Sleepwalking in Patients With Parkinson Disease

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Objective: To report the occurrence of adult-onset (de novo) sleepwalking in a series of 6 patients with idiopathic Parkinson disease (PD).

Design: Case series.

Setting: Outpatient clinic for movement disorders.

Patients and Methods: Of 165 consecutive patients with PD seen for 2 years, 6 patients with adult-onset sleepwalking were identified. These patients underwent a systematic clinical assessment of their extrapyramidal and sleep problems, which included standard questionnaires, clinical examination, and estimation of PD severity (motor score of the Unified PD Rating Scale and Hoehn and Yahr stage). Five of 6 patients had a video-polysomnography recording that was scored according to international criteria.

Results: Patients included 3 women and 3 men with a mean (±SD) age of 66±12 years (range, 46-78 years). The mean (±SD) Unified PD Rating Scale score was 25±9 (range, 10-35) and the mean (±SD) Hoehn and Yahr stage was 2.5±1.0 (range, 1.0-4.0). Medications in these patients included levodopa (n=6), dopamine agonists (n=4), selective serotonin reuptake inhibitor antidepressants (n=3), and hypnotics (n=3). All patients had at least 1 concomitant sleep-wake disorder, including rapid eye movement sleep behavior disorder (n=4) and insomnia (n=4). In 2 of 6 patients, the latency between onset of PD and appearance of sleepwalking was more than 4 years.

Conclusion: Neurodegenerative changes associated with PD at the brainstem level can affect the “ascending” control of state transition (leading to dissociated arousals from non–rapid eye movement and/or rapid eye movement sleep) and the “descending” control of locomotion and muscle tone, together giving rise to various sleep-associated behavioral disturbances including sleepwalking, rapid eye movement sleep behavior disorder, and overlap parasomnia.

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Sleepwalking (SW) corresponds to a complex sleep-associated behavior that includes locomotion, mental confusion, and amnesia regarding the episode. Injuries and acts of violence may also occur.1 About 10% of children and 2% to 3% of adults experience SW, but only 0.6% of adults who reported never having walked in their sleep in childhood did so later in life.2 Most commonly, SW appears in the context of a non–rapid eye movement (NREM) sleep parasomnia. Less commonly, SW can appear in the context of nocturnal epilepsy, parasomnia overlap disorder—in which rapid eye movement (REM) and NREM features are combined, and malingered. Sleepwalking is thought to arise from dissociation between motor and mental arousal, as suggested also by a study using single-photon emission computed tomography.3

To our knowledge, an association of SW with Parkinson disease (PD) was reported only sporadically in the literature (in abstract form in 3 patients4 and in a recent series of 100 consecutive patients5). Night walking was also reported in 10 of 93 patients with REM sleep behavior disorder (RBD) of different origin, including PD. Five of these 10 patients had an underlying unspecified neurodegenerative disorder.6 We describe 6 patients with PD and SW.

REPORT OF CASES

Adult-onset SW was reported by 6 of 165 consecutive patients with PD (3.6%) seen in the outpatient clinic for movement disorders at the Department of Neurology,
University Hospital of Zurich, for 2 years. These patients underwent a systematic clinical assessment of their extrapyramidal and sleep problems, which included standard questionnaires, the motor score of the Unified PD Rating Scale, and the Hoehn and Yahr stage. A video-polysomnography (PSG) was recorded in 5 of 6 patients and scored according to international criteria.

Patients included 3 women and 3 men with a mean±SD age of 66±12 years (range, 46-78 years) (Table). The mean Unified PD Rating Scale score when “on” was 25±9 (range, 10-35) and the mean Hoehn and Yahr stage was 2.5±1.0 (range, 1.0-4.0). Medications included levodopa (n=6), dopamine agonists (n=4), selective serotonin reuptake inhibitor antidepressants (n=3), and hypnotics (n=3).

Sleepwalking had appeared de novo in adulthood in all patients. All patients had at least 1 concomitant sleep-wake disorder, including RBD (n=4), insomnia (n=4), sleep apnea (n=1), periodic limb movements in sleep (n=2), and restless legs syndrome (n=1).

### CASE 1

A 46-year-old woman developed SW at the age of 43 years, 8 years after the onset of PD. She denied SW as a child and she had no family history of SW. She reported the regular use of zolpidem tartrate for more than 10 years before the onset of SW. The frequency of SW was about once every night. She also had “eating attacks” at night (she repeatedly found her mouth and fingers stained with chocolate in the morning) about which she had no recollection. She was known to have insomnia, restless legs syndrome, depression, and anxiety since the age of about 35 years. She had no history of enacted dreams. Video-PSG showed a highly fragmented sleep, excessive limb twitching, and head twists during REM sleep. With treatment with topiramate, 100 mg each night, the SW disappeared.

### CASE 2

A 60-year-old woman with a 21-year history of PD developed SW after bilateral pallidothalamic tractotomy at age 52 years. She denied SW as a child but her son had a history of SW. The frequency of SW was about once per month and included episodes of walking around her house. She was known to have a depressed mood and mild disturbances of executive and memory functions since her surgery. She had a history of enacted dreams almost every night for the last 8 years, that is, concomitant with the onset of SW. Video-PSG showed excessive limb twitching, REM sleep without atonia, and 1 sudden arousal from deep NREM sleep.

### CASE 3

A 72-year-old woman began SW at age 65 years, 4 years after the onset of PD. She denied SW as a child and had no family history of SW. The frequency of SW was about 2 to 3 times per week and included episodes during which she prepared tea or hot milk. She had no recollection of these episodes. She experienced light bruises repeatedly during SW. The patient reported occasional visual hallucinations at night and had been suffering from depression, insomnia, nightmares, and mild disturbances of memory functions for about 10 years. She refused to undergo video-PSG. With treatment with clozapine, 25 mg each night, her SW disappeared.
CASE 4

A 78-year-old man developed SW at age 76 years, 19 years after the onset of PD. He denied SW as a child and had no family history of SW. The frequency of SW was 2 to 3 times per week. According to his wife, during these episodes he would take off his pajamas, leave his house, take a shower, or jump around “like a frog.” Since age 72 years, he had begun to act out his dreams (screaming and punching). The patient also reported the recent onset of hallucinations at night. He had had depression, insomnia, and excessive daytime sleepiness (Epworth Sleepiness Scale, 12 of 24 possible points) for at least 3 years. VideopSG showed fragmented sleep, excessive twitches, and REM sleep with incomplete REM atonia and severe sleep apnea syndrome. Confusional arousals from light NREM sleep were observed.

CASE 5

A 77-year-old man developed SW together with dream-enacting behaviors at the onset of PD at age 59 years. He denied SW as a child and had no family history of SW. The frequency of SW was 2 to 3 times a year. During 1 of these episodes, he fell, hit his head on the edge of a table, and woke up with a bleeding nose. Video-PSG documented sleep fragmentation, periodic movements in sleep, excessive twitches, and REM sleep without atonia. His SW ceased spontaneously.

CASE 6

A 63-year-old man developed SW together with dream-enacting behaviors at the onset of PD at age 59 years. He denied SW as a child and had no family history of SW. His mother, brother, and sister also suffered from PD. The frequency of SW was up to 3 to 4 times per night; he used to wake up in different rooms of his apartment or on the balcony, and did not remember how he had gotten there. Insomnia, depression, memory problems, and nighttime hallucinations began at the onset of PD. Because his tolerance to levodopa and dopamine agonists was poor, he continued taking low medication doses. Repeated video-PSG documented sleep fragmentation, periodic leg movements in sleep, but no dream-enacting behaviors or even increased muscle tone in REM sleep were observed. His SW, dream-enacting behaviors, and hallucinations ceased spontaneously.

COMMENT

We report herein the existence of adult-onset (de novo) SW in 6 patients with PD. To our knowledge, this condition has been reported only sporadically in the literature.4,5

Although SW usually appears to be a late manifestation of moderate to severe PD, the onset of SW at the onset of PD was reported by 2 of our patients and by 3 other patients in the literature.4 The severity of SW is highly variable, with a frequency ranging from 3 to 4 episodes per day to only a few episodes per year. In most cases, an RBD was also found in the patient's history or on video-PSG. Other sleep disorders (insomnia, restless legs syndrome, and periodic limb movements in sleep), depression, mild cognitive deficits, and visual hallucinations were less commonly observed. In 4 patients, SW ceased either spontaneously (n=2) or after treatment with clozapine (n=1) and topiramate (n=1).

The co-occurrence of SW and RBD in most of our patients raises 2 possible pathophysiologic explanations.

First, SW may represent an extreme, severe manifestation of motor dyscontrol in RBD and therefore belongs to the spectrum of this REM parasomnia. Night walking was indeed reported in 10 of 93 patients with RBD of different origins including PD.6 Sleepwalking arising in REM sleep was, however, not documented with video-PSG in this or other reports in the literature. In a recent detailed analysis of 100 consecutive patients with PD and a history of RBD, SW was observed in 1 patient in whom RBD could not be demonstrated with video-PSG.7 In addition, RBD appears to be typically associated with short, jerky, repetitive, and rough movements that contrast with the description of not only longer but also more elaborate and physiologic motor behaviors (such as eating or preparing drinks) in our patients. Finally, 2 of our patients had no history and/or video-PSG evidence of RBD, thus suggesting that RBD is not mandatory for the appearance of de novo SW in patients with PD.

Second, SW and RBD may arise from NREM and REM sleep, respectively, reflecting a common underlying disturbance of motor control during sleep. The coexistence of SW and RBD during NREM and REM sleep, respectively, termed overlap parasomnia, was originally reported by Schenck et al7 in 33 patients. In that series, although an underlying disorder was found in 11 patients, none had PD. In the course of neurodegenerative diseases, overlap parasomnia was rarely documented; reports include a patient with Machado-Joseph disease.8

The pathophysiologic characteristics of SW in PD remain unclear. Various brainstem structures involved in locomotion, muscle tone regulation, and arousal (including the pedunculopontine nucleus) are known to be affected in PD.9,10 We postulate that PD-associated neurodegenerative changes at the brainstem level can affect the “ascending” control of state transition (leading to dissociated arousals from NREM and/or REM sleep) and the “descending” control of locomotion and muscle tone, together giving rise to various sleep-associated behavioral disturbances, including SW, RBD, and overlap parasomnia.

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We plan to utilize videos as part of published papers that highlight and provide convincing information about the observational and visual features of a patient’s neurologic findings. Please refer to Instructions for Authors for instructions on submitting video presentations.

REFERENCES