HS. The team was reluctant to have Jenna take even more sedative medication than she was already taking, but the regimen seemed to work. There were no further reports of sleepwalking.

Etiology

Serotonin Hypothesis

Sleepwalking induced by psychotropic drugs has been frequently reported in the literature, including two cases attributed to quetiapine [7]. In the report by Hafeez and Kalinowski, one man was on 800 mg/day, the other on 400 mg/day at the time of the investigations. In both cases, the sleepwalking disappeared when the doses were reduced. The article suggests that deregulation of serotonergic activity may be the root of the problem and that two areas of the central nervous system may be involved: the serotonergic neurons of the raphe nuclei of the brainstem and the serotonergic neurons that modulate the motor system. These two neuronal systems are normally coordinated so that increased arousal leads to increased motor activity, but when the two systems dissociate, sleepwalking can result. Many of the substances known to precipitate somnambulism, including quetiapine, have been associated with altered central serotonin (5-HT) activity.

The serotonin hypothesis of parasomnias was first proposed by Polish researchers, Juszczaka and Swiergiela [8] who also noted that paroxetine taken before sleep could trigger episodes of somnambulism [9]. Paroxetine is a serotonin reuptake inhibitor similar to the sertraline that Jenna was taking. SSRIs, including sertraline, are known to increase slow wave sleep, linking this class of drugs to sleepwalking [10].

This points to the fact that Jenna's sleepwalking episodes were likely multidetermined, with high doses of quetiapine and sertraline both contributing. Jenna was a heavy cola drinker and caffeine abuse has been implicated in sleepwalking [11]. She also suffered from epilepsy, which could have served as a further contributory cause to her night walking.

GABA Hypothesis

The nonbenzodiazepine receptor agonists used to induce sleep (zolpidem, zaleplon, zopiclone) have been frequently implicated in sleepwalking behaviors. Because these drugs act at the gamma-aminobutyric acid GABA(A) receptor benzodiazepine site, this has led to the GABA hypothesis of sleepwalking, implicating the enhancement of GABA activity at GABA(A) receptors (particularly alpha1-GABA(A) receptors) [12]. Neither Jenna nor the other patients discussed in this article were using sleeping pills.

Diagnosis

Somnography

Jenna's somnography at the sleep clinic showed an increase over normal in slow wave sleep (stages of 3 and 4 of NREM) and frequent arousals [13]. Frequent arousals during slow wave sleep are one of the most commonly reported sleep laboratory findings in

sleepwalkers. One other 'sign,' a burst of hypersynchronous delta waves prior to a behavioral arousal, is not a consistent finding and not specific to this disorder [14]. There is no specific diagnostic sleep protocol to confirm the diagnosis, although the standard recording and scoring of EEG sleep stages does help to rule out sleep seizures and REM Sleep Behavior Disorder (typically occurring later in the night, with patients enacting their dreams and retaining vivid recall of their behavior afterwards). Unless a behavioral arousal from slow-wave sleep happens to occur during an assessment, the results of a sleep study cannot be said to be conclusive. It is difficult to capture actual episodes of sleepwalking in a sleep laboratory; 25 h of sleep deprivation and forced arousals during slow-wave sleep have been advocated to help induce it for diagnostic purposes [15]. This was not tried in Jenna's case because her verbal descriptions were sufficiently vivid to make the diagnosis.

Differentiating Epilepsy

Though treated with phenytoin, Jenna continued to have occasional grand mal seizures. She had previously had seizures at night, and had found herself on the floor by her bed in the morning. There is a form of epilepsy, nocturnal frontal lobe epilepsy, which is not easy to distinguish from arousal parasomnias such as sleepwalking [16]. The two conditions have many features in common [17]. Both show (a) alimentary automatisms (lip smacking, tooth grinding, biting, night eating) (b) locomotor activity (wandering, restless leg syndrome, periodic limb movements) and (c) emotional reactions (fear, nightmares, sexual and aggressive behavior). Common features can be explained by the activation of identical neuronal networks. The main differentiating features in favor of nocturnal frontal seizures are: onset at any age, several attacks per night at any time during the night, brief duration with stereotyped motor pattern. Video-polysomnographic recordings of an episode are the gold standard for diagnosis. Home-made video recordings are helpful [18].

Other Differentials

Besides the possibility of a primary seizure disorder, space-occupying lesions need to be ruled out. There is a report in the literature of an arachnoid cyst coexisting with epileptic nocturnal wandering, but the authors suggest that it could be an incidental finding [19]. In this report, the cyst was left in place and the patient was treated with the anticonvulsant, levetiracetam. The night wanderings were greatly alleviated.

Other syndromes that have frequently been found to co-exist with sleepwalking and that should be suspected in the event of sleepwalking (and treated when found) are: Tourette's syndrome, sleep apnea or upper airway resistance syndrome, migraine headaches, hyperthyroidism, alcoholism [20]. It is generally acknowledged that any factors that deepen sleep, fragment sleep and/or make arousal from sleep more difficult increase the risk for sleepwalking (Table 2).

Management

Physicians should ask their patients about parasomnia behaviors and the use of medications or other substances. Patients who report continuing episodes of behavioral arousals during the first hours of sleep without precise recall of events should be counseled to avoid sleep deprivation and sleep schedule disruptions. If they are undergoing periods of prolonged stress, they should receive preventive treatment (changing or lowering the dose of relevant

Table 2 Triggers to sleepwalking

Short-acting hypnosedatives
Antipsychotics
SSRIs
Alcohol, caffeine, marijuana
Sleep deprivation
Stress
Hormonal transitions
Obesity
Sleep apnea
Thyrotoxicosis
Migraine

medications, lowering stress levels, discontinuing alcohol, caffeine, and marijuana use). All known underlying causes such as sleep apnea must be treated. Pharmacotherapy, after thorough sleep studies, consists of long-acting benzodiazepines and tricyclic antidepressants, but efficacy remains largely anecdotal [21]. Short acting sedatives exacerbate the problem but long acting ones such as clonazepam (0.5–3.0 mg HS) successfully suppress the episodes. Although the mechanism of action of clonazepam has not yet been established, the suggestion is that it involves (a) anticonvulsant effects on subclinical epilepsy, (b) an increase in 5-HT/monoamine synthesis or decrease in 5-HT receptor sensitivity mediated through the GABA system, and (c) regulation of GABA activity, which improves sleep [22]. It needs to be recognized, however, that no properly powered rigorous controlled trials exist for treatment of sleepwalking in adults [23].

2nd Case Example Cara (Not Her Real Name)

Cara was a 50-year-old divorced woman who lived alone and attended the Women's Psychosis Clinic at the Centre for Addiction and Mental Health. She was referred to the Clinic by a male psychiatrist because she had developed an erotomanic transference toward him. She carried a diagnosis of schizoaffective psychosis since her early 20s, and suffered episodic psychotic episodes (delusions and cognitive confusion) intermingled with episodic bouts of depression. Her illness had led to the break up of her marriage and the loss of custody of her daughter to her estranged husband. Her symptoms were controlled by lithium 300 mg TID and pimozide 6–8 mg qAM. Because of mood fluctuation and fleeting psychotic thinking, the pimozide dose needed frequent adjustment—a decrease when the patient became depressed and an increase when she became psychotic. As long as this was attended to, Cara remained in recovery, was able to live independently and, for many years, to be gainfully employed. She mentioned that sleepwalking, a problem she had had in childhood, had recently returned but the care team did not take this symptom seriously. One day she reported that her across-the-hall neighbors told her that she had rung their doorbell in the middle of the night dressed in a nightgown. The patient had no recollection of this incident and was extremely embarrassed, especially because she secretly found the man across the hall attractive and had day-dreamed about him. She wondered what she might have said in her somnambulistic state and, from then on, barricaded her front door with a heavy table each night before going to bed. On reading the literature about lithium and sleepwalking [24], the care team discontinued her lithium and added an SSRI to her drug regimen. The pimozide stayed. There were no further reports of sleepwalking.

Risk Factors

Lithium

There have been several reports of the association of lithium, either alone or together with antipsychotics, with sleepwalking. Charney et al. [25] reported that 10 of 114 psychiatric patients undergoing combined lithium-antipsychotic treatment experienced episodes of sleepwalking. These episodes occurred within 2–3 h after sleep onset and were characterized by confusion during the episode and post-episode amnesia. Two of the patients in this report also experienced grand mal seizures, thought to be unrelated.

Landry et al. [24] gave a sleepwalking questionnaire to patients attending a lithium clinic. Of those who completed the questionnaire, 6.9% reported episodes beginning at the time that lithium (either alone or in combination with other psychotropic drugs) was commenced. Childhood somnambulism was reported by 11.6% and, of those, 27% stated that the episodes had been reactivated by the medication. A history of childhood somnambulism, as in the case of Cara, may thus increase the risk of developing sleepwalking behavior while undergoing psychopharmacological treatment.

Studies in humans and animals indicate that lithium has a net enhancing effect on serotonin function and increases 5-HT release in some areas of the brain. It is likely that lithium-induced somnambulism is related to its effect on the serotonin system [26, 27].

Menopause

With age, the proportion of slow-wave sleep is reduced and parasomnias become far less common after puberty. This has led to speculation about the role of hormonal transitions in sleepwalking episodes. The prevalence of sleepwalking decreases from the pre-pregnant period to the second trimester [28]. During menopause, there is an increase in sleep difficulty in women, mainly due to depression, hot flashes, fibromyalgia, and disordered breathing [29–31]. Both Jenna and Cara were perimenopausal when the sleepwalking began. Both had periods of depression but did not complain of fibromyalgia, vasomotor symptoms, or trouble breathing.

Genetics

Although neither Jenna nor Cara reported a family history of sleepwalking, this condition has long been known to run in families [32]. In 80% of sleepwalkers, a first, second, or third-degree relative walks in their sleep or suffers from night terrors, a related condition. The presence of sleepwalking-like disorder in a first-degree relative increases the chances of developing this disorder by a factor of more than 10 [20]. A large Finnish twin cohort study (11,220 subjects aged 33–60 years, comprising 1,045 monozygotic and 1,899 dizygotic twin pairs) found adult sleepwalking in 3.9% of men and in 3.1% of women; it was reported as a weekly occurrence in 0.4% for both genders. For sleepwalking in childhood, the probandwise concordance rate was 0.55 for monozygotic and 0.35 for dizygotic pairs, and for adult sleepwalkers, the concordance was 0.32 for monozygotic, and 0.06 for dizygotic pairs. More than a quarter of boys and 18.3% of girls who remembered sleepwalking in childhood continued to do so as adults. Of adult male sleepwalkers, 88.9% had a positive history of sleepwalking in childhood, as did 84.5% of adult women sleepwalkers. Those who reported never having walked in their sleep in childhood rarely

did so as adults (0.6%). The proportion of total phenotypic variance attributed to genetic influences was 66% in men and 57% in women in childhood sleepwalking, and 80% in men but only 36% in women in adult sleepwalking, still a substantial genetic effect [3].

Lecendreux et al. [33] performed major histocompatibility complex, class II, DQ beta 1 (HLA-DQB1) typing on 60 Caucasian subjects diagnosed with sleepwalking disorder and their families and 60 ethnically matched subjects who had no diagnosed sleep disorder. Their findings suggest that specific DQB1 genes are implicated in disorders of motor control during sleep, but 65% of sleepwalkers lack this genetic marker. Moreover, it is present in over 13% of controls. The Lecendreux et al. finding has not, thus far, been replicated.

REM vs. Non-REM

Acting Out Dreams

Since somnambulism occurs during non-REM sleep, the patient is not considered to be acting out dreams, but dream-like mentations during sleepwalking have been observed. Thirty-eight patients underwent an interview about the frequency, time, behaviors, and mental content associated with their episodes of sleepwalking and sleep terrors and also underwent overnight video-polysomnograms. Seventy-one percent reported at least 1 dreamlike mentation associated with the sleepwalking/sleep terrors episode and their observed behavior matched what they described. Most (95%) dreamlike mentations consisted of a single visual scene, frequently unpleasant, with aggression occurring in 24% (the dreamer being the victim), misfortune in 54%, and apprehension in 84% [34].

Pillmann reports a case of complex and dramatic sleepwalking behavior in a 26-year-old adult male who tied his 4-month-old daughter to the clothesline in the attic of his house. Pillmann [35] concludes that in some cases of non-REM parasomnia, dream-like mentation may act as a bridge between psychosocial stressors and specific parasomnic behavior. Contrary to common assumptions, wakefulness, rapid eye movement (REM) and non-REM sleep may overlap to significant degrees [36].

Crime/Law

Sleep-related aggression occurs in approximately 2% of sleepwalking episodes [37]. Crimes arising from parasomnias give rise to many difficulties for forensic science and the law courts, well described in recent articles [6, 38–40].

Conclusion

Until the early nineteenth century, as in Shakespeare's MacBeth, sleepwalking was considered a mark of evil, punishment for unconfessed sin [41].

This is no longer the case; the diagnosis of schizophrenia currently carries more stigma than sleepwalking. The combination of these two ill understood conditions increases the stigma and isolation of schizophrenia patients who, perhaps because of antipsychotic treatment, also suffer from a sleepwalking disorder. Such patients may appear confused when woken from sleep; they may strike out at whomever attempts to wake them [42],

behavior that will be misinterpreted as arising from schizophrenia. Raising the antipsychotic dose in such circumstances can exacerbate the problem. The perception that objects have been displaced in the room and the attribution of that fact to malevolent forces is a common schizophrenia symptom that may be the result of sleepwalking. Acute psychosis is usually associated with stress and sleep deprivation, both triggers of sleepwalking. Antipsychotics not only may predispose to somnambulism, they also increase the risk for obesity and for sleep apnea, and for alcoholism, all of which can make sleepwalking more likely. Complex behaviors arising from the sleep period may result in violent or injurious consequences, even death. Those resulting in death may be erroneously deemed suicides [43].

Patients with schizophrenia should be questioned about family history of sleepwalking, childhood history of sleepwalking and current nighttime arousals. Reports suggestive of sleepwalking should be further investigated and, if parasomnias are present, medications need to be adjusted and appropriate treatment initiated.

Conflict of interest None.

References

1. Shatkin JP, Feinfield K, Strober M. The misinterpretation of a non-REM sleep parasomnia as suicidal behavior in an adolescent. Sleep and Breathing 6:175–179, 2002
2. Cohrs S. Sleep disturbances in patients with schizophrenia: Impact and effect of antipsychotics. Central Nervous System Drugs 22:939–962, 2008
3. Hublin C, Kaprio J, Partinen M, et al. Prevalence and genetics of sleepwalking: A population-based twin study. Neurology 48:177–181, 1997
4. Lam SP, Fong SY, Ho CK, et al. Parasomnia among psychiatric outpatients: A clinical, epidemiologic, cross-sectional study. Journal of Clinical Psychiatry 69:1374–1382, 2008
5. Kales JD, Humphrey FJ, Martin ED, et al. Clinical characteristics of patients with sleepwalking: Further studies. Sleep Research 7:192, 1978
6. Cartwright R. Sleepwalking violence: A sleep disorder, a legal dilemma, and a psychological challenge. American Journal of Psychiatry 161:1149–1158, 2004
7. Hafeez ZH, Kalinowski CM. Somnambulism induced by quetiapine: Two case reports and a review of the literature. Central Nervous System Spectrum 12:910–912, 2007
8. Juszczaka GR, Swiergiela AH. Serotonergic hypothesis of sleepwalking. Medical Hypotheses 64:28–32, 2005
9. Kawashima T, Yamada S. Paroxetine-induced somnambulism. Journal of Clinical Psychiatry 64:483, 2003
10. Jindal RD, Friedman ES, Berman SR, et al. Effects of sertraline on sleep architecture in patients with depression. Journal of Clinical Psychopharmacology 23:540–548, 2003
11. Moldofsky H, Gilbert R, Lue FA, et al. Sleep-related violence. Sleep 18:731–739, 1995
12. Dolder CR, Nelson MH. Hypnosedative-induced complex behaviours: Incidence, mechanisms and management. Central Nervous System Drugs 22:1021–1036, 2008
13. Espa F, Ondze B, Deglise P, et al. Sleep architecture, slow wave activity, and sleep spindles in adult patients with sleepwalking and sleep terrors. Clinical Neurophysiology 111:929–939, 2000
14. Pilon M, Zadra A, Joncas S, Montplaisir J. Hypersynchronous delta waves and somnambulism: Brain topography and effect of sleep deprivation. Sleep 29:77–84, 2006
15. Pilon M, Montplaisir J, Zadra A. Precipitating factors of somnambulism: Impact of sleep deprivation and forced arousals. Neurology 70:2284–2290, 2008
16. Hughes JR. A review of sleepwalking (somnambulism): The enigma of neurophysiology and polysomnography with differential diagnosis of complex partial seizures. Epilepsy Behavior 11:483–491, 2007
17. Tassinari CA, Cantalupo G, Högl B, et al. Neuroethological approach to frontolimbic epileptic seizures and parasomnias: The same central pattern generators for the same behaviours. Revue Neurologique (Paris) 165:762–768, 2009

18. Tinuper P, Provini F, Bisulli F, et al. Movement disorders in sleep: Guidelines for differentiating epileptic from non-epileptic motor phenomena arising from sleep. Sleep Medicine Reviews 11:255–267, 2007
19. Jiménez-Genchi A, Díaz-Galviz JL, García-Reyna JC, et al. Coexistence of epileptic nocturnal wanderings and an arachnoid cyst. Journal of Clinical Sleep Medicine 3:399–401, 2007
20. Pressman MR. Factors that predispose, prime and precipitate NREM parasomnias in adults: Clinical and forensic implications. Sleep Medicine Reviews 11:5–30, 2007
21. Guilleminault C, Kirisoglu C, Bao G, et al. Adult chronic sleepwalking and its treatment based on polysomnography. Brain 128:1062–1069, 2005
22. Morishita S. Clonazepam as a therapeutic adjunct to improve the management of depression: A brief review. Human Psychopharmacology 24:191–198, 2009
23. Harris M, Grunstein RR. Treatments for somnambulism in adults: Assessing the evidence. Sleep Medicine Reviews 13:295–297, 2009
24. Landry P, Warnes H, Nielsen T, Montplaisir J. Somnambulistic-like behaviour in patients attending a lithium clinic. International Clinical Psychopharmacology 14:173–175, 1999
25. Charney DS, Kales A, Soldatos CR, et al. Somnambulistic-like episodes secondary to combined lithium-neuroleptic treatment. British Journal of Psychiatry 135:418–424, 1979
26. Price LH, Charney DS, Delgado PL, et al. Lithium and serotonin function: Implications for the serotonin hypothesis of depression. Psychopharmacology 100:3–12, 1990
27. Wegener G, Bandpey Z, Heiberg IL, et al. Increased extracellular serotonin level in rat hippocampus induced by chronic citalopram is augmented by subchronic lithium: Neurochemical and behavioural studies in the rat. Psychopharmacology 166:188–194, 2003
28. Hedman C, Pohjasvaara T, Tolonen U, et al. Parasomnias decline during pregnancy. Acta Neurologica Scandinavica 105:209–214, 2002
29. Eichling PS, Sahni J. Menopause related sleep disorders. Journal of Clinical Sleep Medicine 1:291–300, 2005
30. Moline ML, Broch L, Zak R, et al. Sleep in women across the life cycle from adulthood through menopause. Sleep Medicine Reviews 7:155–177, 2003
31. Tantrakul V, Guilleminault C. Chronic sleep complaints in premenopausal women and their association with sleep-disordered breathing. Lung 187:82–92, 2009
32. Anonymous. Unquiet sleep. British Medical Journal. 281:1660–1661, 1980
33. Lecendreux M, Bassetti C, Dauvilliers Y, et al. HLA and genetic susceptibility to sleepwalking. Molecular Psychiatry 8:114–117, 2003
34. Oudiette D, Leu S, Pottier M, et al. Dreamlike mentations during sleepwalking and sleep terrors in adults. Sleep 32:1621–1627, 2009
35. Pillmann F. Complex dream-enacting behavior in sleepwalking. Psychosomatic Medicine 71:231–234, 2009
36. Mahowald MW, Schenck CH. Insights from studying human sleep disorders. Nature 437:1279–1285, 2005
37. Schenck C, Mahowald M: Long-term nightly benzodiazepine treatment of injurious parasomnias and other disorders of disturbed nocturnal sleep in 170 adults. American Journal of Medicine 100:333–337, 1996
38. Bornemann MA, Mahowald MW, Schenck CH. Parasomnias: Clinical features and forensic implications. Chest 130:605–610, 2006
39. Ebrahim IO, Fenwick P. Sleep-related automatism and the law. Medicine, Science and the Law 48:124–136, 2008
40. Pressman MR, Mahowald MW, Schenck CH, et al. Sleep-related automatism and the law. Medicine, Science and the Law 49:139–143, 2009
41. Riva MA, Sironi VA Tremolizzo L, et al. Sleepwalking in Italian operas: A window on popular and scientific knowledge on sleep disorders in the 19th Century. European Neurology 63:116–121, 2010
42. Pressman MR. Disorders of arousal from sleep and violent behavior: The role of physical contact and proximity. Sleep 30:1039–1047, 2007
43. Mahowald MW, Schenck CH, Goldner M, et al. Parasomnia pseudo-suicide. Journal of Forensic Science 48:1158–1162, 2003

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