Improvement of Sleep Hypopnea by Antiparkinsonian Drugs in a Patient with Parkinson’s Disease: a Polysomnographic Study

Tomokatsu YOSHIDA, IchiyO KONO, Kenji YOSHIKAWA, Hiroaki HASHIMOTO*, Hidehiko HARADA**, and Masanori NAKAGAWA

Abstract

An 80-year-old man was admitted to our hospital because of bradykinesia, muscle rigidity and respiratory dysfunction during sleep. Concerning bradykinesia and muscle rigidity, we diagnosed him as the early/moderate stage of Parkinson’s disease without autonomic dysfunction. Polysomnography (PSG) showed a series of obstructive hypopneas and apneas. After administration of antiparkinsonian drugs, rigidity of the neck and trunk was diminished along with a drastic decrease in hypopnea on PSG. We consider that sleep hypopnea in this patient is caused by involvement of the striated musculature surrounding the upper-airway and/or rigidity in the trunk. These conditions are treatable with antiparkinsonian drugs.

(Key words: sleep hypopnea, Parkinson’s disease, antiparkinsonian drugs, polysomnography)

Introduction

There are several reports on respiratory function or the effects of antiparkinsonian drugs on respiratory dysfunction in Parkinson’s disease (PD) patients (1–15). However, it has been suggested that sleep apnea and hypopnea syndrome have little relation to PD (16, 17).

Vincken et al suggested that involvement of the striated musculature surrounding the upper airway is a cause of upper airway obstruction and induces respiratory dysfunction in PD patients (1). However, the effects of antiparkinsonian drugs remain controversial: some authors reported improvement of respiratory function with antiparkinsonian drugs (8–10), while others reported that respiratory dysfunction did not improve after administration of levodopa (11) or that levodopa itself induced respiratory dysfunction in patients with PD (12–15).

We report a patient with PD accompanied by sleep hypopnea and apnea, whose respiratory dysfunction was ameliorated by antiparkinsonian drugs. To evaluate the effects of antiparkinsonian drugs for respiratory dysfunction during sleep, polysomnography (PSG) was performed before and after therapy.

Case Report

On October 15, 2002, an 80-year-old man was admitted to our hospital because of bradykinesia and muscle rigidity preceded by respiratory dysfunction during sleep. The patient has snored loudly during sleep for the past 20 years and he often dozes in the afternoon. His daughter noticed that his breathing often stopped for several seconds during sleep. Thus, he consulted a physician in our hospital. Medical examination showed bradykinesia and rigidity, so the physician supposed parkinsonism and consulted a neurologist. Thereafter, he was admitted to our hospital for evaluation of parkinsonism and abnormal sleep.

He was 164 cm tall with a body weight of 51 kg. Blood pressure was 138/90 mmHg. The pulse was 72/min. His face was slightly ruddy and mask like. Chest and abdominal examination did not show any abnormal findings. There was no stridor or wheeze heard in the neck. His voice was small. The limbs, neck and trunk were rigid like a cogwheel. The rigidity was marked. Walking was slow, with shortened stride length, and decreased arm swing. Tremor at rest, loss of pos-
YOSHIDA et al

Sleep stage

REM
MOV
AWK
1
2
3
4

Position

F: face down, L: left, R: right, B: on one’s back.

Oxygen saturation

SaO2

Snoring

Apnea and hypopnea


Time 21:00

Figure 1. Polysomnogram performed before therapy showed periodic episodes of sleep hypopnea and obstructive apnea.

Figure 1. Polysomnogram performed before therapy showed periodic episodes of sleep hypopnea and obstructive apnea.

tural reflexes and the freezing phenomenon were not observed. He did not complain of constipation. He was oriented and his memory was preserved. There was neither agnosia nor apraxia. He often took naps.

Routine laboratory tests including blood cell count and biochemical examinations were all normal. Chest X-ray film did not show any abnormal findings. Only premature atrial contraction was shown on electrocardiogram. Echocardiogram was normal. Brain magnetic resonance imaging showed a few ischemic lesions in the deep white matter. R-R interval variance in ECG was normal. Hypotension was not induced by head-up tilt test.

On the fluoroscopic examination of the pharynx and larynx, enlarged tonsils, adenoid or vocal cord abductor paralysis was not shown. He did not have any dysostogenesis such as microgenia.

Based on these findings, we diagnosed him as the early/moderate stage (Hoehn-Yahr II) of PD without autonomic dysfunction.

On November 1, 2002, we started administration of benzeraside/levodopa and cabergoline, and increased the daily dose gradually until the optimal dosage was reached: benzeraside/levodopa 25 mg/100 mg three times per day, cabergoline 1.0 mg once a day. Muscle rigidity of the limbs and neck became mild, walking was faster and arm swing increased. PSG was performed before and after the start of antiparkinsonian drugs (October 28 and December 4). Hypopnea was defined as a set of the following conditions: more than 50% flow reduction compared with that in the awake state, minor desaturation of arterial oxygen (<3%) and decreased thoracoabdominal movement (18). Apnea was defined as a cessation of airflow at the nose and mouth lasting at least 10 seconds (19).

PSG performed before medication showed a number of hypopneas and obstructive apneas (Fig. 1): Hypopnea index (HI) was 19.9/hour, apnea index (AI) was 25.2/hour and apnea-hypopnea index (AHI) was 45.1/hour. PSG performed after medication showed a drastic decrease in hypopneas (Fig. 2): HI was 2.1/hour, AI was 26.8/hour and AHI was 28.9/hour. In addition, he succeeded in turning over in his sleep.
Sleep Hypopnea in PD

Figure 2. Polysomnogram performed after therapy showed a marked decrease of sleep hypopnea, while sleep apnea did not show any improvement. In addition, he succeeded in turning over in his sleep.

Discussion

Apps et al studied respiration during sleep in patients with PD and patient’s with Parkinsonism with autonomic disturbance (16). They observed tachypnea in patients with PD, which was also shown when awake. However, the respiratory rate decreased during sleep, with no evidence of sleep apnea or hypopnea. In contrast, patients with parkinsonism with autonomic disturbance showed frequent central and obstructive apneas. They speculated that tachypnea in patients with parkinsonism without autonomic disturbance was related to the disease itself, to the effects of drugs, to the presence of respiratory disease, or to chest wall rigidity and stiffness, and concluded that sleep apnea was not related to PD. However, all of the patients in the study were receiving optimal therapy with antiparkinsonian medication. In the present case, PSG showed both sleep hypopnea and apnea, and antiparkinsonian medication drastically decreased the hypopnea while apnea was not improved. These findings suggested that hypopnea was substantially related to PD in our case.

Vincken et al tested pulmonary function in patients with extrapyramidal disorders (1). The flow-volume contour in many patients was abnormal, and direct fiberoptic visualization of the upper airway showed flow alternation due to involvement of the striated musculature surrounding the upper airway rather than involvement of chest wall muscles. They concluded that the striated musculature surrounding the upper airway was frequently involved and this caused upper airway dysfunction.

As for the beneficial effect of antiparkinsonian drugs on respiratory dysfunction in patients with PD, Nakano et al reported improvement of respiratory function with levodopa compared with placebo (9), and Vincken et al reported that reversibility of a pattern of upper-airway obstruction was shown in sequence flow-volume curves after administration of levodopa (10). Herer et al reported that levodopa administration in patients with PD induced significant variations in peak expiratory flow and upper-airway obstruction ratios (8).

In the present patient, administration of antiparkinsonian drugs improved the neck rigidity, and he succeeded in turning over in his sleep. There was no naso-laryngeal abnormality found in the upper respiratory tract. Therefore, we consider that sleep hypopnea is caused by PD itself, such as
involvement of the striated musculature surrounding the upper airway and/or rigidity of the trunk, which were diminished by the medication.

In contrast, obstructive apnea was not improved by the medication. The present patient had shown several symptoms of sleep apnea syndrome such as snoring during sleep and excessive daytime sleep for 20 years, therefore, obstructive apneas might not have a direct relation to PD.

In conclusion, our patient with PD without autonomic disturbance shows sleep hypopnea and apnea, and only hypopnea is improved by antiparkinsonian drugs, which does not concur with previous reports.

References