# Chapter 5
# Sleepwalking and Its Variants in Adults

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## Introduction

Somnambulism (from the Latin *somnus* for sleep and ambulare meaning to walk around) or sleepwalking (SW) is a parasomnia that emerges as a partial arousal typically from non-rapid eye movement (NREM) 3 sleep. SW is one of a family of NREM arousal disorders (DoA) that begin as sudden partial awakenings from NREM 3 sleep [1]. These include confusional arousals, sleep terrors (STs), sleep-related eating disorder (SRED) [2, 3], sexual behaviors during sleep (sleep sex or sexsomnia) [4–6], sleep driving [7–9], and parasomnia overlap syndrome when in combination with rapid eye movement (REM) sleep behavior disorder (RBD) [10].

Parasomnias are unusual motor behaviors or experiences that occur during the entry into sleep, during sleep, or during arousals from sleep. A parasomnia becomes a sleep disorder if it is potentially injurious or causes injury to the patient or others, fragments sleep, causes daytime sleepiness or insomnia, or other adverse health effects and negative psychosocial effects.

SW was recognized by Hippocrates: "people groaning and shouting in their sleep, some who choke; others jump from their bed and run outside and remain out of their mind till they wake, when they are as healthy and sane as they were before" [11]. In the Middle Ages, SW was closely tied to religious beliefs, exciting "religious veneration and awe" when believed to be a "consequence of Divine appointment," less appreciated when thought due to "diabolical agency" [12].

Physicians of the late nineteenth century debated at length whether somnambulism, catalepsy, chorea, vertigo, or hysteria represented variants of epilepsy.

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Restricted to clinical observations alone, James Pritchard (a nineteenth century English physician) thought SW, epilepsy, nightmares, and ecstatic states were similar afflictions for "Where they do not coexist with epilepsy, they often seem to stand in place of it" [13]. Johannes Müller (a nineteenth century professor of anatomy and physiology at the University of Berlin) in 1843 wrote how a sleepwalker "rises and performs all acts of life" … and when "attended with danger, he is unconscious of it; he crosses for example, narrow planks, as a child would do, that is not aware of the danger" [14]. The advent of electroencephalography (EEG) and video-polysomnography (video-PSG) in the twentieth century has taught us that SW has a neurobiological basis and can be distinguished from sleep-related epilepsy [15, 16] and other parasomnias [17].

SW in adults has rarely been associated with acts of violence or inadvertent homicide [3, 4, 18–22], indecent exposure [23, 24], pseudo-suicide [25], or impulsive behaviors [26]. These rare occurrences have fostered much of the never-ending fascination SW has fostered in folklore, novels, operas, and film [27, 28]. Most recently, Harry Potter in the 2004 film "Harry Potter and the Prisoner of Azkaban" uses SW as an excuse for being out of bed after hours [29]. In the 2010 movie, "In My Sleep," a man who sleepwalks finds himself covered in blood after waking, with a knife at his side and the police banging at his door, and learns that the wife of his best friend was stabbed to death [30]. In a 2005 episode "Role Model" of the television series House, a woman finds she is pregnant after having sexual intercourse with her ex-husband while sleeping [31].

NREM DoA are thought to represent examples of *state dissociation*, in which the physiological markers of one sleep/wake state intrude into another [32, 33]. Sleep/wake state cycling or synchronization between the primary states of being (wakefulness, NREM sleep, and REM sleep) is often remarkably seamless and rapid, but when states oscillate rapidly, the components of one state of being may intrude or become admixed with another. When this happens, behavioral markers of waking (such as walking, talking, complex motor behaviors) may be associated with those of sleep (high arousal threshold, amnesia, automatic behavior, dream imagery) [33].

The repetitive motor behaviors of many parasomnias and some epileptic seizures may represent release or expression of central pattern generators (CPGs) during sleep [34]. CPGs are species-specific neural networks in the brain stem and spinal cord of humans, which can produce repetitive motor sequences without neocortical input. Release or lack of inhibition of CPGs during sleep is likely to permit SW, STs, confusional arousals, sleep bruxism, periodic limb movements (PLMs) during sleep, sleep-related expiratory groaning, sleep-related eating, faciomandibular myoclonus, and nocturnal frontal lobe seizures.

Ontogeny (the study of how a living organism develops from conception to birth and across its lifespan) may contribute to the appearance and/or disappearance of both normal and abnormal parasomnias in humans. Unihemispheric sleep in NREM is a phylogenetic adaptation observed in some marine mammals and birds. Sleeping with only one-half of their brain at a time allows dolphins, seals, and whales to surface to breathe. The eye contralateral to the wake hemisphere is kept open and out of the water to monitor the environment while they continue to swim, whereas the eye

contralateral to the sleeping hemisphere is closed. Mallard ducks will sleep standing in a line with a duck on each end sleeping with the eye to outside kept open to guard the flock from invaders. The sentinel ducks have more unihemispheric sleep than those who sleep in the middle of the flock, and they can react to threatening stimuli seen through the one open eye. Parasomnias in humans may represent a protective role of sleeping by sleeping with only parts of our brains at one time.

## Clinical Features of NREM Arousal Disorders in Adults

NREM DoA usually begin at the transition out of the first NREM 3 period, 60–90 min after sleep onset. Usually only one event occurs in a night, but if a second occurs, it is usually 60–90 min later and milder. An adult when SW typically (1) appears confused, disoriented, often misperceives objects and persons, perceives threats when none are present; (2) may perform complex, coordinated, and semipurposeful routine or nonsensical behaviors in inappropriate places and times; (3) is difficult to arouse from an episode, but if aroused, may recall fragmentary images; (4) exhibits signs of sympathetic autonomic activation (tachycardia, tachypnea); and (5) has limited or no recall of the events the following day.

SW episodes often begin with the individual sitting up in bed, fumbling with bedclothes, looking about in a confused manner before getting out of bed and slowly beginning to walk around [1, 35]. The eyes are usually open, often wide open with a confused "glassy" stare; whereas the eyes are typically closed during episodes of RBD [1]. The person often walks toward sound, light, or a particular room. Complex motor behaviors such as dressing or undressing, opening drawers or doors, and going to the bathroom or outside can be observed and typically vary from one event to another (i.e., not stereotyped as in epileptic seizures). Inappropriate behaviors and impaired judgment such as climbing out windows, urinating in a closet, a corner, or next to the toilet, or moving furniture are not uncommon. Visual inspection functions but the walker often misidentifies objects (e.g., lifting a glass of water at the bedside to the ear as though it was a telephone receiver). The SW episode typically ends spontaneously within several minutes with a return to sleep in the person's bed or less inappropriate places (couch, someone else's bed, outdoors).

Adults who sleepwalk often also have STs and/or confusional arousals, beginning as one and evolving to another. *Confusional arousals* in adults are characterized by moaning, whimpering, and/or incoherent vocalization; aimless thrashing, flailing, or kicking; looking "possessed," not in terror, but remaining in bed; when spoken to, acting confused and slow to respond, if at all; and exhibiting moderate degrees of sympathetic activation. *Sleep terrors* are heralded by a blood-curdling scream or cry, followed by confusion, intense vocalization, motor agitation, and severe sympathetic activation (profuse sweating, mydriasis, tachycardia, and tachypnea) [35].

Unlike children, adult sleepwalkers often recall fragments, thoughts, and images of many or most of their SW episodes, and some of their behaviors represent enactment of recent events/concerns/stimuli in their sleep environment [36]. Zadra et al.

reported 78 % of 68 adult chronic sleepwalkers remember parts of their SW episodes (16 % always, 37 % often, and 25 % sometimes) and 69 % said that fragmentary images or thoughts often or always accompanied their SW [37]. Oudiette et al. (2009) recorded overnight PSG in 38 adults with chronic severe SW and/or STs and found that (1) 71 % recalled at least one dreamlike mentation related to an in-laboratory SW or ST episode; (2) the dreamlike mentation action corresponded with some of the motor behaviors observed (reenactment); (3) 95 % of mentations were a single visual scene; and (4) mentations were frequently unpleasant, creating feelings of apprehension in 84 %, misfortune in 54 %, and aggression in 24 % (with the sleepwalker being the victim) [38]. Case reports of complex dream-enacting behaviors in chronic sleepwalkers have been reported [39].

## Epidemiology and Risk Factors for Sleepwalking

SW is most common in children and decreases with age. A longitudinal study of child development reported an overall prevalence of 40 % for STs and 14.5 % for SW in children aged 2.5–6 years [40]. The peak incidence of STs is between 5 and 7 years, whereas SW peaks later between 8 and 12 years [41]. An earlier study found 40 % of children (ages 6–16 years) have at least one episode of SW (most between ages 11 and 12 years) but only 2–3 % have more than one per month [42]. The majority of children who sleepwalk stop sleepwalking by the age of 13 years, but SW persists in 24 % of frequent sleepwalkers [42].

SW and other DoA is far less common in adults [41] and epidemiological studies even fewer. Forty to 60 % of SW episodes after late adolescence occur in individuals who had them in childhood, sometimes recurring after having none for several years [42, 43]. A telephone survey of a large representative sample of 4,972 adults in the UK found that 2 % reported SW, 2.2 % STs, and 4.2 % confusional arousals [41]. SW was reported in 3.9 % of men and 3.1 % of women of 11,220 subjects in the Finnish Twin Cohort, but occurred weekly in only 0.4 % of either gender [44]. Of the adults who did sleepwalk, 89 % of men and 84 % of women reported SW in childhood. The proportion who reported never having walked in their sleep in childhood and did so as adults was 0.6 % in both men and women.

A large prospective cross-sectional study found 29 % of 19,136 US adults ($\geq$ 18 years) reported a lifetime history of "nocturnal wandering" (NW), 3.6 % within the previous year [45]. Only 0.2 % had $\geq$ 1 episode per week, 1 % $\geq$ 2 per month, and 2.6 % 1–12 across the year. NW decreased with age, excepting those who report $\geq$ 1 episode per week. NW was not associated with gender. Twenty-six percent reported having had NW as a child or adolescent but without any episodes within the prior year. Individuals who self-reported NW in the previous year were twice as likely to report a family history of NW compared with those who denied NW (30.5 % vs. 17.2 %, odds ratio [OR] 2.12). NW when present that year was most often chronic: 7.2 % reported episodes had been present for less than 6 months, 5.8 % for 6–12 months, 6.2 % for 1–5 years, and 80.5 % for > 5 years.

Logistic regression analysis demonstrated that the risk for frequent NW (≥ 2 episodes per month) increased significantly if the individual who also reported obstructive sleep apnea (OSA) syndrome (OR 3.9), circadian rhythm sleep disorder (OR 3.4), insomnia disorder (OR 2.1), alcohol abuse or dependence (OR 3.5), major depressive disorder (OR 3.5), obsessive-compulsive disorder (OR 3.9), or use of over-the-counter sleeping pills (OR 2.5) or selective serotonin reuptake inhibitor antidepressants (OR 3.0). They found a higher risk for having at least 1 NW episode in the previous year in individuals sleeping less than 7 h per night after adjusting for possible confounding factors such as age, sleep, and mental disorder. The association between NW and major depressive disorder or obsessive compulsive disorder could not be attributed to the use of psychotropic medication(s).

Another recent large prospective cross-sectional study of 1,000 Norwegian adults (≥ 18 years) found that 22.4 % reported a history of SW but only 1.7 % reported SW within the past 3 months [46]. The lifetime and current prevalence of STs was 10.4 % and 2.7 %, confusional arousal 18.5 % and 6.9 %, self-injury during sleep 4.3 % and 0.9 %, injuring someone else during sleep 3.8 % and 0.4 %, sexual acts during sleep 7.1 % and 2.7 %, and sleep-related eating 4.5 % and 2.2 %, respectively [46]. About 12 % reported ≥ 5 parasomnias. The lifetime prevalence of SW among a random sample of 276 young adult Nigerians was 23 % and an adult prevalence of 1.42 % [47]. The prevalence of SW in > 1,000 Brazilian adults (ages 20–80 years) in 2007 was 2.8 % [48].

## Familial or Genetic Predisposition to NREM Arousal Disorders

A family history is one of the strongest risk factors for DoA in children and adults. The first (and perhaps the most spectacular) report of familial SW by Clerici in 1930 described a family of six members (husband, wife (who was also his cousin), and their four children), all of whom gathered one night at 3 AM around a table in their servants' quarters [49]. A landmark study by Kales et al. (1980) found 80 % of 25 sleepwalkers and 27 with STs had one or more relative who was affected by SW, STs, or both [50]. The Finnish Twin Cohort mentioned earlier found no difference in the concordance rate for SW between 1,045 monozygotic or 1,899 dizygotic twin pairs with concordance rates of 0.55 and 0.35 for monozygotic and dizygotic childhood twin pairs and 0.32 and 0.06 for monozygotic and dizygotic adult pairs, respectively. Genetic influences predicted that 66 % of the total phenotypic variance for SW in men and boys, 36 % in women, and 57 % in girls [44].

Guilleminault et al. have long emphasized how underlying primary sleep disorders (most often obstructive sleep-disordered breathing (OSDB), less often restless legs syndrome (RLS)) trigger SW or other DoA in children and adults; treating them often eliminates the DoA [41, 51–54]. Most recently, a report found that craniofacial risk factors for sleep-disordered breathing (SDB; particularly maxillary and/or mandibular deficiencies) were often present in families of adults referred for SW

[55]. They argue that these inherited craniofacial abnormalities predispose to OSDB and lead to sleep fragmentation and DoA.

A prospective case-control study found a higher incidence of parasomnias in children with idiopathic epilepsy compared with their siblings or healthy controls [56], but parasomnias were not more common in a prospective study of adults with a wide variety of different epilepsies and seizure types compared with healthy controls [57]. However, DoA and sleep-related bruxism is more common in patients and their relatives with nocturnal frontal lobe epilepsy (NFLE) [58]. The lifetime prevalence of DoA was 6-fold higher in patients with NFLE and 4.7 times greater in their relatives compared with controls.

Lecendreux et al. (2003) performed HLA-DQB1 typing in 60 Caucasian subjects with SW and their families and 60 ethnically-matched controls without any diagnosed sleep disorder [59]. Licis et al. (2011) collected DNA samples and performed parametric linkage analysis on 9 affected and 13 unaffected family members in a 4-generation SW family. They found that (1) 35 % of the sleepwalkers tested positive for DQB1*0501 compared with 13.3 % of the controls (OR 3.5); (2) SW was inherited as an autosomal dominant disorder with reduced penetrance and the genetic locus for it was at chromosome 20q12-113.12; and (3) SW in a first degree relative increased the chances of developing this disorder by a factor of 10 [60]. More research is needed to confirm whether SW is a mono- or polygenetic inherited disorder and to confirm the connection with inherited craniofacial abnormalities.

## Factors Which Predispose, Prime, or Precipitate Sleepwalking

SW or STs in adults is most likely to occur when priming factors (such as sleep deprivation or situational stress) are coupled with provoking triggers (noise, light, sound) in individuals who have a familial or genetic predisposition for it [61]. The frequency of DoA episodes can be lessened by identifying and eliminating these. Genetic susceptibility or family history predispose to DoA [44, 50, 55, 60, 62, 63]. *Priming* factors include conditions and substances that increase NREM 3 sleep or make arousal from sleep more difficult: sleep deprivation/restriction, alcohol, certain medications, situational or emotional stress, and fever [61, 64] Noise, touch, or forced awakenings from NREM sleep can precipitate SW in predisposed adults [43].

New onset or recurrence of SW in adults warrants consideration of other primary sleep disorders, including OSDB, RLS, periodic limb movement disorder (PLMD), RBD, jet lag, and/or shift work [52, 55, 64–68]. Other stressors that can precipitate SW in predisposed adults are stressful life events, changes in sleep environment, family or workplace conflicts, emotional stress, infections, extreme fatigue, pain, or changes in exercise pattern [61, 69–73].

A plethora of small and/or single-case reports demonstrate that SW and its variants can be triggered by nonbenzodiazepine hypnotics, neuroleptics, antidepressants, lithium, sodium oxybate, and beta-blockers in predisposed individuals [7, 9, 61, 74–96]. In summary, SW in adults is most likely to occur when priming factors

(such as sleep deprivation or situational stress) are coupled with provoking triggers (noise, light, sound) in individuals who have a familial or genetic predisposition for it [61]. When these occur, a "perfect storm" for SW occurs [97]. Box 5.1 summarizes factors that predispose, prime, or perpetuate DoA.

## Adult Sleepwalking Common in Psychiatric Populations

The majority of adult sleepwalkers does not have a Diagnostic and Statistical Manual of Mental Disorders (DSM)-based psychiatric disorder or highly disturbed personality traits, [5, 52, 61, 97, 98] but SW and/or STs are common in certain adult psychiatric populations [99]. A coexisting bipolar or adjustment disorder increased the risk for confusional arousals 13.0 and 3.1 times, respectively, among 4,972 UK adults [41]. Major depressive or obsessive compulsive disorders were major risk factors for $\geq 2$ episodes of NW in US adults [45]. Panic or anxiety disorders, simple phobias, or suicidal thoughts were more likely to be reported in a case-control study of 21 adolescents who reported SW and/or STs in the prior year compared with 30 healthy controls [100]. SW (along with trance, possession, and paranormal experiences) were more frequently reported in patients with dissociative disorders than healthy controls [101]. Adult-onset SW was thought to contribute to delusions, aggression, and accidental death in some patients with schizophrenia [99].

Nonbenzodiazepine hypnotic (with or without concomitant psychotropic) use may contribute to the higher incidence of DoA in patients with psychiatric problems. Five percent of 255 Taiwanese adults (average age 42 years, 54 % females) taking zolpidem (average dose 10 mg) reported SW and/or amnesic sleep-related behavioral problems [102]. All had concomitant psychiatric problems, including schizophrenia, anxiety, and affective or adjustment disorders. Complex sleep-related behaviors among these patients included watching television, telephone use, eating, or conversation with their family. A recent prospective cross-sectional study of 66 psychiatric patients with childhood-onset (56 %, $n = 37$) or adult-onset (44 %, $n = 29$) SW found that lifetime use of zolpidem was associated with adult-onset SW [89]. Adult-onset sleepwalkers had more frequent SW episodes and sleep-related eating than child-onset walkers and were 5.4 times more likely to complain of frequent insomnia. SW recurred in only 40 % of childhood-onset walkers when comorbid psychiatric problems developed.

## Sleepwalking Variants

Adults while sleeping rarely exhibit episodes of eating, drinking, driving a motor vehicle, or injure or murder individuals who attempt to help them. Often these complex motor behaviors emerge from NREM 3 sleep.

## *Sleep-Related Violent Behavior*

The most common sleep disorders that can result in injurious or violent behaviors are DoA and RBD; less often OSDB, nocturnal seizures, narcolepsy with cataplexy, psychogenic or dissociative disorders, and malingering [18, 20, 27, 103]. Violent behavior during sleep (VBS) was self-reported by 1.6 % of 19,961 adults (aged 15 years or older) from multiple European Union countries [20]. During VBS episodes, 79 % report vivid dreams and 31 % hurt themselves or someone else. The greatest risk factor for VBS is to have a family member with VBS (OR 9.0), although SW (OR 2.0) and STs (OR 4.2) in the family increased the risk of VBS [20]. Only 12 % of people who had VBS had consulted a physician.

Violent behavior during SW tends to occur when the episode is already underway and the individual is approached by another person (often one trying to help), occurs more in males, and is associated with more stressors, disturbed sleep, and excessive use of caffeine and drug abuse [21, 27, 39, 104]. VBS attributed to DoA in 32 cases from the medical or legal literature were present in 100 % of the confusional arousal, 81 % of STs, and 40–90 % of the SW episodes [21].

Dramatic reports of somnambulistic violent behavior and/or homicide still gain considerable attention in the media [22] and often prompt a referral to sleep specialists [39, 105]. VBS during an episode of SW or STs have resulted in attempted or completed homicide, suicide, or inappropriate sexual behaviors. Clinical features of historical cases of VBS attributed to DoA are summarized in Box 5.2.

A number of criminal cases have claimed that SW or its variant was induced by alcohol intoxication [90]. Alcohol-induced SW as a criminal defense has been based upon the concept that alcohol increases NREM 3 sleep. Some studies have shown that alcohol modestly increases the percent of NREM 3 sleep in the first 2–4 h of a night [90]. However, the amount and percentage of NREM 3 sleep is often reduced or absent in those who regularly abuse alcohol. It is possible for an individual to perform violent acts without conscious awareness while sleeping, but whether SW occurred at the time of the crime remains uncertain. Guidelines developed for determining a putative role of sleep disorder in a violent act are summarized in Box 5.3 [106].

## *Sleep Driving and Drug-Impaired Driving Overlap Syndromes*

A rare variant of SW in adults is driving a motor vehicle for long distances without conscious awareness while sleeping (so-called sleep driving) [7]. Episodes of driving a motor vehicle have been rarely reported in individuals with SW. These must be distinguished from the far more common reports of adults driving under the influence of nonbenzodiazepine hypnotics often coupled with other substances. Sleep driving in a sleepwalker is very rare: the US Federal Drug Administration (FDA) found only 14 cases of sleep driving between 1992 and 2006, which were *not* due to the use or misuse of nonbenzodiazepine hypnotics.

Bizarre, dangerous, or socially inappropriate behaviors have been associated with the more often inappropriate use or abuse of nonbenzodiazepine hypnotics (zolpidem, zopiclone, or zaleplon) [7, 9, 107]. Alcohol, a Z-drug, or other substances in patients with a history of SW can be a priming factor for SW [86] and zolpidem at higher doses is more likely to cause SW [108]. Nevertheless, these so-called "Z-drugs" can precipitate a sleepwalker to walk. The majority of media and police reports of "sleep driving" occur in individuals with no prior history of DoA. Impaired driving (or inappropriate eating or sexual behavior) associated with sedative/hypnotic use is often misattributed to SW (and better called drug-impaired driving). Zolpidem was the major intoxicant in 2.3 % of 8,121 driving while intoxicated (DWI) arrests in Wisconsin from 1999 to 2005; higher than prescribed levels were found in 35 % cases and 23 % of the arrests were between 0800 and 1200 hours.

Z-drug-impaired driving bears only superficial resemblance to the behavior presented as characteristic of "true" sleep driving [109]. Sleepwalkers when sleep driving without medication, alcohol, or other substance use are able to stand and walk unaided but are unable to interact with police. However, drivers who have been prosecuted for driving under the influence of Z-drugs (1) appear drowsy, tired, confused, and disoriented and have unsteady gait, slowed speech, impaired coordination, and short-term memory loss [7]; (2) are often severely physically impaired, unable to stand up or maintain balance, but able to understand or interact with police; (3) have often failed to take the medication at the correct time or remain in bed for sufficient time and/or have combined Z-drugs with other central nervous system depressants and/or alcohol; and (4) often have blood levels of Z-drugs that exceed therapeutic ranges [7, 8].

## *Nocturnal Eating and Sleep-Related Eating Disorder*

SRED was originally described in 1991 [110], but diagnostic criteria for it continue to evolve [111–116]. It is a parasomnia that is characterized by recurrent episodes of involuntary or compulsive eating after awakening typically from NREM 2 or 3 sleep, which have adverse health consequences [111–113, 117] SRED is a dysfunctional nocturnal eating (NE) behavior diagnosed if a patient reports one or more of the following: (1) ingestion of unusual, inedible or toxic substances; (2) difficulty falling back to sleep or report nonrestorative sleep; (3) sleep-related injury or potentially injurious behaviors; (4) morning anorexia; or (5) adverse health consequences.

Sleep clinicians have been asked to distinguish SRED from nocturnal eating syndrome (NES), clinical features of which are summarized in Table 5.1. However, the distinction is often near to impossible because of symptom overlap between them. NES is a primary eating disorder characterized by evening hyperphagia ($\geq 25$ % of daily caloric intake after the evening meal and/or $\geq 2$ nighttime awakenings with ingestions per week), frequent nocturnal awakenings from sleep with conscious compulsive eating, morning anorexia, and a circadian delay in meal timing [118, 119].

**Table 5.1** Clinical features that may help distinguish sleep-related eating disorder from nocturnal eating syndrome

| Sleep-related eating disorder (SRED): a parasomnia | Nocturnal eating syndrome (NES): a primary eating disorder |
|---|---|
| Recurrent episodes of involuntary eating and drinking during the main sleep period associated with little or no awareness of these | Evening hyperphagia: consuming $\geq 25\,\%$ of daily food intake after the evening meal and/or awakening to eat $\geq 2$ nights per week for $\geq 3$ months |
| Consumption of inedible or toxic substances | And 3 of the 5 features<br>Morning anorexia<br>Strong urge to eat between dinner and sleep onset and/or nocturnal awakenings<br>Insomnia $\geq 4$ nights per week<br>Belief that eating is necessary to fall asleep or return to sleep<br>Mood frequently depressed or mood worsens in the evening |
| Dangerous behaviors performed in the pursuit of food or while cooking it | Awareness and recall of the evening and nocturnal eating episodes |
| Eating foods in combinations, amounts, and/or raw states they would have never been prepared or eaten when awake | |
| May exhibit dangerous and/or atypical behaviors when cooking (stove left on, kitchen a mess) | Associated with significant distress and/or impaired functioning |
| Can cause morning anorexia, weight gain, and/or other adverse health consequences from this | |
| Adverse health consequences from recurrent binge eating of high caloric food | Not secondary to substance abuse/dependence, medication(s), a general medical, and/or psychiatric disorder |
| Not associated with anorexia nervosa, bulimia, binge-eating, or other daytime eating disorders | |

NE is associated with obesity, binge eating, and psychological distress [120]. A recent study by Vinai et al. (2012) had eating disorder and sleep specialists evaluate 28 consecutive adults (mean age $44.5 \pm 12.5$ years, 57 % females) with SRED [111]. They found 79 % also met diagnostic criteria for NES [111]. All of these patients with SRED also complained of evening hyperphagia, morning anorexia, insomnia, and mood disorders. Video-PSG findings did not distinguish between groups.

Three case series have reported comorbid SW in 48–65 % of patients with SRED [110, 114, 116]. SW without SRED may precede SRED and then it often becomes the predominant SW behavior [110]. Amnesia for SRED episodes most often occurs with concomitant sedative-hypnotic use (especially zolpidem). Several case series or single-case reports describe SRED provoked by sedative-hypnotics or other medications, most often zolpidem, but also zopiclone, zaleplon, quetiapine, risperidone, olanzapine, and sodium oxybate [87, 121–127]. SRED is associated with psychiatric disorders, especially depression and dissociation.

NE and SRED are very common in patients with RLS [128, 129]. Thirty-three percent of 100 patients with RLS endorsed symptoms of SRED compared with 1 % of 100 age-matched normal controls [129]. A prospective study by Howell and Schneck (2012) evaluated how often 88 patients presenting with RLS and 42 with psychophysiological insomnia reported either NE or SRED [129]. They found that (1) 61 % of the RLS and 36 % of SRED patients complained of NE compared with 12 % of psychophysiological insomnia patients; (2) NE could not be attributed to arousal because the patients with insomnia were more likely to report prolonged nocturnal awakenings (93 %) than the patients with RLS (64 %); (3) the frequency of amnestic SW and SRED was far greater among RLS patients taking sedative-hypnotics than those with insomnia (80 vs. 8 %, respectively); and (4) the frequency of NE decreased from 68 to 34 % after the RLS patients were treated with dopaminergic agents. They concluded that NE is common in RLS and often lessens with dopaminergic therapy. Amnestic SRED and SW occur primarily when RLS patients take sedative-hypnotics. Most of the few video-PSG studies on SRED have been done on drug-free subjects. Vertugno et al. (2006) recorded 45 episodes of NE in 22 of 35 consecutive drug-free patients and found that (1) eating episodes always occurred after *complete* awakenings from NREM 1, 2, or 3 sleep (and REM sleep in 1 patient) with an EEG background consistent with normal wakefulness; (2) patients interviewed during the eating episodes were fully conscious, remembered the events the following morning, and reported a compulsion to eat (but denied feeling hungry or thirsty); (3) awakenings were not closely related to PLMs or respiratory-related arousals; and (4) the mean time delay from awakening to food intake was 7 min (range 0.3–81 min), eating duration 6 min (0.4–20 min), and return to sleep after returning to bed was 14 min (0.5–107 min). Elevated PLM indexes in 63 % and RLS dyskinesias in 14 % (and SRED are particularly common in patients with RLS) of the patients were observed. A peculiar video-PSG feature in 29 (83 %) of their patients was periodic recurring chewing and swallowing movements throughout all stages of sleep, but especially NREM 1 and 2. It resembled rhythmic masticatory muscle activity (RMMA) seen during sleep in patients with sleep bruxism (and less often normal controls). Half of these oral movements were linked to EEG arousals and often existent with PLMs. The authors speculated that the high prevalence of RMMA and PLMs during sleep in patients with SRED coupled with compulsory food-seeking behavior and reported efficacy of dopaminergic medications argue for a dopaminergic dysfunction underlying the pathogenesis of SRED.

A case-control video-PSG study by Brion et al. (2012) compared clinical histories and PSG findings in 15 patients with SRED, 21 sleepwalkers, and 20 age- and sex-matched healthy volunteers [112]. They found that (1) patients with SRED were mainly women, had disease onset in adulthood, suffered nightly episodes and insomnia, and had more frequent eating problems in childhood and higher current anorexia scores than sleepwalkers and controls; (2) unlike controls, 66 % of patients with SRED had a past or current history of SW; (3) SRED episodes typically occurred during the first half of the night; (4) SRED patients had frequent arousals from NREM 3 (not seen in controls); (5) SRED patients had higher awareness during their parasomnias than sleepwalkers; and (6) only 10 % of sleepwalkers ate during

their SW episodes. On video-PSG, eating episodes occurred mostly within 1 min after awakening from NREM 2 ($n = 9$) or NREM 3 ($n = 6$) sleep. The frequencies of RLS, PLM, and sleep apnea were similar across the three groups. The authors concluded that patients with SRED share several clinical commonalities with sleepwalkers (although their level of awareness is higher) with having prior or current eating behavior problems.

Treatment strategies for SRED include eliminating medications that contribute to it and correcting comorbid sleep disorders (e.g., RLS and rarely OSA). The majority of patients with drug-induced SRED improve. If symptoms persist, the patients, especially those with RLS, should consider dopaminergic agents (such as pramipexole) [130, 131]. SRED in some patients with SW were controlled with clonazepam [110]. Preliminary studies show that topiramate may be effective, but larger randomized controlled trials are needed [132–134].

## *Parasomnia Overlap Disorder and Status Dissociatus*

Parasomnia overlap disorder (POD) refers to a particularly rare sleep disorder characterized by the combination of injurious SW, STs, and RBD [5, 135]. The original case series of 33 patients by Schenck et al. [5] reported two subgroups: idiopathic ($n = 22$, mean age $9 \pm 7$ years) and symptomatic ($n = 11$, mean age $27 \pm 23$ years). POD was regarded as symptomatic when parasomnia began with a neurologic (e.g., narcolepsy, brain tumor, multiple sclerosis) or psychiatric disorder (major depression, schizophrenia, post-traumatic stress disorder, or chronic ethanol/amphetamine abuse and withdrawal). Little has been published since on POD: clinical and video-PSG findings of POD in 40-year-old woman following an acute inflammatory encephalitis where neuroimaging showed a lesion in the pontine tegmentum as a possible anatomic cause for it [136] and two small case reports of POD with sexual behaviors during sleep [10, 137]. These describe persistence of muscle tone during REM sleep and sudden transitions from NREM 3 sleep to partial wakefulness with accompanying complex motor behaviors. Parasomnia overlap syndrome may be found in some patients with Parkinson's disease (PD). A recent prospective prevalence study found that 36 (9 %) of 417 patients with PD reported SW, adult-onset SW in 22 (5 %) patients; sleepwalkers were more likely to have higher scores on a RBD- validated questionnaire, hallucinations, and nightmares [138].

Status dissociatus (SD) is a condition in which sleep/wake state differentiation is completely uncertain [139]. SD has been observed in patients with fatal familial insomnia [140–144], Morvan's syndrome [145, 146], and perhaps delirium tremens. Isolated case reports of its occurrence in individuals with multiple system atrophy, brainstem lesions, or Guillain–Barre syndrome have been reported [147–149]. Complex motor behaviors emerge from indeterminate EEG and other biophysiological markers of sleep and wake. A most interesting paper by Stamelou et al. (2012) argues that the twilight state of paradoxical unresponsiveness and complex nonepileptic repetitive stereotyped perioral, eye, and limb movements are seen in the majority of patients with anti-N-methyl-D-aspartate-receptor encephalitis [150].

### *Sleep-Related Sexual Behaviors*

Sleep-related sexual behavior (SRSB) is inappropriate sexual behavior occurring without conscious awareness when sleeping [4, 10, 137, 151–153]. Alves et al. (1999) reported a 27-year-old man with a history of SW since the age of 9 years. Sleep-related violent behavior began at age 20 years and amnestic episodes of complete sexual orgasm with his wife beginning at age 23 years. A literature review in 2007 detailed 31 cases (mean age 32 years, 81 % males) from sexual vocalizations or body movements, fondling, masturbation, sexual intercourse (with or without orgasm), and sexual assault/rape. PSG recorded in 26 (35 % for forensic concerns) captured sexual vocalization (moaning) from NREM 3 sleep in 3 and sexual intercourse provoked by the bed partner from NREM 1/wakefulness in one. A video-PSG study of three men with SRSB found OSDB in one, DoA in another, and RBD in the third [151]. SRSB with video-PSG was recently reported in a 60-year-old woman with parasomnia overlap syndrome [10]. Diagnosing SRSB especially when forensic issues are concerned requires corroborated history, video-PSG with extended EEG, and electromyography (EMG) montages (to identify other sleep disorders such as OSDB, sleep-related seizures, RBD, POD, and psychogenic or dissociative disorders), neuropsychological assessment (primarily to identify the presence of psychiatric comorbidities) and urine drug screen.

## Indications for Video-Polysomnography in Adult Sleepwalkers

Comprehensive in-laboratory video-PSG is not routinely indicated for "typical" SW or STs in prepubertal children [154–157]. However, video-PSG is usually warranted to evaluate parasomnias in older adolescents or adults that (1) begin or recur in adulthood; (2) occur more than 2–3 times per week; (3) are potentially injurious or have caused injury to the patient or others; (4) are accompanied by symptoms suggestive of SDB, PLMD, suspected or known epilepsy, and/or excessive daytime sleepiness; or (5) could be seizure-related but the initial clinical evaluation and a standard EEG was inconclusive [154]. Video-PSG is occasionally requested to evaluate forensic cases where parasomnias and/or other sleep disorders or drugs are a contributing factor [4, 19, 61, 106].

## Video-Polysomnographic Features of Disorders of Arousal

Unfortunately, only a third of patients with paroxysmal nocturnal events will have one of their habitual events on a single night of in-laboratory video-PSG [158, 159]. Aldrich et al. (1991) found that 1–2 consecutive nights of video-PSG provided valuable diagnostic information in 69 % of 41 patients whose paroxysmal motor behaviors were "prominent," 41 % of 11 referred for "minor motor activity in sleep," and 78 %

of 36 patients with known epilepsy [158]. Video-PSG was diagnostic in 65 % and "helpful" in another 26 % of 100 consecutive adults referred for frequent sleep-related injuries, identifying DoA in 54, RBD in 36, sleep-related dissociative disorders in 7, nocturnal seizures in 2, and OSA in 1 [98].

V-PSG recordings confirm that the majority (> 90 %) of DoA arise from NREM 3 sleep (occasionally from NREM 2 sleep), but not always in the first third of the night [160, 161]. Occasionally, a sudden run of rhythmic high-voltage delta EEG activity lasting 10–30 s may herald the onset of a DoA episode. This EEG pattern is called hypersynchronous delta activity (HSD). Initially misconstrued as diagnostic for DoA events, it is now regarded as a nonspecific EEG arousal pattern, observed in healthy controls during spontaneous arousals from NREM 3 sleep or in arousals from NREM sleep due to obstructive respiratory events.

Onset of a DoA on video-PSG is typically heralded by the abrupt onset of tachycardia (severity of it varies: ST > SW > confusional arousal). Tachycardia accompanies NFLE seizures, but change in heart rate is often absent in RBD events. The EEG following onset of a DoA most often shows either high-amplitude delta intermixed with beta or alpha activity or hypersynchronous theta or delta activity [162]. Often, muscle artifact obscures part or most of the DoA event; worse yet, the patient may pull off the electrodes and run from the bed. Less often, scorable epochs of NREM 2 sleep or repeated microsleeps are observed. Figure 5.1 shows the onset of a confusional arousal from NREM 3 sleep. Wakefulness or REM sleep should prompt consideration of other parasomnias.

Sleep researchers from Montreal have published an elegant series of experimental studies evaluating how to increase the likelihood of recording a typical SW or ST event in a single night of in-laboratory video-PSG [69, 163, 164]. Twenty-five hours of total sleep deprivation in 40 adult sleepwalkers increased the frequency and complexity of SW events recorded in the laboratory, resulting in $\geq 1$ event in 90 % of the patients and none of the controls [163]. Many of the DoA behaviors recorded were mild: playing with the bed sheets or electrodes, turning and resting on one's hands while staring about looking confused, resting on one's knees, or trying to get out of the bed [36]. To ensure 25 h of total sleep deprivation, subjects were asked to arrive in the laboratory at their customary bedtime but remain awake until 1 h later than their usual wake time.

To further provoke DoA events, 10 adult sleepwalkers and 10 controls underwent two consecutive nights of video-PSG. After a normal night of baseline recording in the laboratory, patients and controls were deprived of sleep for 25 h and then presented with recurring 3-s blasts of 1000 Hz pure sounds in ascending intensities of 10 dB from 40 dB to 90 dB via earphones with a minimum of 1 min between two stimuli during NREM 3 sleep and the first and second NREM–REM sleep cycles. Auditory stimuli were presented in the targeted sleep stage after at least 1 min of stable EEG and EMG until an EEG arousal, a behavioral episode, or a maximum of six auditory stimuli was reached [36]. The majority of DoA events occurred from NREM 3 sleep, a few from NREM 2 sleep, and none from REM sleep. Using these techniques, the investigators found that they could trigger 1–3 SW events in 100 % of their subjects (and none of their controls). The mean intensity of auditory

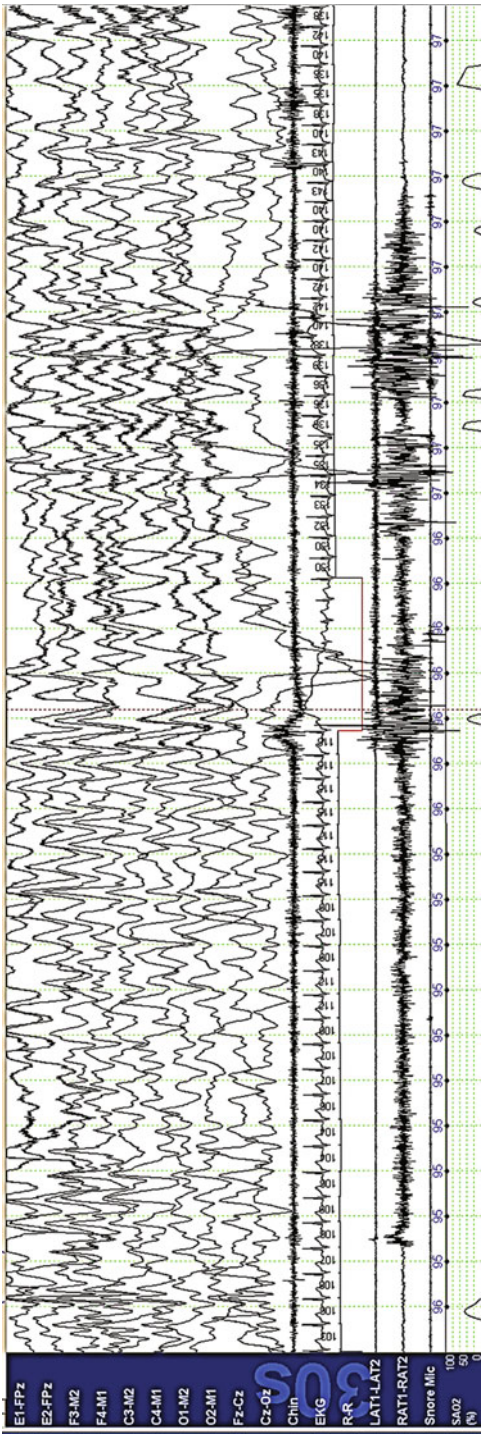


**Fig. 5.1** Confusional arousal from NREM 3 sleep in a 6-year-old child (note the tachycardia)

stimulus needed to induce DoA episodes was similar to that needed to induce non-DoA arousals in sleepwalkers and controls (approximately 50 dB). These findings suggest that sleepwalkers are neither more easy nor more difficult to awaken from NREM 3 sleep than controls, but the sleepwalkers suffer from an atypical and distinct arousal reaction [36, 69].

Most studies have failed to find meaningful or consistent differences in sleep macroarchitecture in children or adults with DoA, except for a greater number of arousals selectively from NREM 3 sleep in those with DoA, even on nights without SW/STs [69, 159, 165–167]. Sleep researchers have found differences in sleep microarchitecture and EEG power in adults with SW or STs compared with controls: a slower decay of EEG delta power of NREM 3 sleep across recurring cycles of NREM sleep and nonspecific alterations in cyclic alternating pattern during NREM sleep consistent with increased NREM 3 sleep instability [51, 168, 169]. More work is needed to see if individuals with DoA can be identified by abnormalities in their sleep microarchitecture.

## Differential Diagnosis of Sleepwalking in Adults Usually Requires Confirmation by Video-Polysomnography

Video-PSG is usually required to confirm the particular parasomnia in adults with frequent NW and identify if there are other primary sleep disorders contributing to it. The differential diagnosis for NW in adults is summarized in Box 5.4. Most often, DoA in adults needs to be distinguished from sleep-related hypermotor seizures, RBD, pseudo-RBD due to severe OSDB, and rarely dissociative events or malingering.

The clinical features of sleep-related hypermotor seizures are summarized in Box 5.5. Figure 5.2 shows an example of a nocturnal frontal lobe seizure unexpectedly recorded in a 16-year-old patient, which began from NREM 3 sleep. Longer lasting nocturnal frontal lobe seizures can lead to "episodic nocturnal wandering," but more often, postictal wandering is associated with seizures that are temporal lobe in origin [170–174]. Nocturnal temporal lobe seizures tend to be *less* frequent, do *not* cluster, and usually do *not* have the hyperkinetic motor activity of NFLE.

RBD is usually identified by the presence of REM sleep without atonia with or without RBD behaviors. The timing of RBD episodes is quite different from DoA. RBD episodes usually appear in the first 90 min after sleep onset, typically last 1–5 min, and recur 3–5 times at 90–120 min intervals across an entire night of sleep during recurring periods of REM sleep. As opposed to DoA, patients with RBD are easily aroused from an event. Once aroused, they are able to recount dreams (if not too demented) that correspond to the observed behaviors [175, 176]. Their eyes are typically closed during events. Their heart rates do *not* increase during RBD events (perhaps reflecting loss of sympathetic autonomic regulation).

RBD motor behaviors can be simple (talking, shouting, excessive jerking of limbs or body) or complex (arm flailing, slapping, kicking, sitting up, leaping from bed, running, crawling, gesturing, swearing) [135]. Because many of the RBD motor

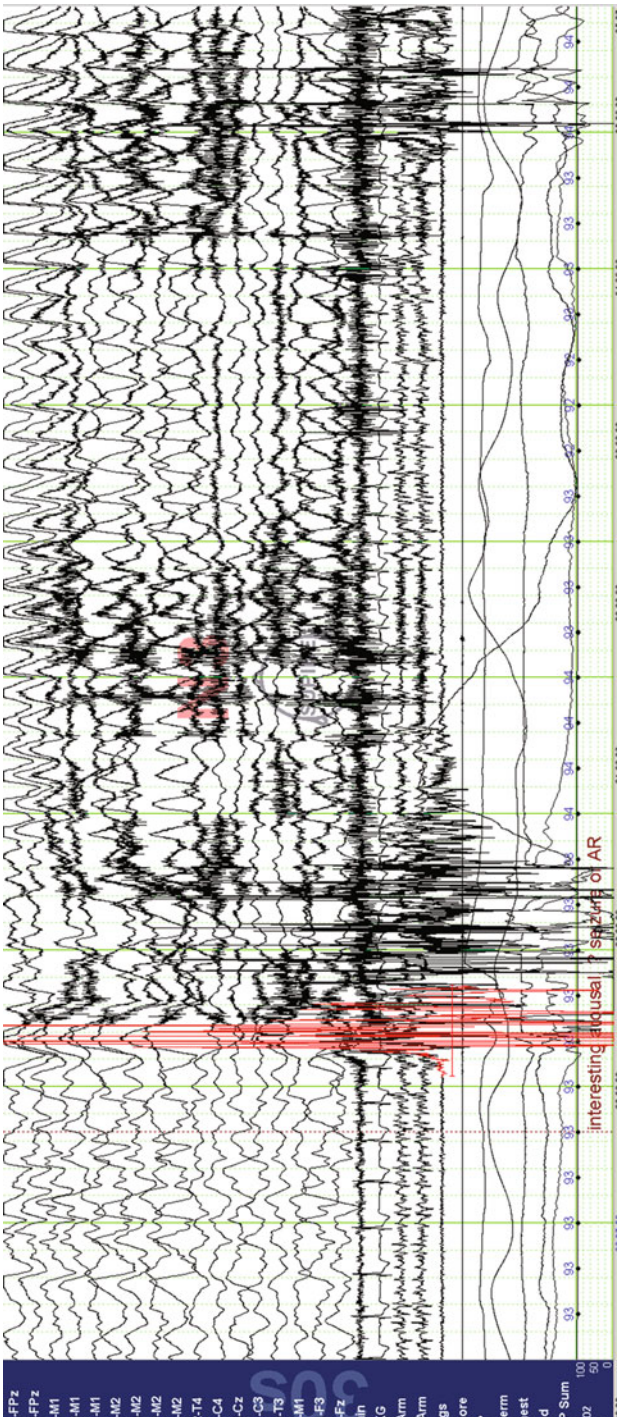


**Fig. 5.2** Stereotyped rhythmic activity accompanied by hypermotor leg movements, chewing, vocalization, and complete awakening in a 16-year-old patient with developmental delay

and/or vocal behaviors usually last only a few seconds, we have found it particularly useful to review carefully the epochs of REM sleep in the video-PSG when the excessive phasic motor activity is observed, confirming clinical manifestations of RBD that are easily missed. Figure 5.3 shows a 60-s epoch recorded on a video-PSG in a 79-year-old man with severe OSDB and occasional dream enactment behavior. Note the excessive tonic activity in the chin EMG, excessive phasic activity in the leg EMG channels, atrial fibrillation in the electrocardiogram (EKG), and an obstructive apnea that lasts for 31 s and causes desaturation to 64 % but no arousal.

Violent behaviors from any stage of sleep can be observed in patients with severe OSA. In 2005, Iranzo et al. reported 16 adults who presented with dream-enacting behaviors and unpleasant dreams [177]. Severe OSA (mean apnea–hypopnea index (AHI) 68 per hour) but no REM sleep without atonia was found on video-PSG. Abnormal sleep behaviors occurred only during apnea-induced arousals. Continuous positive airway pressure (CPAP) therapy eliminated the abnormal behaviors, unpleasant dreams, snoring, and daytime hypersomnolence. We recently recorded video-EEG for VBS in a 45-year-old boxer in whom apnea triggered recurring violent arousals from NREM 2 sleep in which the 45-year-old traumatized boxer who would assume boxing postures, loud vocalization, and fierce expression, but he regained consciousness within seconds and the EEG within 1 min was consistent with wakefulness (Fig. 5.4 a and b).

## Treatment Strategies for Adult Sleepwalkers

Treatment of SW in adults (summarized in Box 5.6) consists of reassurance, education of the patient and family, setting up a safe sleep environment, and reducing or avoiding priming or precipitating factors [178, 179]. Identifying and treating other primary sleep disorders (OSDB, RLS) may lessen or eliminate DoA. A prospective case-control study of 50 young adults with chronic SW and compared with an equal number of age-matched controls found that many of the sleepwalkers also had OSDB [52]. Treating them with CPAP or surgery controlled their SW (whereas benzodiazepines or psychotherapy did not). Nonadherent CPAP patients continued to have episodes of SW.

Pharmacotherapy may be appropriate for frequent chronic and/or potentially injurious SW or STs in adults when these are (1) potentially injurious or have caused injury or sleep disturbance to the individual or others; (2) chronic and frequent; (3) capable of causing legal issues regarding violent or sexual behavior (although nocturnal sleep-related violence is rarely, if ever, recurrent); (4) chronic and causing individual or family distress; and (5) associated with secondary consequences (e.g., weight gain, social embarrassment, excessive daytime sleepiness). For some adult sleepwalkers, clonazepam can be taken only on “predictable” nights or nightly for several months, then gradually discontinued or reinstituted if behaviors recur. In patients with POD, clonazepam with/without melatonin may be effective.

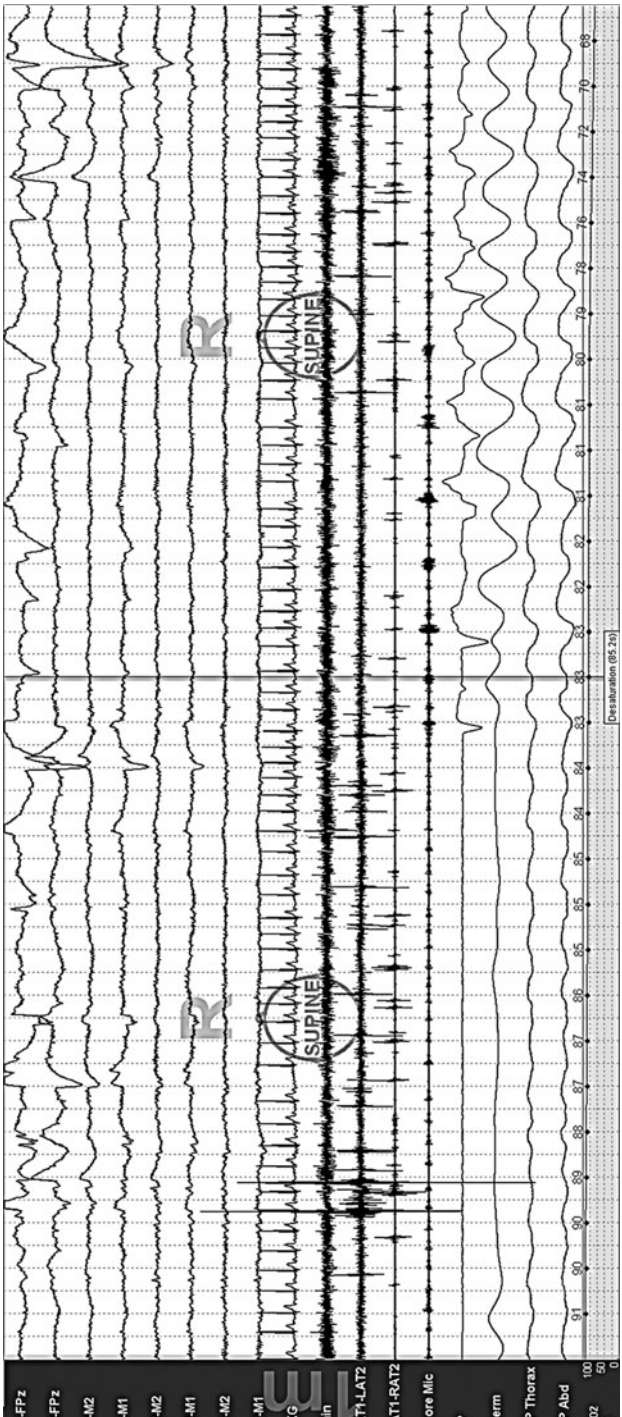


**Fig. 5.3** PSG during REM sleep recorded on 79-year-old patients with symptoms suggestive of OSA and RBD. Note excessive tonic activity in the chin EMG, excessive phasic activity in the leg EMG channels, a prolonged obstructive apnea lasting for 31 s and causing desaturation to 64 % (but no arousal), and atrial fibrillation in the EKG channel

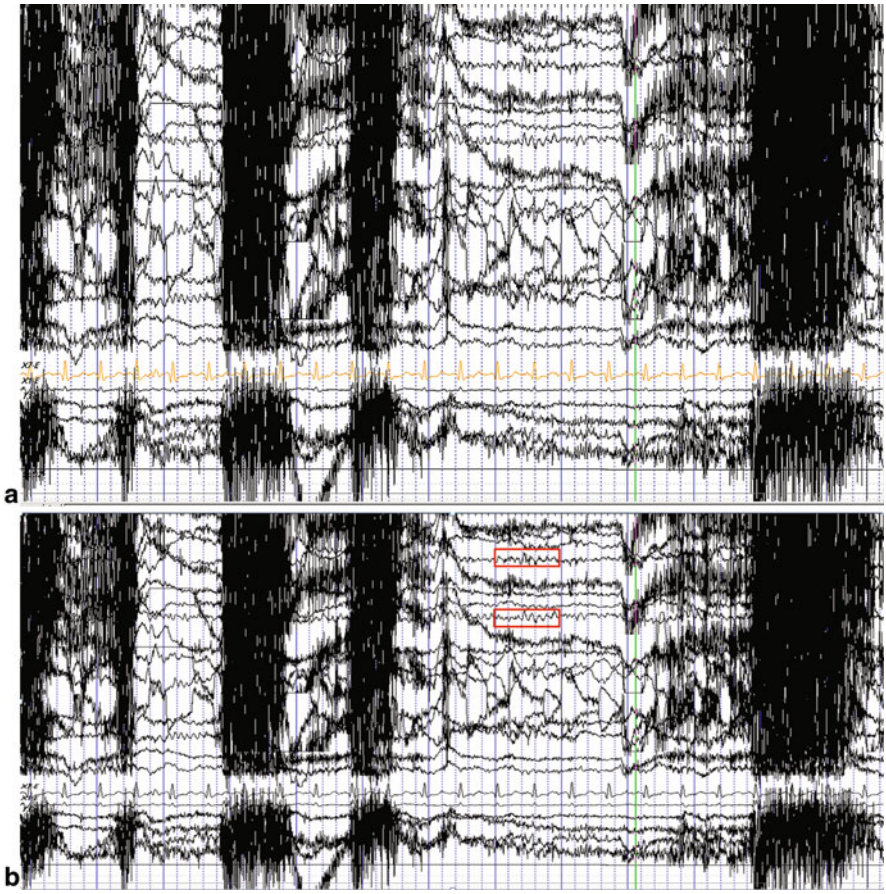


**Fig. 5.4** **a** Abrupt arousal from NREM 2 sleep: 45-year-old patient sits up, cries out, and assumes boxing posture. **b** An epoch of 15 s of violent arousal from NREM 2 sleep in a boxer: EEG background in between muscle artifact 1.5 min later shows normal 9-Hz dominant alpha rhythm of wakefulness (*boxes*)

Clonazepam (0.5–2 mg QHS) has been used for many years with good results in adults with frequent and potentially injurious DoA (or RBD) [35, 180]. The value of clonazepam to suppress frequent DoA in adults is based on five small case series, which together show that 83 % of 61 sleepwalkers responded to it [178, 181]. A single-blinded prospective trial found 10 mg diazepam in adult sleepwalkers often, but not always, suppressed SW [182]. Two large case series have found clonazepam dosed only at bed provided complete or substantial control of injurious sleep behaviors (most often DoA or RBD) with a relatively low risk of adverse effects, dosage tolerance or dependency [98, 183].

Clonazepam has an elimination half-life of 30–40 h, and maximum plasma concentrations occur within 1–4 h after oral ingestion [184]. Clonazepam used to treat DoA, RBD, or other injurious parasomnias in adults is often limited by side effects: sedation especially upon awakening, early morning clumsiness, impotence, and confusion [183, 185, 186]. Clonazepam taken only at night can cause memory or word-finding difficulties, depression, disinhibition, and if the dose is too low, it may trigger SW/ST or confusional arousals, especially in older adults with or without dementia. Clonazepam (1) is relatively contraindicated in patients with a history of substance or alcohol abuse, untreated sleep disordered breathing, cognitive or motor dysfunction, or a nocturnal dissociative disorder and (2) should be used with caution in patients with dementia, gait disorders, or concomitant OSA [187].

## Conclusion

Adult SW most often occurs in individuals with a past history of SW as a child. Adult SW most often arises when priming, precipitating, and provoking factors trigger them in an individual with an inherent, familial, and/or genetic tendency for deep sleep. New onset or recurrence of chronic or injurious SW in adult requires a comprehensive clinical and diagnostic evaluation to differentiate NW from other parasomnias and to identify other treatable primary sleep disorders. SW is more common in patients with neurodegenerative diseases and is often associated with RBD or parasomnia overlap syndrome. Treatment remains conservative by providing a safe environment, avoiding triggers, and reserving pharmacologic intervention for extremely disruptive behavior.

## Case Example

A 31-year-old woman presented following an episode of waking up in the street in front of her home in her nightgown, with a knife in one hand and an unopened package of deli ham in the other. Her partner stated that the patient would infrequently (1–2 times per year) perform unusual behaviors after falling asleep for 1–2 h. Sometimes she would move objects around on a desk, sit at the table sipping from an empty cup, or walk around the house and return to bed. She would wake up on the living room sofa, having gone to bed the night before. If awake, her partner would gently redirect her back to bed and she would sleep the remainder of the night without further incident. Upon awakening, the patient did not remember anything that occurred while SW. Since starting her new job, the episodes increased to 1–3 times per week, and the most recent episode of leaving the house raised special concern. Further questioning revealed that she had numerous episodes of SW as a child. However, these decreased to 1–2 per year during her teenage years.

Review of her medical history was notable for recently starting thyroid replacement therapy, otherwise her medications included an antihypertensive and oral

contraception. Social history was significant for starting a new job that had been stressful and required that she start work at 4 AM. The physical examination was remarkable for a bone-mass index (BMI) of 35 and a neck circumference of 17 cm. The partner reported that the patient snored but did not witness any pauses in breathing. Due to symptoms suggestive of sleep disordered breathing (OSA), the patient underwent PSG (sleep study), which demonstrated moderate OSA. Other hematologic parameters were normal. She was prescribed continuous positive airway pressure (CPAP) therapy and was instructed to increase her sleep time by 1–2 h in order to sleep 7–9 h per night. Follow-up at 1, 3, and 6 months demonstrated complete resolution of the SW behaviors.

### *Comments*

This case demonstrates classic findings in the adult sleepwalker. She has a history of SW as a child. SW events were increased by sleep deprivation, stress, and OSA, causing arousals from sleep. Treatment consisted of treating medical issues, in this case OSA, improving sleep hygiene, providing a safe environment, and reassurance.

## Practical Points

- SW occurs in 2 % of adults but occurs weekly in less than 0.5 %.
- SW in adults is typically associated with a past history or family history of SW.
- Common precipitating factors for adult SW include sleep restriction/deprivation, emotional or situational stress, and use of certain medications.
- Video-PSG is usually needed to differentiate adult SW from NFLE, RBD, parasomnia overlap syndrome, pseudo-RBD due to severe OSA, psychogenic or dissociative events, or malingering.
- A secondary cause should be sought in de novo sleepwalking in adults (e.g., medication changes, psychiatric disorders, SDB, untreated medical disorders, neurodegenerative disease).
- Treatment for SW should focus on providing a safe environment and reassurance and pharmacotherapy should be reserved only for cases where the behavior poses a risk to safety.

Box 5.1: Factors Which Prime, Predispose, or Precipitate Sleepwalking or Sleep Terrors in Adults
- Genetic predisposition = strongest factor;
- Cumulative partial sleep deprivation or sleep restriction;
- Situational, mental, or emotional stress;
- Extreme exercise, fatigue, or exhaustion;

- Environmental stimuli (noise, light, touch, attempts to arouse);
- Circadian rhythm disorders (shift work, jet lag, night shift);
- Other primary sleep disorders: obstructive sleep-disordered breathing, restless legs syndrome, periodic limb movements during sleep, narcolepsy with cataplexy;
- Drugs (nonbenzodiazepine hypnotics, alcohol, antidepressants, antipsychotics, antihistamines, other sedative-hypnotics, sodium oxybate);
- Fever or infections.

Box 5.2: Summary of the Clinical Features of Historical Cases of Violence Attributed to Sleepwalking and Its Variants [21]

- Majority were male, most between ages 17–35 years;
- The provocation was quite minor and the violent response greatly exaggerated;
- Most violence began following direct provocation or close proximity to the sleepwalker;
- Most often, the victims encountered or sought out the sleepwalker;
- Violence was more often inaccurately directed, but was precisely delivered in other cases;
- Violence most often directed at the bed partner, occasionally a child or a subject's mother;
- Implements employed included guns, hammers, shovels, scissors, axes, razors, a bayonet, or throwing a child or baby out the window or against a wall;
- During the episode, the individual fails to recognize the victim but exhibits feelings of horror or fear upon awakening after the event and recognizing what has occurred;
- Most lack recall of the event, but two were aware that they had shot someone but uncertain who;
- Immediate triggers were physical stimulation or loud noises, and often occurred when the individual had been under psychological stress, or had a recent quarrel, argument, or threatened attack;
- Most but not all had a previous history of SW.

Box 5.3: Guidelines for Determining a Putative Role of Sleep Disorder in a Violent Act

- Episode typically brief, lasting < few minutes;
- Spatial orientation and fine motor coordination intact but no facial recognition or memory of event;

- Upon return of consciousness, perplexity, horror, or remorse without attempt to escape, conceal, or cover-up the action; pain perception for wounds often delayed;
- History of parasomnias;
- Usually some degree of amnesia for the event; however, islands of partial memory may remain;
- Victim may be someone who merely happened to be present but may have been the stimulus for the arousal;
- Acts of violence usually follow periods of poor sleep, some related to obstructive sleep-disordered breathing, and/or insomnia related to anxiety or depression;
- Recurrence of violent behaviors during sleep are rare.

Box 5.4: Differential Diagnosis for Nocturnal Wandering in Adults
- NREM arousal disorder (confusional arousal, sleepwalking, sleep terror);
- Sleep-related epilepsy;
- REM sleep behavior disorder (RBD) and pseudo-RBD due to obstructive sleep apnea;
- Sleep-related panic attacks;
- Nightmare disorder;
- Sleep-related dissociative disorder;
- Sleep-related choking, laryngospasm, or gastroesophageal reflux;
- Sleep-related rhythmic movement disorder with vocalization;
- Sleep-related expiratory groaning (catathrenia);
- Post-traumatic stress disorder (PTSD);
- Sudden death when sleeping due to myocardial infarction, Brugada syndrome, untreated OSA, sudden unexpected death in epilepsy, and trauma.

Box 5.5: Clinical Characteristics That Suggest Sleep-Related Hypermotor Seizures
- An abrupt, often explosive, onset awakening the patient from grossly undisturbed NREM 2 sleep;
- Asymmetric dystonic or tonic postures;
- Thrashing, pedaling, and kicking of the lower extremities;
- Tend to be "fairly" stereotyped in appearance for the individual patient;
- Brief (typically lasting 20–30 s, less than 1–2 min);
- Patients are often aware during the seizure, but say they cannot control their movements or vocalizations;

- No postictal confusion or amnesia;
- 20 % have no scalp-recorded ictal EEG activity accompanying them.

Box 5.6: Treating Sleepwalking and Sleep Terrors in Adults
- Present the diagnosis to patient and family and educate them about the nature of these;
- Allow episodes to run their course:
  - Interfere only to prevent injury;
  - May try gently redirecting the individual back to bed and resist waking the person;
- Secure the bedroom and home to prevent injury:
  - Nightlights; window and door locks;
  - Motion detectors in hallways;
  - Alarms or barriers at door/stairs;
  - Remove sharp objects from bedroom floor;
  - Consider ground floor bedrooms and moving the bed partner;
  - Secure hazardous objects such as kitchen knives and guns;
- Regular bed- and wake-times with adequate amounts of sleep;
- Decrease noise, light, pain, nocturia, stress, and dyspnea, which may contribute to partial arousal;
- Avoid visual, auditory, or tactile stimuli especially during the first third of the night, which may trigger an event;
- Avoid sleep restriction/deprivation, jet lag, night/shift work, extreme exercise, fatigue, and emotional or situational stress;
- Identify and treat other primary sleep disorders (sleep apnea, restless legs, narcolepsy RBD, sleep-related epilepsy, parasomnia overlap syndrome, insomnia, nocturnal eating, and gastroesophageal reflux);
- Avoid precipitating medications or substances: alcohol, antipsychotics, antidepressants, antihistamines, sedative-hypnotics, and benzodiazepines;
- Clonazepam is potentially injurious and may cause excessive daytime sleepiness or daytime stress.

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