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Chapter

Somnambulism: Recent Findings

Sachi Sri Kantha

Abstract

Somnambulism is characterized as a parasomnia occurring during slow-wave sleep stages. It is also an autosomal dominant disorder, and its genetic locus has been identified at chromosome 20 q12-q13.12. The prevalence of somnambulism among children and adults has been estimated as 1–6%. Among the specific environmental factors precipitating somnambulistic episodes in adults, prescription drug use, alcohol, emotional stress, sleep deprivation, and certain psychiatric conditions have been suggested. Somnambulistic patients frequently cause injuries to themselves, family members who sleep nearby, and in-hospital settings to nurses who attend on them. Even cases of homicidal somnambulism have been reported. The lack of a suitable animal model to study somnambulism in controlled settings hinder appropriate pharmaceutical protocols to prevent this disorder among humans. Partial or total alleviation of somnambulistic symptoms in children and adults deserves the attention of health researchers.

Keywords: parasomnia, sleepwalking, treatment, trauma, violence

1. Introduction

In 1969, while introducing the electroencephalographic (EEG) recordings of sleep stages, Barbara Long [1] wrote, “We know that patients in hospitals, away from their usual sleeping environments and beset by the problems created by illness, frequently have difficulty meeting one of their most basic physiologic needs—the need for sleep—at a time when they require it most.” In this commentary, Long briefly introduced insomnia as one of the health problems suffered by hospitalized patients. Subsequent reports by Williams [2] and Walsleben [3] passingly mentioned other sleep disorders, but failed to elaborate on somnambulism (in the lay term, sleepwalking).

In one of the anonymous early reports [4] that appeared on somnambulism in 1834, the recommendation offered for treating somnambulism was “to seize the arms suddenly, and halloo in the ears until the sleep awake: of the application of a jug of cold water, by pouring it suddenly upon the head. In this latter case, however, care should be taken to have the body well rubbed with dry towels after the operation.” Whether somnambulism will be permanently cured by this sort of crude treatment is open to doubt.

Nevertheless, medical knowledge on somnambulism, especially after the discovery of rapid eye movement (REM) sleep in 1953 [5] and the use of polysomnographic detection methods [6, 7], has accumulated in the past 65 years. Though it was widely believed
until 1956, that somnambulism occurs during the dream phase (i.e., REM sleep stage), Jacobson et al. [8] demonstrated in nine subjects (seven men and two women, aged 9–23 years) that somnambulistic episodes occur during the N3 stage of slow-wave sleep (SWS) or non-rapid eye movement sleep (non-REM sleep). Specific electroencephalogram features of the N3 stage of SWS include delta waves (0.5–2 Hz, amplitude >75 μV) and slow oscillations (<1 Hz) [9]. For what is known about somnambulism until now, five reviews [9–13], with over 50 citations can be consulted profitably.

The objective of this chapter is to review recent developments on the presumed etiology, associated disorders, and treatment strategies currently available for somnambulism. The interdisciplinary relevance of somnambulism between medicine and law is also highlighted.

2. Etiology

Somnambulism or sleepwalking is one of the 15 parasomnias among the 81 recognized major sleep disorders. It is characterized by sudden arousal occurring at the SWS stages, resulting in walking behavior during a state of altered consciousness [14]. Somnambulism prevalence of 1–6% was indicated by Kleitman [15], “depending on whether the survey applied to the general public, hospital patients or to limited to children with sleep abnormalities.” Thirty years later, Shapiro and Dement [16] offered a wider range (1–15%) of somnambulism prevalence, with children at the higher end of the range, relative to adults.

According to Kleitman [15], French physiologist Georges Henri Roger (1860–1946) had provided a good description of a typical sleepwalker, in his 1932 book Les Troubles du Sommeil—Hypersonnies, Insomnies, parasomnies. To paraphrase Kleitman [15],

“The sleeper performs various acts with a certain degree of dexterity and can avoid obstacles. But his behavior is characterized by a rigidity, which gives him the appearance of an automaton. He can answer questions correctly and is quite receptive to suggestions. He obediently carries out rather bizarre orders and tends to preserve attitudes passively imposed on him, exhibiting cataleptic properties. At the end of 15 to 30 minutes of activity, he goes back to bed, sometimes fully clothed, and wakes up next morning, quite surprised to find himself dressed. He can carry out dangerous tasks, like walking along the edge of a roof, which he would be afraid to do when awake” [15].

Hereditary factors in the prevalence of somnambulism were first recorded in a short note, appearing in Gazzetta degli Ospitali 1930 by Clerici in a six-member Italian family (comprising of husband, cousin-wife, and their four children), all suffering from somnambulism. This fact was subsequently confirmed in other countries as well [6–7, 17].

Among a sample of 60 Caucasian subjects with somnambulism disorder, distributed in three countries (France, Switzerland, and Germany), Lecendreux et al. [18] reported that compared to eight (13.3%) age-matched controls, 21 (35%) somnambulists were positive for DQB1 allele of HLA-DQB antigen, and claimed this finding as a first genetic maker for somnambulism. HLA stands for human leukocyte antigen. Subsequently, Licis et al. [19] studied a four-generation Caucasian family in the USA consisting of 22 members, among whom nine were somnambulists and 13 were unaffected. Genome-wide linkage analysis from gathered DNA samples revealed the following facts—(1) genetic locus for somnambulism is present at chromosome 20q12-q13.12; (2) sleep-walking may be transmitted as an autosomal dominant trait with reduced penetrance;
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DOI: http://dx.doi.org/10.5772/intechopen.102462

(3) seven among the nine somnambulists were males; (4) adenosine deaminase gene (ADA) is the most likely candidate gene in the chromosome 20q12-q13.12 linkage interval due to its association with SWS during with somnambulism occurs.

Since 1956, polysomnographic studies in somnambulist patients have been conducted in the USA, Canada, and a few European countries [7, 8, 20–32]. A chronological compilation of significant polysomnographic studies is presented in Table 1. Two

<table>
<thead>
<tr>
<th>Country</th>
<th>Year</th>
<th>Somnambulists</th>
<th>Controls</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Sample no.</td>
<td>Age (yr)</td>
<td>Sample no.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Range</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>USA</td>
<td>1956</td>
<td>34</td>
<td>n.r²</td>
<td>60</td>
</tr>
<tr>
<td>USA</td>
<td>1965</td>
<td>9</td>
<td>9–23</td>
<td>Nil</td>
</tr>
<tr>
<td>UK</td>
<td>1990</td>
<td>5</td>
<td>28.4 ± 7.8</td>
<td>352</td>
</tr>
<tr>
<td>USA</td>
<td>1991</td>
<td>8 [SW]²</td>
<td>18–51</td>
<td>Nil</td>
</tr>
<tr>
<td>Germany</td>
<td>1998</td>
<td>10</td>
<td>27.6 ± 3.4</td>
<td>n.r²</td>
</tr>
<tr>
<td>France</td>
<td>2000</td>
<td>3[SW]², 2[SW + ST]³</td>
<td>22–40</td>
<td>31.2 ± 2.2</td>
</tr>
<tr>
<td>Switzerland</td>
<td>2000</td>
<td>1</td>
<td>16</td>
<td>Nil</td>
</tr>
<tr>
<td>Poland</td>
<td>2005</td>
<td>1</td>
<td>26</td>
<td>1</td>
</tr>
<tr>
<td>Italy</td>
<td>2007</td>
<td>8</td>
<td>21–49</td>
<td>12</td>
</tr>
<tr>
<td>Canada</td>
<td>2008</td>
<td>30</td>
<td>16–47</td>
<td>Nil</td>
</tr>
<tr>
<td>France</td>
<td>2009</td>
<td>8 [SW]², 30[SW + ST]³</td>
<td>11–72</td>
<td>25</td>
</tr>
<tr>
<td>Switzerland</td>
<td>2013</td>
<td>10</td>
<td>66 ± 11.6</td>
<td>Nil</td>
</tr>
<tr>
<td>France</td>
<td>2014</td>
<td>12</td>
<td>27.4 ± 8.4</td>
<td>Nil</td>
</tr>
<tr>
<td>Czech Rep</td>
<td>2015</td>
<td>52</td>
<td>19–63</td>
<td>20</td>
</tr>
<tr>
<td>France</td>
<td>2017</td>
<td>89[SW]², 80[SW without SS]², 9[SW with SS]³</td>
<td>29.2 ± 7.1</td>
<td>32.8 ± 10.4</td>
</tr>
</tbody>
</table>

1n.r = not reported, presumed to be in late teens to 20s, as the subjects were naval recruits and Electronics school students.
2Patients suffered from sleepwalking only.
3Patients suffered from both sleepwalking and sleep terror.
4Patients suffered from sleepwalking without sexsomnia.
5Patients suffered from sleepwalking and sexsomnia.

Table 1.
Significant polysomnographic studies in somnambulist patients.
recognizable features revealed were, (1) Earlier studies, with two exceptions, did not have proper control groups; (2) Until 2015, the total number of somnambulist patients was less than 40. In the studies reported by Hurwitz et al. [21] and Oudiette et al. [28], the higher number of patients suffered from sleepwalking and sleep terror. The earliest study by Andre-Balisaux and Gonsette [6] in Belgium was omitted in Table 1, due to my lack of access in checking the complete text of the original paper. Among the specific environmental factors precipitating somnambulistic episodes in adults, prescription medication use [33], alcohol [34], emotional stress [35], sleep deprivation [27], and certain psychiatric trauma [36] have been suggested. As such, a multifactorial etiology for somnambulistic disorder deserves recognition.

3. Associated disorders

Somnambulism-associated disorders/syndromes are presented in Table 2. These include alcoholism [34], brain tumor [37], bulimia [38, 39], epilepsy [40–43],

<table>
<thead>
<tr>
<th>Somnambulism-associated Disorders/Syndromes</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcoholism</td>
<td>Himemiya-Hakucho and Fujimiya [34]</td>
</tr>
<tr>
<td>Brain tumor</td>
<td>Prashad et al. [37]</td>
</tr>
<tr>
<td>Bulimia</td>
<td>Guirguis [38]</td>
</tr>
<tr>
<td>Epilepsy</td>
<td>Pierce and Lipcon [40]</td>
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<td></td>
<td>Tinuper et al. [41]</td>
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<td></td>
<td>Cornejo-Sanchez et al. [42]</td>
</tr>
<tr>
<td></td>
<td>Duffau et al. [43]</td>
</tr>
<tr>
<td>Hyperthyroidism</td>
<td>Ajlouni et al. [44]</td>
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<tr>
<td></td>
<td>Pradalier et al. [47]</td>
</tr>
<tr>
<td></td>
<td>Lopez et al. [48]</td>
</tr>
<tr>
<td>Nocturnal hypoglycaemia</td>
<td>Bell [49]</td>
</tr>
<tr>
<td></td>
<td>Cebrian et al. [50]</td>
</tr>
<tr>
<td>Parkinson disease</td>
<td>Puryazova et al. [51]</td>
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<tr>
<td></td>
<td>Di Fabio et al. [29]</td>
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<td></td>
<td>Oberholzer et al. [52]</td>
</tr>
<tr>
<td>Sexsomnia</td>
<td>Dubessy et al. [32]</td>
</tr>
<tr>
<td>Sleep-disordered breathing</td>
<td>espa et al. [53]</td>
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<td></td>
<td>Guillaminault et al. [54]</td>
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<td></td>
<td>Guillaminault et al. [55]</td>
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<tr>
<td>Tourette's syndrome</td>
<td>Barabas et al. [56]</td>
</tr>
<tr>
<td></td>
<td>Jimenez-Jimenez et al. [57]</td>
</tr>
</tbody>
</table>

Table 2. Somnambulism associated disorders/syndromes.

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hyperthyroidism [44], migraine [45–48], nocturnal hypoglycemia [49, 50], Parkinson’s disease [29, 51, 52], sexsomnia [32], sleep-disordered breathing [53–55], and Tourette’s syndrome [56, 57]. In a cross-sectional case–control study conducted in Montpellier, France, with 100 adult sleepwalkers (55 men and 45 women, aged 18–59 years), Lopez et al. [48] found 44% suffered from chronic pain, compared to 20% of controls. Those sleepwalkers with chronic pain were more likely to be older and had higher daytime sleepiness. As one of the limitations in this study, Lopez et al. [48] had noted that pain sensitivity was not measured. Di Fabio et al. [29] reported that 9% of their 417 Parkinson’s disease patients were somnambulists, and concluded that sleepwalking in Parkinson’s disease is associated with depression, higher disease severity, and higher functional disability.

4. Brain cortex volume in somnambulists

Compared to age and gender-matched control group, in 14 drug-free polysomnographically confirmed adult somnambulists (age range 21–39 years), an MRI study reported by Heidbreder et al. [58] showed significant decreases of gray matter in the left dorsal posterior cingulate cortex and posterior midcingulate cortex. The mean disease duration of adult somnambulists was reported as 19.2 ± 7.7 years (range 6–33 years). Though this finding is of interest, it remains to be replicated [59] in other sleep laboratories with children somnambulists as well. Whether children somnambulists also show similar significant gray matter decrease in the cingulate cortex areas deserves clarification. This is because, while the prevalence of somnambulism peaks in childhood, its percentage decreases in adulthood.

5. Treatment strategies: pharmacological and other treatments

No drug has been developed specifically to cure somnambulism [60]. However, based on case histories, clonazepam—a tranquilizer of benzodiazepine class patented in 1960, at 0.25–0.5 mg dose to be taken 1–2 h before sleeping [61–63], imipramine—a tricyclic antidepressant [64], paroxetine—a selective serotonin reuptake inhibitor [65] and ramelteon, a melatonin receptor agonist, at a dose of 4 mg/day [66] have been suggested for successful treatments. Though clonazepam has been the most widely used medication for non-REM sleep parasomnias with reported success, its use has been restricted in France, for the reason that it may increase sleep-disoriented breathing and induce sedation the following day [32].

In a recent review on medication-induced somnambulism, Stallman et al. [33] had identified 29 medications that are possible triggers for sleepwalking. For convenience, these medications have been categorized into five classes as follows—(1) benzodiazepine receptor agonists and other gamma-aminobutyric acid (GABA) modulators, (2) antidepressants and other serotonergic agents, (3) antipsychotics, (4) β-blockers, and (5) “others,” including antibiotic ciprofloxacin.

Other types of treatments include hypnosis [67, 68], psychotherapy, aversive behavioral therapy [69], and the use of customized bed alarms. A recent review with 60 references by de Cock [70] is recommended for additional details on this theme.
6. Interdisciplinary relevance to medicine and law

While studying the records of 20 patients admitted to intensive care units, who had a diagnosis of self-injurious sleep behavior disorders, between 1982 and 1990, at the Minnesota Regional Sleep Disorders Center, USA, Schenck and Mahowald [62] recognized that three among the 20 suffered from chronic night terrors/sleepwalking abnormality. The influence of various stressors commonly found in the intensive care units (such as sleeping in an unfamiliar setting, sleep disruption/deprivation, medication administration/withdrawal, and psychologic distress) may aggravate the condition of sleepwalkers is one inference from this study. From a retrospective chart review study of patients older than 15 years, admitted to the emergency department at a university hospital in Berne, Switzerland, Sauter et al. [71] inferred that life-threatening injuries associated with somnambulism may occur. As such, when patients present with falls of unknown origin, the possibility of somnambulistic cause should be considered. The 11 trauma patients, Sauter et al. [71] encountered, had injuries identified as contused facial lacerations, head injury, contusion of ribs, cervical spinal dislocation, and paraplegia below the thoracic vertebral body.

Self-caused injuries [71, 72] notwithstanding, to a confused public, somnambulism seems to be a puzzling phenomenon that flirts between the boundaries of medicine and the law. The pros and cons of the rights and penalties for this class of sleep disorder patients who commit serious crimes, including homicides [73], during somnambulistic episodes, continue to be debated without any resolution [74–84]. A controversial issue is the use of somnambulism defense in cases involving alcohol-induced homicides. In reviewing the scientific evidence and forensic considerations, Pressman et al. [85] had inferred that a defense of alcohol-induced sleepwalking or confusional arousal becomes a very attractive legal strategy with the potential of acquittal for the defendant. This, despite the fact “there is no direct experimental evidence that alcohol predisposes or triggers sleepwalking or related disorders.”

Quite a number of reports have appeared in the last three decades, implicating somnambulism with sexual misconduct as well [86–88]. Among the nine cases of sleep-related violence and nine cases of sexual behavior in sleep, published between 1980 and 2012, the sleepwalking defense was proposed in 11 among these 18 cases [89]. In their review of these 18 cases, Ingravallo et al. [89] indicated that the trial outcome was in favor of the defendant in 14 of 18 cases. As the forensic evaluations widely differed from case to case, they had concluded that “an international multidisciplinary consensus for the forensic evaluation of sleep-related violence and sexual behavior in sleep need to be developed as an urgent priority.” Though clinical descriptions of somnambulists with sexsomnia as anecdotal case reports show an increase in this century [32], probably due to its titillating factor in the digital mass media, it is not easy to replicate somnambulist-sexsomnia episodes in hospitalized setting, except on unusual conditions that such patients have a bed sharer of the same sex or opposite sex, during the observation period.

7. Conclusions

As somnambulism has not been recorded in non-human primates, no animal model exists to study somnambulism under controlled settings for elucidation of its biochemical mechanism of origin [90]. As such, the available pharmacological treatments have to be considered as the scatter-shot approach of case studies without
appropriate controls [91]. Thus, partial or total alleviation of somnambulistic symp-
toms in children and adults deserves the attention of health researchers. Adequately
powered, well-designed clinical trials remain the need of the moment. The plea made
by Chinthapalli [92], an associate editor of the British Medical Journal, that medical
schools should consider sleep medicine education seriously in their curriculum also
deserves recognition.

Conflict of interest

I declare no conflict of interest.

Notes

I became interested in the somnambulism phenomenon while serving as a Visiting
Professor at Kyoto University, Primate Research Institute, Inuyama (Japan) and
studying the activity-sleep behavior of New World and Old World Monkeys, between
2002 and 2006. I appreciate the discussions I have had with veterinarians, Dr. Kiyoki
Matsubayashi and Dr. Juri Suzuki, during that period.

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