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Review

A review of sleepwalking (somnambulism): The enigma of neurophysiology and polysomnography with differential diagnosis of complex partial seizures

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Abstract

The goal of this report is to review all aspects of sleepwalking (SW), also known as somnambulism. Various factors seem to initiate SW, especially drugs, stress, and sleep deprivation. As an etiology, heredity is important, but other conditions include thyrotoxicosis, stress, and herpes simplex encephalitis. Psychological characteristics of sleepwalkers often include aggression, anxiety, panic disorder, and hysteria. Polysomnographic characteristics emphasize abnormal deep sleep associated with arousal and slow wave sleep fragmentation. In the differential diagnosis, the EEG is important to properly identify a seizure disorder, rather than SW. Associated disorders are Tourette's syndrome, sleep-disordered breathing, and migraine. Various kinds of treatment are discussed, as are legal considerations, especially murder during sleepwalking.

Keywords: Sleepwalking; Somnambulism; EEG; Polysomnography; Sleepstages; Non-rapid eye movement sleep; Drugs; Heredity; Arousal; Night terrors

1. Introduction

Sleepwalking (SW), also known as somnambulism, has been an enigma to neurophysiologists and polysomnographers, especially because complex motor behavior usually occurs during deep (stages 3 and 4) sleep [1]. The goal of this review is to describe all aspects of this condition and to try to understand how such complex behavior could emerge during deep sleep. Night terrors have been known to share a common genetic predisposition with SW and therefore, findings in one usually apply to the other [2].

2. Factors initiating sleepwalking

2.1. Drugs

2.1.1. Lithium

One of the earliest reports on SW was in 1979 when Charney et al. [1] stated that 9% of psychiatric patients on a combined lithium–neuroleptic treatment exhibited SW. As SW usually occurs during deep slow wave sleep, the increase in this stage of sleep caused by lithium could help to explain why this medication tended to increase SW. In this early study, the occurrence of seizures in two patients was considered unrelated to the somnambulistic episodes. Possible confirmation regarding lithium was published in 1986 with the description of a patient with a schizoaffective disorder who experienced SW when taking lithium, but this patient was also receiving chlorpromazine, triazolam, and benztropine [3]. In another study 7% of psychiatric patients developed SW while on lithium alone or in combination with other psychotropic medications. In 12% of these sleepwalkers, this *parasomnia* (any dysfunction associated with sleep) occurred in childhood, and 27% of these patients had their childhood SW reactivated by lithium [4].

2.1.2. Zolpidem tartrate

As early as 1999, this popular sleep medication (Ambien) was reported to initiate SW, which ceased when the same medication was stopped [5]. The latter

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authors had reported on two other similar cases, and both had a previous history of SW in their youth. Another patient was reported to develop SW associated with zolpidem, but this case possibly was complicated by a previous brain injury [6]. Finally, one more patient was reported to develop SW, not on zolpidem monotherapy, but by the interaction with valproic acid (VPA). The SW stopped during the withdrawal of VPA, and with a rechallenge of VPA, sleepwalking again recurred [7]. The three preceding references establish the point that the drug may initiate SW, and there were five other cases in the literature that were unnecessary to mention. In other sections of this report, a few single cases may be mentioned to establish a given point, and the repetition of similar cases seems to add little.

2.1.3. Bupropion

One patient used bupropion as an aid to stop smoking and developed SW, associated with eating behavior and amnesia. This drug is a dopaminergic, central nervous system (CNS) stimulant [8].

2.2. Sleep deprivation

As a tool to provoke SW, sleep deprivation was investigated in 10 patients. Although stereotypical complex behavior during sleep did increase with sleep deprivation, the authors considered that SW itself did not increase. This technique was viewed as an efficacious method to increase complex behavior during sleep, but was considered only as a preliminary stage of SW [9].

In a later study the effect of 38 hours of sleep deprivation was investigated in 10 adult patients. Sleepwalkers showed a significant increase in the frequency and complexity of SW episodes, and, likely as an important finding, a greater number of arousals from slow wave sleep also occurred [10].

In a more recent study (2007) investigators summarized that the primary factors initiating SW included conditions and substances that would increase slow wave sleep. These factors included sleep deprivation, but also alcohol, stress, fever, and various medications [11].

2.3. Continuous positive airway pressure (CPAP) trial

One patient with severe obstructive sleep apnea was given a nasal CPAP trial, and two episodes of SW were observed during a period of delta sleep rebound [12].

2.4. Pregnancy

One patient developed SW only during her pregnancy [13]. On the other hand, in another study on 325 pregnant females, the total number of parasomnias declined, especially with a decrease in SW from the prepregnant period to the second trimester. The conclusion was that most parasomnias decrease during pregnancy [14].

2.5. Psychic conditions

An early (1976) study indicated that ''psychic moments'' or ''certain states of tension'' may trigger SW [15].

2.6. Summary

As both stimulants (buproprion) and depressants of the CNS like sleep medication, in addition to various other conditions (sleep deprivation, pregnancy), may initiate SW, one possible conclusion is that any condition that changes the excitation–inhibition balance in the CNS may precipitate SW.

3. Factors as a presumed etiology of sleepwalking

3.1. Trauma (psychiatric)

After exploring SW in one patient by means of a pivotal dream, the investigator concluded that a severe traumatic experience in early childhood likely was the etiology of the SW [16].

3.2. Herpes simplex encephalitis

A 64-year-old man with herpes simplex encephalitis developed SW along with a memory disturbance. CT, MRI, and SPECT revealed lesions in the right temporal lobe [17].

3.3. Stress

In a report from the United Kingdom, the author emphasized that the constitutional basis for SW was beyond doubt, but that the actual expression may be related to stressful life events, resulting from individuals' personality, their relationships, and their circumstances of living. Therefore, the authors concluded that there was a need for provision of a secure environment and also for counseling, in addition to possible medication [18]. This latter study included one interesting point about the prevalence of SW: one-third of a million individuals in the United Kingdom are considered sleepwalkers.

3.4. Thyrotoxicosis

The authors collected the histories of eight patients with new-onset SW episodes that coincided with the onset of thyrotoxicosis. The disappearance of SW with the successful achievement of euthyroidism supported the cause-and-effect relationship. This hypothesis was further supported by the absence of a family history, by the adult onset, and by the relapse of SW in two patients when their thyrotoxicosis became poorly controlled as a result of poor compliance with medications. One possible mechanism to explain the findings was the prolongation of non-rapid eye movement (NREM) sleep, although not necessarily

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predicted as a consequence of thyrotoxicosis. Another factor possibly initiating SW may be the fatigue associated with this thyroid condition [19].

3.5. Heredity

3.5.1. Introduction

As early as 1980 authors made clear that SW and night terrors (NTs) shared a common genetic predisposition. SW was considered more prevalent and a less severe manifestation of the same substrate that underlies NTs. Evidence for the inherited aspects included the fact that 80% of SW pedigrees and 96% of NT pedigrees included one or more individuals (other than the proband) who were affected by SW, NTs, or both. The authors concluded that inheritable factors predispose the individual to develop SW and/or NTs, but the expression of the trait may be influenced by environmental factors [2].

Further evidence for inheritance was provided by a short study of 37 children, one or both of whose parents had sleepwalked during their childhood. At 8 years of age these children were more likely to manifest SW than control children [20].

3.5.2. Twin study

An extensive Finnish study involved 11,220 adult subjects, including 1045 monozygotic and 1899 dizygotic twin pairs. Childhood SW was more frequent in females (6.9% vs 5.7% in males). However, as adults the prevalence was reversed, with 3.9% of males versus 3.1% in females. This difference in sex ratio between children and adults represents one of the few differences found between these two age groups. Otherwise, SW is similar in its manifestations in children and adults. For SW in childhood, the concordance rate for probands was 0.55 for monozygotic and 0.35 for dizygotic pairs, and for adults, the values were 0.32 and 0.06, respectively. Among the sleepwalkers, those who reported this parasomnia in childhood and also as adults were 25% males and 18% females. The proportion of total phenotypic variance attributed to genetic influences was 66% in males and 57% in females in childhood SW and 80% in males and 36% in females in adult SW. Thus, these data demonstrate a substantial genetic effect in both childhood and adulthood [21].

3.5.3. Gene study

HLA-DQB1 typing was performed on 60 SW patients, matched with controls. Thirty-five percent of the SW patients were DQB1-050 positive versus 13% of the controls (odds ratio = 3.5, 95% CI = 1.4–8.7). Excess transmission was observed for DQB1-05 and DQB1-04 alleles in familial cases. These findings suggested that specific DQB1 genes were implicated in SW [22]. The genetic susceptibility has recently been taken for granted, as noted in studies in 2007 [11]. One last point on genetics is that SW apparently is not observed in infrahumans; the well-known researcher Jane Goodall believes that this phenomenon does *not* occur in chimpanzees. There is medical and evolutionary interest in determining why this SW behavior became established only during the time of human existence [23].

4. Clinical characteristics of sleepwalkers

An early description of patients with SW included the finding that sleepwalkers demonstrated high levels of psychopathology. Specifically, the patients showed active, outwardly directed behavioral patterns suggestive of difficulties in handling aggression [24].

Another study also emphasized aggression, in particular inhibited aggression, and an attitude characterized as a highly developed mental defense against anxiety, as determined by Rorschach tests [25]. Anxiety was emphasized in one other report, which included a discussion of SW and NTs, both scoring highly on the anxiety and hysteria scales (of the Crown–Crisp experimental index). One additional finding was that patients who sleepwalk also scored highly on externally directed hostility [26], similar to the aforementioned aggression [24,25]. In two other patients with SW and NTs, another finding was panic disorder and, often, a family history of this disorder. The authors hypothesized that SW/NTs and panic disorder involved a similar constitutional vulnerability related to a dysregulation of the brain stem [27].

Some investigators have failed to find abnormal behavior and have concluded that patients with SW have normal psychometric tests. Also, one conclusion was that the *Diagnostic and Statistical Manual, Third Edition, Revised,* (DSM-III-R) Axis I psychiatric tests were not abnormal. On the other hand, 45% of the 33 patients had previously received psychological or psychiatric therapy for their parasomnia, but without benefit [28]. A general conclusion was reached in another report that summarized these parasomnias as indicators of an underlying mental disorder [29]. More specific characteristics were listed in still another study that concluded that these patients with SW were overanxious and had panic disorders, simple phobias, and suicidal thoughts [30]. Consistent with these latter characteristics, another report stated that some patients with SW had a history of major psychological trauma, with scores on a dissociation questionnaire similar to those of persons with posttraumatic stress disorder. These same patients scored highly on anxiety, phobia, and depression scales. Even those who had no apparent major psychological trauma scored highly on these latter scales [31].

In summary, there is evidence that sleepwalkers frequently demonstrate aggression, anxiety, hysteria, panic, and phobias.

5. Polysomnographic characteristics of sleepwalking

In an early study in 1995, SW was characterized as occurring during the first 3 hours of sleep when sleep stages 3 and 4 were most prevalent. The episodes were noted to

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last from 30 seconds to 30 minutes [32]. In another study, analysis showed decreased sleep efficiency and also stage 2 sleep, but increased stage 3 and 4 slow wave sleep. Also reported was an increase in the arousal index and wakefulness after sleep onset, and sleep fragmentation was concentrated mainly during stages 3 and 4. Slow wave activity during the 2 minutes before the SW episode was greater than that in the 10 minutes before the same episode or before awakening. The final conclusion in this latter study was that abnormal deep sleep was associated with increased fragmentation that might be responsible for the SW episodes [33]. Additional data revealed a lower level of these slow waves during the first NREM period and also a greater number of awakenings from slow wave sleep. The general conclusion was that sleepwalkers appear to suffer from an abnormality in the neural mechanisms responsible for the regulation of slow wave sleep [34].

As just mentioned regarding the first sleep cycle [34], another group confirmed that patients with SW experienced more disturbed sleep during this early sleep cycle. Also confirmed was that these patients had more arousals and a smaller amount of slow wave activity during the first cycle. One additional finding was that just before the confusional arousal, an increase in very slow delta waves occurred. One other finding during the actual SW episodes is that some diffuse alpha activity can be seen during the delta slow waves of stage 3/4 sleep. The authors hypothesized that this arousal, appearing as very slow delta activity, may represent a *cortical* reaction to a generalized brain activation [35]. This reviewer would emphasize that this latter conclusion is crucial to the understanding of SW.

A more recent study [36] has included some exceptions to these latter general findings. Although SW and NT episodes usually manifest themselves during the first episode of slow wave sleep, the present authors pointed out that these episodes may appear *anytime* during NREM sleep. Although hypersynchronous delta activity is often considered the hallmark of SW, these same authors concluded that this activity was nonspecific. Further findings were that altered consciousness characterized the SW or NT episodes and that low delta power appeared in the first cycle and a slow decline in delta power was seen in successive sleep cycles. The final conclusion was that these data indicated that sleepwalkers have a chronic inability to sustain slow wave sleep. Another study [37] investigated ''normal'' sleep in sleepwalkers and found a higher ratio of hypersynchronous delta activity over the time spent in stages 2, 3, and 4 on the frontocentral derivations. However, these investigators concluded that these hypersynchronous slow delta rhythms had a low specificity for the diagnosis of SW [37].

The cyclic alternating pattern (CAP) is a phenomenon of changing patterns in sleep that often cycle and alternate every 20–30 seconds, and recently this pattern has been studied in patients with SW [38]. The important finding was a higher CAP rate in sleepwalkers, and the instability of NREM sleep in these patients was at times detectable only by calculation of the CAP rate. A more recent study from the same laboratory [39] confirmed an abnormal CAP rate and also confirmed previous studies showing a decrease in delta power in cycles 1 and 2 during non-sleepwalking nights. The general conclusion was that during non-sleepwalking nights, there was an instability of NREM sleep.

In summary, there is evidence that in sleepwalkers, abnormal deep sleep is associated with slow wave sleep fragmentation and that abnormal neural mechanisms are present for the regulation of slow wave activity with the inability to sustain slow wave sleep. Also, the CAP rate is often abnormal, and, according to one group, the delta activity in deep sleep may be relatively nonspecific.

6. EEG characteristics of sleepwalking

A very early study in 1978 on SW reported that theta activity from the temporal lobe was seen only with sphenoidal electrodes and not with routine scalp EEGs. Although the theta rhythm was not a sharp wave or spike discharge, the authors did raise the question of SW as a paroxysmal disorder from the temporal lobe [40]. A negative study in 1980 reported that waking EEGs were not helpful, and all-night records were needed if abnormalities were to be found [41]. On the other hand, another study did report that in the waking state, ''disturbed'' records appeared in nearly one-half (47%). Localized slow, spike, or sharp wave activity, generalized bursts of high-voltage sharp waves, spike and slow activity, or spike and wave complexes were seen. Most of these latter patterns refer to epileptiform activity and, therefore, would be viewed as controversial, especially because SW has not usually been considered a form of epilepsy [42]. Also, another study did report that sharp waves were, in fact, seen in the majority (12/13) of sleepwalking children and in some (5/13) adults. On the other hand, this same author did conclude that there was no clear relationship between SW and epilepsy [43].

Sleep records were emphasized in another study that did not report any epileptiform activity. Similar to the findings reviewed here on polysomnographic characteristics (see Section 5), these authors reported a higher proportion of delta waves and also more interruptions in deep sleep [44]. A sleep record was recommended by others to clarify the diagnosis of SW [45]. This advice proved to be crucial in a patient who appeared at first to be a sleepwalker, but ictal epileptic discharges were seen at the onset of the SW episodes, proving that the events were, in fact, seizure episodes [46]. A similar study of episodes appearing at first as SW was reported, but, with ambulatory EEG monitoring, ictal phenomena appeared just before the episode. Also, as confirmation, interictal discharges were seen from sphenoidal electrodes with an epileptogenic focus in the left anterior temporal area. Control over these episodes with carbamazepine helped to confirm a seizure disorder [47]. Epileptiform patterns were not part of another report that did *not* find ''delta wave buildup'' just *before* a behavioral

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arousal. *After* this arousal, diffuse delta activity with or without alpha and beta rhythms appeared. The general conclusion was that SW is a disorder of abrupt arousal [48].

The main summary point in this section is that EEG monitoring should be considered to identify those patients with a seizure disorder as having episodic nocturnal wandering, rather than sleepwalking.

7. Differential diagnosis

As indicated in the previous section on EEG characteristics, the major differential diagnosis with SW is a seizure disorder, often called "episodic nocturnal wandering" [1,32]. With long term video/EEG video, other groups have succeeded in differentiating sleepwalking from epilepsy [49,50]. The clear evidence for an epileptic seizure, as opposed to SW, is the recording of rhythmical ictal patterns before the onset of and during the episode [46]. In addition to the recording of ictal patterns in ambulatory video/EEG video, interictal discharges from scalp (or sphenoidal) electrodes have further confirmed the epileptiform nature of the episodes [47]. One example of "epileptic nocturnal wandering" was reported with long term video/stereo/EEG monitoring with intracerebral electrodes, demonstrating a well-localized discharge within the right temporal structures with secondary spread to the cingulate regions [51]. Not only can *temporal* lobe epilepsy be found in some of these patients, but also nocturnal *frontal* lobe epilepsy can appear as a complex partial seizure with repetitive motor movements, similar to sleepwalking [52]. Although small foci in the supplementary motor area may not reveal clear ictal patterns and artifacts are common, most cases of temporal or frontal lobe epilepsy reveal ictal rhythms during a seizure episode.

Additional comments are needed to contrast SW with seizure phenomena. First, the onset in SW is usually during the earliest sleep cycle in stage 3/4, whereas seizures often occur during stage 1/2 anytime throughout the night. Vocalization and automatism are uncommon in SW, whereas seizures often are accompanied by a scream and by automatisms. Sleepwalkers usually return to bed, whereas seizure patients do not. Awakening is uncommon in SW, but more common in seizures. After the episode, confusion is uncommon in SW, but common in seizure patients. Another form of parasomnia is REM-behavior disorder, which usually involves violent motor activity acting out dreams in older men, whereas SW rarely involves violent behavior, more often in children.

8. Pathogenesis

One early article in 1976 made an assumption that it was the corpus striatum that was involved in the overall coordination of stereotypic motor movements and, thus, possibly involved in SW [15]. One other report suggested a possible pathogenesis by assuming that the "dissociation between body sleep and mind sleep" arises from the activation of the thalamocingulate pathways with a persisting deactivation of other thalamocortical arousal systems [53]. The lack of full awareness and the amnesia for the episode suggest that the latter arousal system may well be deactivated.

9. Associated disorders

9.1. Tourette's syndrome

In one report of 171 children, one-third had Tourette's syndrome. Of the 13 identified as sleepwalkers, 10 also had Tourette's syndrome. As 19% of the Tourette cases had SW and 8% of the children had SW, these data indicate only that there is a relationship between these two disorders, but the strength of the relationship cannot be easily determined. The conclusion was that this combination of SW and Tourette's may be due to a disturbance of serotonin metabolism [54].

9.2. Sleep-disordered breathing

Abnormal respiratory events occur frequently in patients with SW, likely responsible for the great number of arousal reactions in triggering the sleepwalking [55]. In another study, the majority (58%) of children with SW also had sleep-disordered breathing, and one-half also had a family history of the breathing abnormality. Most of these patients were treated with tonsillectomy, adenoidectomy, and/or turbinate revisions. Successful treatment of the disordered breathing eliminated the SW in all patients [56]. The importance of the latter conditions was further emphasized by another study that pointed out that all children with SW had either obstructive sleep apnea or an upper airway resistance syndrome [38].

9.3. Migraine

An early report in 1983 emphasized the relatively high frequency of SW (30%) in children suffering from migraine headaches. Also, 67% of sleepwalkers had migraine [57]. Three years later, another study confirmed the high frequency of SW in patients with migraine, whereas the frequency of SW in patients with nonmigraine headaches was similar to that of the general population. Migraine was usually of the visual type, occurring in childhood, whereas the SW was seen earlier in late infancy [58].

10. Legal considerations

10.1. Homicide or attempted homicide

As early as 1983, investigators reported that two sleepwalkers had episodes resulting in the deaths of three other individuals. Both sleepwalkers were employed, married, and functioning well without serious psychopathology, but the legal consequences were not made clear in this

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report [59]. Another case report described a 14-year-old boy who rose from his bed at 2:00 AM and savagely stabbed his 5-year-old cousin. The conclusion in this case was that the sleeping mind was not in touch with reality, including amnesia for the events, as is the usual finding in similar cases [60]. Another case of SW was described in which murder was committed; the author acknowledged that in 1992, these disorders had not received much professional attention [61]. These latter cases led to the conclusion that cases of homicide or attempted homicide during SW should lead to somnambulism as a legal defense and, therefore, to acquittal of the sleepwalker. The evidence in this latter case included a personal and family history of SW and neurological, psychiatric, and psychological assessments [62]. The possibility that these violent episodes could be a REM-behavior disorder is unlikely, because a clear history of previous SW is necessary in court cases. Also, SW usually occurs in young adults, whereas REM-behavior disorder is seen in older adults. Still another patient was indicted for homicide and attempted homicide in which SW was a legal defense, leading to an acquittal. The author pointed out that it was generally accepted that SW is a state of automation in which individuals are unaware of and have no control over their behavior [63]. A last report discussed in general that murder during SW leads to somnambulism as a defense, and this defense is usually successful [64]. In June 2007, some news programs stated that various defense attorneys are attempting to use SW as a defense against murder. However, clear evidence for SW before the murder would seem to be required.

10.2. Indecent exposure

This next case involves a naked sleepwalker who was actually convicted of indecent exposure. Sleepwalkers are seldom convicted of the crimes they commit while asleep, and this case, therefore, represented an exception [65]. The next patient was not an exception because the indecent exposure was viewed as being committed during SW and the jury found the defendant not guilty [66]. A 43-year-old man was described as having repeated sleep-related injuries during inappropriate exposure, violent nocturnal activity, including frenzied running, but also throwing punches, wielding knives, driving an automobile long distances, and repeatedly injuring his wife. The author proposed a new medicolegal concept, consisting of "a parasomnia with continuing danger as a noninsane automatism" [67].

10.3. Sexual behavior

Two cases were presented involving sexual behavior performed while asleep, called *sleep sex*. In the second case, sexual battery was the indictment and SW was used as a legal defense [68]. Another individual who was accused of sexual misconduct with a child and was successfully defended with SW in the form of a noninsane automatism [69]. One other fascinating example involved a 27-year-old man with a history of sexual behavior during sleep. His wife reported episodes of amnestic sexual behavior that began 4 years before referral. During these episodes, the patient typically had total amnesia but achieved successful sexual intercourse with his wife [70]. In another report, 11 patients had "sexsomnia," *specific* behavior of a sexual nature during sleep. Distinctive features included a more prominent automatic arousal, more restricted motor activities, and some form of dream mentation [71]. Finally, sexsomnia or sleep sex was described in another report, including explicit sexual vocalizations, violent masturbation, and complex sexual acts including anal, oral, and vaginal penetration. This report also included a description of a patient in England who was charged with three counts of rape and was acquitted on the basis of the automatism associated with SW sexual behavior [72].

11. Treatment

11.1. Hypnosis

Four of six SW patients reported total alleviation of this parasomnia with the use of hypnosis [73]. In another report, subjects had severe somnambulism, but otherwise were free of psychiatric illness and responded well to six brief sessions of specialized hypnosis. Lasting improvement was noted 1 year later [74]. Of 27 adults with SW, 74% reported "much or very much improvement" with self-hypnosis practiced at home [75].

11.2. Anticipatory awakening

An 8-year-old boy was treated by awakening him for 5 nights just before the SW episodes were anticipated. Later these episodes of sleepwalking stopped entirely [76]. Still another report indicated that parents would awaken their three SW children several hours after they went to sleep and just before the typical time of the SW episode. This procedure proved immediately successful in all three children [77].

11.3. Medication

11.3.1. Benzodiazepines

11.3.1.1. Diazepam. Nightly diazepam at 10 mg alleviated symptoms of intractable SW, and tolerance did not develop in any of the few patients in the study [78].

11.3.1.2. Clonazepam. In five of six SW patients, clonazepam effectively suppressed the somnambulism and, together with phenytoin, was successful in the sixth patient [79]. In a 43-year-old man, clonazepam was successful for at least 5 years [67]. One 27-year-old SW man was put on 2 mg/day with "remarkable clinical improvement" [70], and yet another report also indicated the usefulness of clonazepam [80]. Benzodiazepines, in general, were mentioned in other reports [18,28].

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11.3.2. Imipramine

Two patients with NTs and SW responded well to imipramine, but not to diazepam [81]. One 10-year-old child was fully responsive to therapeutic doses of imipramine [27]. No well-controlled studies on treatment have been published, and in some reports, only a few patients were mentioned. Despite this obvious limitation to these studies, however, they provide some evidence of the possible efficacy of the different types of treatment.

12. "Essence" of sleepwalking: A summary

Many different conclusions have been drawn to explain this intriguing phenomenon. They include a paroxysmal disorder of the temporal lobe [40], a disorder of abrupt arousal [48], a noninsane automatism [69], a dissociation between body sleep and mind sleep [49] and an abnormality in the neural mechanisms responsible for the regulation of slow wave sleep [34]. Also included was a phenomenon with a "protective dissociative mechanism, mobilized when intolerable impulses, feeling and memories escape within sleep with a diminished control of a mental defense mechanism, erupting with a limited motoric or affective form with restricted awareness and subsequent amnesia for the event" [31]. Finally, other hypotheses are a NREM parasomnia with an awakening disorder [80], partial waking with an alteration of the microstructure of sleep [82], and sudden arousal from NREM sleep [36].

The present reviewer would suggest that sleepwalking involves an arousal that appears as very slow delta rhythms superimposed on deep stage 3/4 sleep. At times, this arousal may be from sleep-disordered breathing or may be related to an uncommon relatively low threshold of arousal in this deep sleep stage. The arousal does not activate brain stem activity, and therefore, the delta rhythms of stage 3/4 sleep continue during the SW episode, projected from the brain stem and appearing on the EEG. On the other hand, the cerebral cortex is activated, therefore supporting complex behavior, but to a suboptimal level to explain the irrational behavior that often occurs and also the amnesia for the events. In summary, SW involves suboptimal arousal of the cerebral cortex, but without any change in the deep sleep of the brain stem, appearing as slow delta rhythms in stage 3/4 sleep. This proposed functional separation of the brain stem from the cerebral cortex is reminiscent of a similar condition long ago discussed in brain death. In this instance, brain stem reflexes may be absent, but EEG activity can still be present [83]. Thus, the brain stem and the cerebral cortex are in different functional states, just as it may be in sleepwalking.

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