

Chapter 23

The Quantitative EEG Change in Parkinson's Disease

Akihiko Morita

Nihon University School of Medicine, Japan

ABSTRACT

In this study, the author presents results regarding the quantitative EEG (qEEG) in Parkinson's disease (PD). This study is the first to assess the distribution of a qEEG change in a large number of PD patients. Based on the pathological changes observed in PD, PD patients present significantly diffuse slowing in their qEEG compared with normal control subjects. Moreover, slowing of the qEEG in each electrode corresponded significantly with the progression of PD and cognitive impairment. This finding is consistent with Braak's findings in PD (Braak, H. et al., 2004).

BACKGROUND

Parkinson's disease (PD) is characterized by its clinical features, which include bradykinesia, rigidity, tremor, and postural instability. However, patients with PD also have a range of non-motor symptoms and motor complications that are related to the disease. The non-motor symptoms are common at all stages of PD and represent a key determinant of quality of life. These symptoms include neuropsychiatric, autonomic, gastrointestinal, and sensory disturbances, as well as sleep disorders. Neuropsychiatric symptoms of PD include anxiety, anhedonia, apathy, depression, and dementia, among others (Aarsland, D. et al., 1999).

Diffuse or localized slowing of the electroencephalogram (EEG) in a large number of patients with PD, as evaluated by visual inspection, has been previously described (Neufeld, M. Y. et al., 1988; Yeager, C. L. et al, 1966). Yeager and colleagues (1966) reported that 36.3% of 223 PD patients exhibited abnormal recordings on the EEG, which were characterized by diffuse slowing, localized slowing, or both. Moreover, Neufeld and colleagues (1988) found that a mild slowing on the EEG was present in 34% of 128 patients with PD. However, reports assessing the relationship between PD patients and EEG changes using the quantitative EEG (qEEG) technique were limited.

In this study, we report our results on the assessment of the difference in qEEG recordings between PD patients and age-adjusted normal con-

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trol (CTRL) subjects (Serizawa, K. et al., 2008), the relationship between disease progression in PD patients and slowing of the EEG (Morita, A. et al., 2009), and lastly, the relationship between progression of cognitive impairment in PD and slowing of the EEG (Morita, A. et al., 2011). These results provide the first evaluation of qEEG findings assessed in a large number of PD patients following confirmation of the absence of intracerebral ischemic lesions by cranial magnetic resonance imaging (MRI).

METHODS

Subjects

The clinical diagnosis of sporadic PD was made according to the UK PD Brain Bank criteria (Gibb, W. R. & Lees, A. J., 1988). Based on clinical features and neuroradiological findings, including brain computed tomography (CT) and MRI, which were obtained at > 12 months after onset, we excluded other forms of parkinsonism, such as (1) dementia with Lewy bodies (DLB) (Geser, F. et al., 2005; McKeith, I. G. et al., 1996), (2) drug-induced parkinsonism, (3) vascular parkinsonism, and (4) atypical parkinsonism with absent or minimal responses to anti-parkinsonian drugs. All patients were scanned to obtain T1- and T2-weighted images, fluid-attenuated inversion recovery (FLAIR) images, and diffusion images by a cranial MRI (1.5-T Siemens Magnetom Symphony, Munich, Germany). We also included patients without any intracerebral ischemic changes, including asymptomatic lacuna or slight periventricular hyperintensity on T2 and FLAIR images, in accordance with the reported classification of PVH (Fazekas, F. et al., 1987). At more than 12 months after onset, the subjects had exhibited good responses to anti-Parkinsonian drugs and did not have a history of visual hallucinations

or fluctuations in their cognitive ability, which suggested clinical diagnosis of DLB.

PD progression was assessed by the Hoehn and Yahr staging scale (Hoehn, M. M. & Yahr, M. D., 1967). Cognitive function in PD patients was assessed using the Mini-Mental State Examination (MMSE), which is based on the Diagnostic and Statistical Manual of Mental Disorders, 4th ed. (DSM-IV) criteria for dementia in accordance with a previously reported study (Burn, D. J. et al., 2006).

The CTRL subjects were also defined as participants without any organic intracerebral disease who were ≥ 60 years old and exhibited no intracerebral ischemic lesions on a cranial MRI.

Participants who were being treated with drugs that influenced qEEG, such as anti-anxiety or psychotropic drugs, were excluded prior to the study.

EEG Recordings and qEEG Analysis

The EEG recordings and qEEG analysis were performed as follows: the EEG, directly after checking for alpha blocking, was obtained from each patient in a resting awake condition with their eyes closed. Eye-movement electrodes were used to confirm and identify the eye movements of early drowsiness. The EEG was recorded on a magnetic optical disk from 16 electrode locations according to the 10-20 International system using a digital EEG instrument (Neurofax EEG-1100, Nihon Kohden, Tokyo, Japan) and referenced to the ipsilateral earlobes. A minimum of 60 sec of qEEG data was visually selected for each patient and digitized at 200 Hz (Nuwer, M. R. et al., 1999). A high-frequency filter was set at 60.0 Hz with a time constant of 0.3 seconds. Thirty or more epochs with a 2.56-sec duration were collected from the subsequent resting period with the eyes closed for qEEG analysis. The procedure used for analysis involved application of fast Fourier transformation of the collected EEG signals with

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