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Comparison of Quantitative Electroencephalogram During Sleep in Depressed and Non-Depressed Patients with Parkinson's Disease

Authors' Contribution:
Study Design A
Data Collection B
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Data Interpretation D
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Literature Search F
Funds Collection G

ABCEF 1,2 Ke Liu
B 1,2 QinYing Ma
A 1,2 MingWei Wang

1 Department of Neurology, The First Hospital of Hebei Medical University, Shijiazhuang, Hebei, P.R. China
2 Brain Aging and Cognitive Neuroscience Key Laboratory of Hebei Province, Shijiazhuang, Hebei, P.R. China

Corresponding Author: MingWei Wang, e-maiL: ben0521@126.com
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Background: Depression is one of the most important factors affecting quality of life in Parkinson's patients. Most research on Parkinson's disease with depression has focused on neuroimaging, and there have been few quantitative electroencephalogram studies. Sleep is a biomarker for depression; therefore, the aim of this study was to identify differences in quantitative electroencephalograms during sleep in depressed and non-depressed patients with Parkinson's disease.

Material/Methods: We assessed 38 Parkinson's disease patients (26 depressed patients, 12 non-depressed patients) and 20 normal subjects using the Geriatric Depressive Scale for Depressive Symptoms and quantitative electroencephalogram analysis of amplitude of different frequency bands in different sleep stages using Met-lab software and Fast Fourier Transformation.

Results: Non-rapid eye moment 2 and the Frontal 4 Electrode amplitude in the delta and theta ranges were progressively and significantly greater in the depressed-Parkinson's disease group ($p<0.05$) than in the control group. In the depressed Parkinson's disease group, from the comparison of non-rapid eye moment 2 and rapid eye moment, in Frontal 4 the amplitude in the delta ranges of non-rapid eye moment 2 was greater than in the non-depressed group, and in Central 3, Central 4, Occipital 1, and Occipital 2, the amplitudes in the beta ranges of rapid eye moment were greater ($p<0.05$) than in the non-depressed group.

Conclusions: The higher amplitude in theta in frontal areas in NREM2 and the higher amplitude in beta in parietal and occipital lobe areas in REM relative to NREM2 were significantly different in depressed and non-depressed patients with Parkinson's disease.

MeSH Keywords: Depression • Electroencephalography • Parkinsonian Disorders • Sleep

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Background

With population aging, Parkinson's disease (PD) has become a common neurodegenerative disease in the elderly [1]. Its motor symptoms (MS) include quiescent tremor, muscular rigidity, and movement retardation, and gait and posture abnormalities are common. In recent years, pathology research has led to the recognition that PD still has many non-motor symptoms (NMS), among which, depression is one of the most common. In general, the incidence of depression in PD is 40–50% [2], but depression has an extremely important effect on quality of life [3], so the early diagnosis and treatment of depressed PD is of great significance.

The diagnosis of depressive symptoms in PD remains difficult, particularly because signs and symptoms of depression overlap with motor and other non-motor symptoms. The pathogenesis of PD associated with depression is unclear. It is probable that dyskinesia leads to depression in PD [4]. Nevertheless, some studies have indicated that the mesolimbic dopamine system and midbrain cortical pathways are involved in PD with depression [5,6]. Neuroimaging studies on depressed PD focused on neuroimaging, have shown that patients with PD associated with depression also often have atrophy of the frontal lobe [7]. In addition, the distribution of brain perfusion imaging is also considered as a potential biomarker for PD associated with depression [4], and increased metabolism of the amygdala was found in PD with depression [8]. Blood-based biomarkers are of increasing interest in PD, and there is evidence that lower plasma levels of peripheral levels of serotonin (5-HT) and its metabolite, 5-hydroxyindoleacetic acid (5-HIAA), in PD patients is associated with more severe depression [9]. In addition, a diffusion magnetic resonance imaging (MRI) connection and symmetry study demonstrated that the prominent circuits involved in negative emotion recognition are impaired in comorbid depressive symptoms in PD [10].

Sleep is an important and complex biological process in humans. Its occurrence is closely related to certain structures in the central nervous system and to the role of various transmitters [11]. Changes in sleep patterns are a sensitive biological marker of variations in brain function. Sleep recording polysomnography (PSG) offers valuable information about brain activity in many mental disorders. Previous research has shown that sleep can be a biomarker for depression [12]. With respect to sleep electroencephalograms (EEG) in depressed patients with PD, previous studies have found shortened rapid eye moment (REM) sleep latency [13], but there has been little detailed analysis of quantitative EEG data during sleep in patients with depressed PD. We speculate the depressed PD patients have neurotransmitter transformation; therefore; to some extent, the brain wave activity changes accordingly. The aim of the present study was to examine differences in sleep quantitative EEGs obtained from depressed and non-depressed patients with PD to assess the value of the neurophysiological method and to discover the pathophysiological mechanism of the disease.

Material and Methods

The Ethics Committee of the First Hospital of Hebei Medical University has approved the study (approval no. HBYKDX-FH-N-1356).

Participants

We recruited 38 PD inpatients or outpatients from of the First Hospital of He Bei Medical University, consisting of 26 PD patients with depression compared to 12 patients without depression. The inclusion criteria were: (1) fulfilled the UK PD Society Brain Bank diagnostic criteria for PD [14], and (2) did not undergo surgical treatment with deep brain stimulation. The exclusion criteria were: (1) presence of dementia according to the Movement Disorders Society criteria [15]; (2) presence of psychiatric comorbidity or any other brain central nervous system (CNS)-related disorder; and (3) patients with moderate or severe dementia (Mini-Mental State Examination (MMSE) ≤24). All patients were taking appropriate medications for the treatment of PD, including dopaminergic agonists, levodopa, and others: 32 were taking Madopar or Sinemet, 8 were taking pramipexol, 6 were taking piribedil, 4 were taking selegiline, and 1 was taking entacapone. None of them were using antidepressants, antipsychotics, or sedatives. The mean levodopa equivalent daily dose of each patient group is given in Table 1. Disease and motor severity were assessed in Parkinson's disease patients using the Hoehn and Yahr scale [16]. The HY scale is the most commonly and widely used scale to describe the severity of motor function in patients with PD [17]; it provides a comprehensive assessment of clinical features and impaired motor function [18] and is more practical and easier to use. Twenty healthy volunteers with similar age and sex distributions to PD patients were enrolled as a control group. Healthy controls did not exhibit signs of movement disorder in the past and on examination.

Neuropsychiatric measurement

Depressive symptoms were evaluated using the Geriatric Depressive Scale (GDS). GDS represents the core of depression in old age, with 30 entries. Each of the 30 items marks a score of 1 for depression, ranging between 0 and 30. Ten of the 30 entries are scored in reverse order with the answer "no" indicating depression, and 20 items are scored in positive order with the answer "yes" indicating the existence of depression. Scores greater than 14 are used as criteria for

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Table 1. Clinical features of the control group and of the subgroups with Parkinson's disease.

	d-PD	nd-PD	Control	p (ANOVA)
Subjects	26	12	20	–
Age (yr)	67.06±8.65	66.22±5.97	67.07±0.82	0.787
Sex (Male/Female)	14/12	7/5	11/9	0.52
Education (yr)	14.2±4.3	14.3±4.1	14.4±3.9	0.98
Duration of PD (yr)	5.24±4.48	4.00±3.15	–	0.443
Age of onset (yr)	61.81±7.77	62.17±7.89	–	0.904
Hoehn-Hahr scale	1.66±0.76	1.72±0.75	–	0.833
UPDRS III (on)	21.8±11.5	20.2±10.8	–	0.64
LEDD (mg)	428.6±254.1	376.2±248.9	–	0.23
GDS	18.03±6.78	6.22±1.48	–	0.000*

GDS – Geriatric Depressive Scale; d-PD – depressed-Parkinson's disease; nd-PD – non-depressed Parkinson's disease, UPDRS – Unified Parkinson's Disease Rating Scale, LEDD – Levodopa equivalent daily doses. * Statistically significant (p<0.05).

assessing depression [19], with higher scores indicating more severe depression.

Polysomnographic recording

Polysomnographic (PSG) EEG data were analyzed according to the American Academy of Sleep Medicine (AASM) standard [20]. The PSG montage includes frontal (F3, F4), central (C3, C4), and occipital (O1, O2). EEG leads are linked with ears reference at 10 kilohm (kΩ) resistance, a bilateral electro-oculogram, and chin electromyographic recordings. Respiration was monitored using nasal airflow and chest or abdomen movement by wearing a pectoral girdle. PSG was recorded with a Grass polygraph (amplifier gain 10 000; bandpass 0.3–100 Hz), and signals were digitized at a sampling rate of 256 Hz or512 Hz using Harmonie software (Stellate Systems, Montreal, Canada). PSG variables included sleep latency and efficiency, time and number of awakenings, and sleep stage percentages.

Quantitative EEG analysis

All quantitative EEG analyses were performed on derivations F3, F4, C3, C4, O1, and O2. For amplitude analyses of non-rapid eye moment 2 (NREM2) and REM, the data generated by EEG signals extraction were exported in the form of EDF file format from the computer and using Met-lab software and Fast Fourier Transformation method calculation. Fast Fourier Transformation Formula for $X(k)=\Sigma n=\{0, N-1\}\times(n)\ e^{-j2\pi/NNK}$. Met-lab Fast Fourier Transformation code for y=FFT(x). We analyzed and compared sleep data from different groups at the same period and different sleep periods. Amplitude analyses were computed for the following frequency bands: delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), and beta (13–30 Hz).

Statistical analysis

SPSS software version 17.0 was used for statistical analysis. Measurement data are expressed as the mean ± standard deviation. One-way analyses of variance (ANOVA) was performed to assess differences between PD patients with depression, PD patients without depression, and controls on demographic, clinical, polysomnographic, and quantitative EEG data. Non-parametric Wilcoxon symbols test was used for abnormally distributed variables. Statistical significance was set at p<0.05.

Results

Table 1 shows the distribution of the individuals in the study group and the control of their clinical features. There were no statistically significant differences between the groups with respect to age, education, age at onset, duration of disease, or grading on the Hoehn-Yahr scale [16], but the depressed PD group had higher GDS scores (P=0.000).

Table 2 shows the variance of polysomnographic characteristics between the PD subgroups and the control group. There was a statistically significant difference between the depressed PD group and the control group. The depressed PD group had long wake-up time (P=0.000), increasing NREM2 percentage (P=0.000), and decreasing NREM1 percentage (P=0.000) and REM percentage (P=0.001).

Table 3 shows the results for the analysis of variance in NREM2 between the PD subgroups and the control group with respect to the EEG amplitude of F3, F4, C3, C4, O1, and O2 of different frequency bands. Results in Table 3 show that the F4 electrode

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Table 2. Polysomnographic characteristics of the control group and of the subgroups with Parkinson's disease.

	d-PD	nd-PD	Control	P
Sleep efficiency	46.82±21.92	45.52±18.11	37.6±27.49	0.146
Sleep latency	73.89±79.77	113.5±91.94	99.49±69.99	0.169
Wake up of time	172.77±107.19	120.92±95.41	48.86±54.68	0.000*
Number of awakening	22.45±17.82	16.25±13.73	14.46±11.23	0.28
N1 percentage of sleep	22.8±20.69	36.41±30.42	51.66±27.65	0.000*
N2 percentage of sleep	52.15±24.53	42.4±26.31	25.31±26.21	0.000*
N3 percentage of sleep	13.72±14.14	13.23±14.99	10.34±8.87	0.229
REM sleep latency	159.82±157.74	91.04±111. 86	109.62±66.13	0.78
REM percentage of sleep	7.10±7.99	69.23±124.18	54.54±72.43	0.001*
AHI	12.87±24.04	18.32±29.66	5.66±20.88	0.191
Mean heart rate	56.39±19.63	54.09± 19.80	64.03±14.96	0.071

N1 – non-rapid eye moment 1; N2 – non-rapid eye moment 2; N3 – non-rapid eye moment 3; REM – rapid eye moment; d-PD – depressed-Parkinson's disease; nd-PD – non-depressed Parkinson's disease; AHI – apnea hypopnea index. * Statistically significant (p<0.05).

Table 3. Means for F4 electrode amplitude in NREM2 in the various frequency bands between the subgroups with PD and the control group.

	EEG amplitude	d-PD	nd-PD	Control	P
F4	Delta	0.1782±0.0891	0.1710±0.0657	0.1423±0.0671	0.04*
	Theta	0.0848±0.0346	0.0730±0.0342	0.0566±0.0226	0.048*
	Alpha	0.0517±0.0329	0.0499±0.0206	0.0353±0.0140	0.14
	Beta	0.0333±0.0179	0.0346±0.0145	0.0309±0.0114	0.607

PD – Parkinson's disease; EEG – electroencephalogram; NREM2 – non-rapid eye moment 2; d-PD – depressed-Parkinson's disease; nd-PD – non-depressed Parkinson's disease; F4 – Frontal 4. * Statistically significant (p<0.05).

amplitude in the theta (P=0.048) ranges were progressively greater in the depressed PD patients than in the non-depressed PD group and control group, and the delta ranges in the depressed PD group were higher than in the controls (P=0.04), but no difference was found with the non-depressed group, and these results were statistically significant (ANOVA, p<0.05). The difference in amplitude between the PD subgroups and the controls can be seen in Figure 1. We also analyzed the REM data, but no significant differences were found between the PD subgroups and controls.

Table 4 shows the results for the analysis of difference in comparison of NREM2 and REM in the depressed PD group and non-depressed group. In the depressed PD group, the amplitude in F4 in the delta ranges (P=0.047) of NREM2 was greater than REM, and the amplitudes in C3 (P=0.033), C4 (P=0.045), O1 (P=0.044), and O2 (P=0.025) in the beta ranges of REM were greater than NREM2, but there were no significant differences

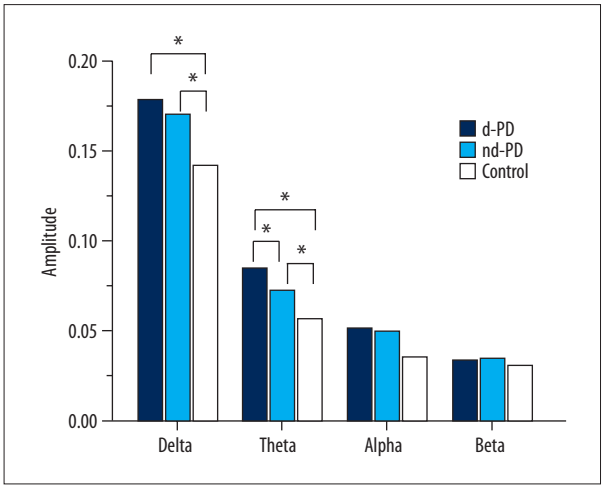

Figure 1. Comparison of NREM2 between the PD subgroups and the control group for the EEG amplitude of F4. * Statistical significance (p<0.05).

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Table 4. EEG amplitude in NREM2 and REM in the various frequency bands in subgroups with PD.

EEG amplitude	N2 sleep stage	REM sleep	P
d-PD F4(delta)	0.1782±0.0891	0.2989±0.2893	0.047*
nd-PD F4(delta)	0.1710±0.0657	0.1991±0.1381	0.611
d-PD C3(beta)	0.0311±0.0149	0.0521±0.0465	0.033*
nd-PD C3(beta)	0.0326±0.0138	0.0432±0.0325	0.41
d-PD C4(beta)	0.0334±0.0226	0.0537±0.0449	0.045*
nd-PD C4(beta)	0.0329±0.0139	0.0453±0.0375	0.394
d-PD O1(beta)	0.0323±0.0194	0.0531±0.0476	0.044*
nd-PD O1(beta)	0.0313±0.0126	0.0444±0.0322	0.301
d-PD O2(beta)	0.0299±0.0143	0.0505±0.0429	0.025*
nd-PD O2(beta)	0.0311±0.0131	0.0429±0.0323	0.353

PD – Parkinson's disease; EEG – electroencephalogram; NREM2 – non-rapid eye moment 2; REM – rapid eye moment; d-PD – depressed-Parkinson's disease; nd-PD – non-depressed Parkinson's disease; F4 – Frontal 4; C3 – Central 3; C4 – Central 4; O1 – Occipital 1; O2 – Occipital 2. * Statistically significant (p<0.05).

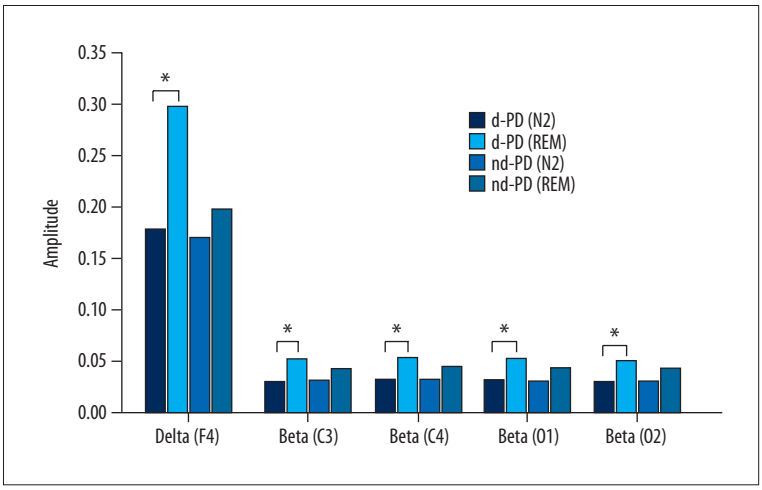

Figure 2. Comparison of NREM 2 and REM in the depressed PD group and non-depressed group for the EEG amplitude of F4, C3, C4, O1, and O2. * Statistical significance (p<0.05).

in the non-depressed PD group. The difference of amplitude in the depressed PD group between NREM2 and REM is shown in Figure 2.

Discussion

We compared quantitative EEG amplitudes during sleep between the PD patients and controls and performed a detailed analysis of different sleep stages in the depressed PD group and non-depressed PD group.

We found the depressed PD group had long wake-up time, which has been previously demonstrated. Approximately two-thirds of depressed patients have insomnia, and women are more likely to report insomnia than men [21], perhaps because they have too little deep sleep and more shallow sleep (Table 2). However, the depressed PD group did not have shorter rapid eye movement latency.

The comparison of quantitative sleep EEG amplitude data analysis between the PD with depression group versus the PD group without depression and the controls showed significant increases in theta amplitude ofNREM2 in right frontal areas, which is associated with emotional processing. Both the depressed PD group and the non-depressed PD group showed higher delta amplitudes than the control group. These findings are consistent with previous neuroimaging research results. These changes in neural structure and functional imaging have been demonstrated in patients with major depression [22–24]. The higher delta amplitude in the frontal area is explained by a previous study that revealed local cerebral blood flow in the orbitofrontal cortex was decreased in PD patients with depression [4], and in the same area, patients with PD with

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depression had lower metabolism than patients without depression [25,26]. The orbitofrontal cortex may play a key role in the etiology of PD with depression [27]. The degeneration of dopaminergic neurons causes orbitofrontal dysfunction, which disrupts serotonergic neurons in the dorsal raphe and leads to depression. In this present study, we only found increased right hemisphere theta amplitude in NREM2, suggesting that frontal theta asymmetry is a potential biomarker for depression. The evidence is primarily based on animal studies and emotion theory analysis [28,29]. The mean frontal theta power of the left hemisphere significantly increases in patients without depression when listening to music, but in depressed patients the frontal theta asymmetry was reversed during music listening, which has been previously demonstrated [30].

On the other hand, higher amplitudes in the beta ranges of REM than NREM2 were found in PD patients with depression, but there is scant data on this. The theoretical basis for this comparison is that the sleep cycle is formed by interactions between monoamine energy neurons involved in slow-wave sleep and cholinergic neurons involved in REM sleep, and maintains a dynamic balance. According to the cholinergic-adrenergic hypothesis of mania and depression, central cholinergic factors are essential to the pathogenesis of affective disorders. The cholinergic predominance relative to monoaminergic activity is important in explaining REM sleep disinhibition in depression [31]. Our data support the idea that the difference in sleep quantitative EEG amplitude involves REM. We found higher beta amplitudes in C3, C4, O1, and O2 regions in REM than in NREM2. Two previous studies shed light on this issue. One study discovered that a depressed PD group had higher perfusion in the occipital lobe region and cuneus compared to the non-depressed group [4]. Another study showed that glucose metabolism is increased in the cuneus lobe and surrounding regions, which have been found to be important in elderly depressed PD patients [32,33]. Increased cerebral blood flow and increased brain metabolism in these areas accelerate brainwave activity. The mechanism of hyper-infusion of cuneus in PD with depression remains unclear, but may be to the reduced γ-aminobutyric acid (GABA) levels [34], while GABAergic neurons are mostly distributed in the occipital cortex of depressed patients [35]. A study found that the relative powers of the beta bands were stronger in the central regions in patients with major depressive disorder [36], and the authors speculated the cause may be related to the higher number of beta segments. The depressed PD group had low levels of monoamine energy neurons, therefore increasingly the activity of REM sleep. However, because of the loss of dopamine neurons, the inhibitory effect on the release of GABA-derived transmitters is relieved, making GABA-energy neurons more active in REM.

The present study has certain limitations. There were not enough samples, and there was no complete exclusion of drug intervention. PD patients were taking their usual medication during the study, excluding antidepressants, antipsychotics, and sedatives. It remains unclear whether dopaminergic medication influences sleep mechanisms. Future studies with larger sample sizes should investigate whether these EEG changes in Parkinson's disease can predict depression in the long term. Also, future studies should integrate neural pathway and structural functional image to explore the pathogenesis of Parkinson's disease with depression.

Conclusions

In general, our research identified the different types of quantitative EEG during sleep in depressed and non-depressed PD patients. The higher amplitude in theta in frontal areas in NREM2 and the higher amplitude in beta in parietal and occipital lobe areas in REM relative to NREM2 are the characteristic changes of EEG during sleep in Parkinson's disease patients with depression. These findings provide a new direction for the diagnosis of Parkinson's disease with depression.

Conflict of interest

None.

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