Analysis of Quantitative Electroencephalogram Characteristics in Patients with Persistent Posture-Perceived Dizziness

Zhuowen Li, Dehao Zhao, Wenshi Wei

Department of Neurology,Huadong Hospital Affiliated to Fudan University,Shanghai,China

Introduction. To analyze the characteristics of quantitative electroencephalogram (QEEG) in patients with persistent posture-perceived dizziness, explore the pathophysiological mechanism of PPPD, and try to find quantitative electroencephalogram related parameters applied to PPPD patients.

Methods. Twenty-five patients with dizziness from June 2022 to December 2022 were selected, and healthy people who visited the physical examination center during the same period were selected as the control group. The EEG information of the two groups was collected, and the quantitative EEG indexes including the relative energy value of each frequency band and the ratio of fast to slow were obtained by fast Fourier transform.

Results. Compared with the control group, the PPPD group had higher relative energy values in F4, C4, P4α frequency band, lower relative energy values in C3, P3, T5α frequency band, lower relative energy values in C3, T3, T4α2 frequency band, higher relative energy values in F8β frequency band, higher relative energy values in FP2, F4, F8β1 frequency band, and higher energy values in P4, T5θ frequency band. The fast/slow ratio of C4 and P4 decreased.

Conclusions. Quantitative EEG analysis suggests the possible role of anxiety, excessive vigilance of vestibular and balance systems, and persistent high visual dependence in the pathogenesis of PPPD.

Keywords. Persistent posture-perceived dizziness;Quantitative electroencephalogram;Sleep disorders; Anxiety; Fatigue

INTRODUCTION

Persistent postural-perceptual dizziness (PPPD) is a common chronic condition that accounts for 10% of primary dizziness diagnoses (Guerra-Jiménez et al., 2017). It is frequently encountered in vertigo specialist clinics. The symptoms of PPPD include chronic dizziness, non-rotational dizziness, and a persistent sense of imbalance lasting more than three months. These symptoms can be worsened by factors like upright posture, movement, motion exposure, visual stimuli, or sleep deprivation (Das et al., 2023; Staab, 2020). PPPD patients often experience additional problems, including anxiety, depression, sleep disorders, and difficulties with gait(Passamonti et al., 2018; Seemungal and Passamonti, 2018; Sezier et al., 2019; Waterston et al., 2021) .After experiencing acute dizziness, individuals with PPPD become excessively vigilant towards strong dizziness or vestibular imbalances. This heightened vigilance leads to increased anxiety(Adamec et al., 2020). They also rely heavily on visual cues for balance and orientation, resulting in an excessive dependence on visual stimuli (Riccelli et al., 2017). Hyperawareness of somatosensory information related to posture further contributes to the persistent dizziness in PPPD patients (Cousins et al., 2017). As a result, individuals may adopt a cautious gait, experience muscle pain, and display anxiety and avoidance behaviors. Fatigue and difficulties with concentration have also been reported, likely due to the increased effort required to perform tasks that were once automatic and could be exacerbated by psychological factors (Stephan et al., 2016).

Although a complete understanding of the underlying pathophysiological mechanisms of PPPD remains elusive, it is believed that following the resolution of acute dizziness, the balance system relies more heavily on visual cues for self-balance and posture control. Consequently, individuals continue to rely on external objects or fix their gaze on specific points during movement, hindering the recalibration of the balance system(Indovina et al., 2015; Lee et al., 2018; Nigro et al., 2019; Sun and Xiang, 2020).

Treatment options for PPPD include pharmaceutical interventions, vestibular nerve rehabilitation therapy, psychotherapy, and cognitive behavioral therapy (Yu et al., 2018), all of which have shown efficacy. Early initiation of treatment has been linked to long-term effectiveness based on research on chronic subjective dizziness (Dieterich et al., 2016; Whitney et al., 2016) . The presence of persistent symptoms in undiagnosed or untreated individuals highlights the need for further investigations into the role of endogenous biomarkers in PPPD diagnosis and treatment.

Electroencephalogram (EEG) allows non-invasive observation of ongoing electrical brain activity, providing insights into overall cerebral cortex function (Fetz, 2007). Clinical evaluation of resting state EEG recordings typically involves visual analysis. Using fast Fourier transform, quantitative electroencephalogram analyzes EEG signals in the frequency and time domains, generating reliable results. Quantitative EEG indexes, such as the relative energy values of different frequency bands and the ratio of fast to slow activity, are derived from EEG signal acquisition. Alterations in EEG spectral values reflect changes in cortical activity and connectivity, potentially revealing pathophysiological developments in PPPD progression and shedding light on underlying mechanisms(Fetz, 2007).

This study aimed to collect quantitative EEG indexes, specifically relative power values and fast/slow ratios, from 16 brain regions (bilateral frontopolar, bilateral frontal, bilateral parietal, bilateral central, bilateral anterior temporal, bilateral middle temporal, bilateral posterior temporal, and bilateral occipital), across four frequency bands (α, β, θ, δ). By exploring the relationship between the relative energy values of each frequency band and the ratio of fast to slow activity in each brain region, we compared findings between the PPPD group and the healthy control group. The objective was to identify quantitative EEG indexes that are relevant to PPPD characteristics.

1.DATA AND METHODS

1.1Participants

Between June 2022 and December 2022, we enrolled 25 patients with PPPD, all of whom completed general information collection. PPPD patients included 11 males and 14 females. Vertigo was evaluated by DHI. In addition, in order to obtain more accurate diagnosis results, all patients were required to undergo at least 5 minutes of EEG examination in a awake, quiet state with eyes closed. Written informed consent was obtained from all enrolled patients. Inclusion criteria: Diagnosis was based on clinical criteria defined by the Barany Society. Exclusion criteria: 1) history of other central or peripheral vertigo diseases; 2) the presence of definite vestibular dysfunction; 3) mental illness or severe cognitive impairment, unable to cooperate with the EEG and scale assessment; 4) history of drug or alcohol dependence or abuse.

1.2Methods

1.2.1Clinical baseline information collection: All patients in the study completed the general information collection, including gender, age, education level, personality, disease course, hypertension, diabetes, heart disease, hyperlipidemia, smoking history, drinking history and medication status.

1.2.2 Assessment of vertigo in PPPD patients: Dizziness Handicap Inventory (DHI) was used to measure the severity of vertigo symptoms in patients(Zamyslowska-Szmytke et al., 2021); Patients were divided into three groups based on a cutoff of 30 and 60, with 30 to 60 classified as mild impairment, those with scores between 31 and 60 classified as moderate impairment, and those with scores between 61 and 100 classified as severe impairment.

1.2.3 EEG information acquisition: During the existence of symptoms of PPPD patients, EEG information was collected in the awake, quiet state with eyes closed.

The EEG electrodes were placed according to the international standard 10-20 lead system, with A1 and A2 as the reference electrodes. Electrodes were placed at FP1 and FP2 (left and right frontal pole), F3 and F4 (left frontal region, right frontal region), C3 and C4 (left central region, right central region), P3 and P4 (left parietal region, right parietal region), O1 and O2 (left occipital region, right occipital region), F7 and F8 (left anterior temporal region, right anterior temporal region), T3 and T4 (left middle temporal and right middle temporal), T5 and T6 (left posterior temporal and right posterior temporal). To obtain accurate EEG information, we need to perform an EEG examination for at least 5 minutes. Eeg information was collected by three experienced neuroelectrophysiologists. Three segments of each patient's electroencephalogram, each 10s without any artifacts, in the resting state with closed eyes, were intercepted, and the average was taken. The quantitative electroencephalogram indexes including the relative energy values of each frequency band (α, β, θ, δ) and the ratio of fast to slow (α+β) /(θ+δ) were obtained by fast Fourier transform.

1.3 Statistical Methods

SPSS25.0 software was used for statistical analysis. Categorical data were expressed by percentage and analyzed by X2 test. Mean ± standard deviation was used to describe the measurement data conforming to normal distribution, and median (interquartile range) was used to describe the data not conforming to normal distribution. The t test was used to compare the measurement data that conformed to the normal distribution between groups, and the rank sum test was used to compare the measurement data or categorical data that did not conform to the normal distribution between groups. In terms of $P < 0.05$ was considered statistically significant.

2.RESULTS

2.1 Basic information of the subjects

A total of 25 PPPD patients (11 males and 14 females) and 25 healthy controls were enrolled according to the PPPD diagnostic criteria. There was no significant difference in baseline information between the two groups. Clinical characteristics of PPPD patients are shown in table 1.

Table 1 Clinical characteristics of PPPD patients

Clinical characteristics	
course of disease（month）	64.47±89.54
character	21.05%Introverted
	52.63%moderately
	26.32%extroverted
degree of education(years)	12.84±3.58
hypertension（%）	47.37%
diabetes（%）	31.58%
cardiopathy（%）	15.79%
hyperlipemia（%）	47.37%
Smoking（%）	15.79%
Drinking（%）	15.79%
Assessment of symptoms	
DHI Score	43.37±23.63
DHI-P	16.11±6.72
DHI-E	10.42±8.91
DHI-F	16.95±10.63
HAMA	10.68±6.71
PSQI	8.74±5.19
FS-14	6.26±3.98

Clinical characteristics of PPPD patients see inTable..DHI is representative of Dizziness Handicap Inventory，DHI-P is representative of Physical indicator，DHI-E is representative of sentiment indicator,DHI-F is representative o functional indicator;;HAMA is representative of Hamilton Anxiety Scal;;PSQI is representative of Pitsburgh sleep quality indexs;FS- 14 is representative of fatigue severity scale.

2.2 QEEG characteristics of the subjects

(1) Analysis of the relative energy values of each frequency band in each brain region of PPPD patients

Comparison of QEEG between PPPD group and control group showed that: 1) Compared with the control group, the relative energy value of α band in the PPPD group increased in F4 (right frontal region), C4 (right central region), P4 (right parietal region), and decreased in C3 (left central region), P3 (left parietal region), T5 (left posterior temporal region). The relative energy value of α2 band decreased in C3 (left central region), T3 (left middle temporal) and T4 (right middle temporal), and the difference was statistically significant (P＜0.05);

2) Compared with the control group, the relative energy value of β band in F8 (right anterior temporal) increased in PPPD group, and the relative energy value of β1 band in FP2(right frontal pole), F4 (right frontal region) and F8 (right anterior temporal) increased, the difference was statistically significant (P＜0.05);

3) The θ energy values of P4 (right parietal region) and T5 (left posterior temporal region) increased, with statistically significant differences (P＜0.05)(See Table 2,3)

Table2 Quantitative EEG spectroscopic analysis of the left hemisphere in patients with PPPD

	PPPD group (n=25)	Control gruop (n=25)	r
FP1δ	19.37±11.50	14.67+7.71	0.148
FP1θ	16.01±6.26	14.55±6.80	0.495
FP1α	43.84±18.54	53.01±16.57	0.117
FP1α1	17.70（12.80，28.90）	25.90（13.50，51.10）	0.255
FP1α2	14.60（11.40，28.30）	21.00（11.80，29.30）	0.661
FP1β	16.80（14.50 ，25.80)	14.80(8.30,20.30)	0.170
FP1β1	10.20(7.50，12.50)	8.50（4.70，11.50）	0.249
FP1β2	6.70（5.30，10.80）	6.10（3.20，10.50）	0.255
F3 δ	18.10（9.00，21.20）	12.40（9.40，19.70）	0.365
F3 θ	15.22±5.20	13.53±5.90	0.353
F3 α	45.47±18.48	54.13±16.13	0.133
F3 α1	22.00（14.70，34.90）	24.80（14.40，48.40）	0.422
F3 α2	20.81±12.50	23.38±12.90	0.537
F3 β	20.10（15.90，26.80）	16.7（8.80，20.20）	0.165
F3 β1	10.6（8.30，13.60）	8.00（5.00，12.50）	0.237
F3 β2	8.50（5.40，15.0）	6.50（3.80，10.70）	0.204
C3 δ	11.90（8.70，17.00）	11.10（7.30，15.20）	0.389

C3 θ	14.10(8.30，20.50)	10.4(8.60，14.70)	0.174
C3 α	46.9（24.20，51.40）	58.3（40.40，67.40）	0.016*
C3 α1	21.78±11.87	28.34±18.67	0.205
C3 α2	18.73±11.30	26.47±14.19	0.071
C3β	26.00（20.70，29.80）	18.10（10.60，28.70）	0.148
C3β1	12.40（9.80，13.80）	8.3（5.30，15.30）	0.209
C3β2	12(8.60,16.40)	8.1(4.80,14.60)	0.075
P3 δ	13.00（8.70，18.50）	12.40（7.00，13.90）	0.157
P3 θ	12.40（9.40，20.50）	10.70（8.00，14.90）	0.099
P3 α	46.91±16.64	57.84±15.64	0.044*
P3 α1	22.94±11.77	26.83±18.74	0.449
P3 α2	23.97±13.54	31.01±17.42	0.173
P3 β	22.90（16.90，27.60）	14.00（10.90，25.20）	0.085
P3 β1	10.80（9.10，15.50）	7.70（6.60，15.60）	0.194
P3 β2	8.30（6.80，13.50）	6.30（4.30，9.60）	0.102
O1 δ	9.80（7.80，16.10）	9.10（5.40，15.40）	0.422
O1 θ	9.60（8.00，13.70）	9.00（7.60，13.00）	0.704
O1 α	53.80（41.90，67.40）	59.80（49.00，77.40）	0.220
O1 α1	17.10（11.50，32.30）	22.60（11.00，39.20）	0.651
O1 α2	29.00（16.30，48.50）	30.60（17.40，42.90）	0.781
O1 β	19.60（15.30，28.50）	13.20（10.00，26.50）	0.140
O1 β1	10.40（8.30，15.20）	8.00（6.20，15.10）	0.249
O1 β2	7.10（4.60，10.70）	5.60（3.30，10.10）	0.199
F7 δ	23.76±11.19	22.21±12.80	0.693
F7 θ	14.80（11.90，20.60）	12.70（8.40，16.00）	0.148
F7 α	35.14±13.95	45.80±19.44	0.060
F7 α1	11.40(9.90，24.70)	23.90（9.70，46.00）	0.194
F7 α2	15.80（11.50，25.10）	15.80（9.80，19.80）	0.781
F7 β	21.60（15.30，34.60）	16.50（9.30，24，30）	0.140
F7 β1	11.40(8.40，15.30)	8.80(4.80，11.80)	0.157
F7 β2	7.80（6.00，18.10）	7.20（4.20，12.80）	0.267
T3 δ	18.50（11.00，29.50）	18.00（7.00，34.70）	0.770
T3 θ	14.30（9.50，17.50）	11.80（7.40，15.70）	0.129
T3 α	31.37±16.25	38.50±17.57	0.203
T3 α1	15.10（8.00，25.20）	10.30（5.30，25.90）	0.965
T3 α2	14.20（10.00，17.90）	20.70（13.00，28.20）	0.034*
T3 β	31.10（18.40，35.10）	18.50（10.80，50.20）	0.148
T3 β1	13.30（10.20，16.70）	10.20（5.20，20.50）	0.397
T3 β2	14.40（6.80，23.50）	8.80(4.40，26.20)	0.274
T5 δ	17.98±7.40	16.05±10.96	0.527
T5 θ	13.90（11.50，17.60）	10.20（8.20，13.30）	0.013*
T5 α	40.22±14.47	51.71±17.82	0.036*
T5 α1	19.10（10.20，26.90）	19.70（11.80，30.00）	0.781
T5 α2	16.70（11.70，27.10）	24.20（16.80，33.30）	0.205

T5 β	24.90（16.90，29.10）	17.30（10.30，25.70）	0.099
T5 β1	13.40（10，50，16.50）	10.70（5.90，16.50）	0.243
T5 β2	8.90（5.20，23.90）	7.20（4.30，11.50）	0.165

Analysis of relative energy values in each frequency band with left hemisphere brain region of PPPD patients. * Represents a significant difference between groups（P＜0.05）. Values in the table are Mean (SD) for normally distributed variables;Median [Minimum, Maximum] for skewed variables.

Table3 Quantitative EEG spectroscopic analysis of the right hemisphere in patients with PPPD

	PPPD group (n=25)	Control gruop (n=25)	r
FP2δ	19.60（10.60，26.50）	14.00（10.70，19.20）	0.274
FP2 θ	12.80（11.00，17.30）	12.00（10.10，16.60）	0.599
FP2 α	56.48±17.92	45.30±19.71	0.076
FP2 α1	20.80（12.50，35.20）	27.80（12.60，58.00）	0.381
FP2 α2	20.50±10.28	24.05±14.24	0.384
FP2 β	15.00（6.40，17.50，）	18.10（13.00，24.30）	0.070
FP2 β1	11.30（7.30，12.80）	7.90（3.90，11.10）	0.032*
FP2 β2	7.00（4.20，9.70）	5.90（1.80，9.10）	0.267
F4 δ	16.93±8.96	12.27±7.63	0.093
F4 θ	13.00（11.70，18.80）	12.40（9.50，16.70）	0.328
F4 α	59.30±16.64	46.52±17.45	0.027*
F4 α1	22.80（13.00，38.40）	26.4（12.40，51.60）	0.511
F4 α2	20.43±8.73	27.09±15.21	0.109
F4 β	11.40（7.60，18.80）	18.30（13.90，24.40）	0.077
F4 β1	11.50（8.20，13.50）	6.80（4.60，10.80）	0.047*
F4 β2	6.80（3.70，9.90）	6.30（2.40，8.90）	0.286
C4 δ	16.10（8.80，19.50）	10.8（7.50，16.80）	0.093
C4 θ	12.80（9.20，16.30）	12.30（8.50，13.40）	0.118
C4 α	58.58±17.83	46.07±14.61	0.023*
C4 α1	22.4(12.50，36.00)	22.8（11.20，50.40）	0.804
C4 α2	20.55±7.61	28.69±15.75	0.053
C4 β	17.87±11.34	22.38±9.10	0.185
C4 β1	12.60（11.60，15.60）	6.70（5.00，14.90）	0.804
C4 β2	7.80（5.60，10.80）	7.3（3.50，9.50）	0.422
P4 δ	14.86±7.86	12.13±7.51	0.280
P4 θ	12.50（10.00，16.50）	10.10（8.00，12.50）	0.017*
P4 α	66.50（44.10，71.10）	46.50（37.60，59.30）	0.021*
P4 α1	21.60（11.30，34.60）	25.70（10.90，51.10）	0.521
P4 α2	23.54±12.04	30.56±16.91	0.149
P4 β	14.30（8.90，24.90）	21.30（16.90，25.50）	0.157
P4 β1	12.30（10.20，15.70）	7.40（5.90，13.90）	0.133
P4 β2	7.40（5.10，9.80）	6.50（3.40，10.20）	0.405
O2 δ	14.37±10.12	12.33±7.95	0.493
O2 θ	9.60（7.30，15.00）	11.00（8.60，13.70）	0.704

O2 α	67.90（42.60，76.00）	54.00（37.10，68.70）	0.343
O2 α1	13.30（9.30，31.10）	21.60（13.40，39.00）	0.249
O2 α2	22.30（13.50，52.30）	14.60（19.50，48.70）	0.493
O2 β	11.70（9.20，22.80）	16.40（11.10，27.50）	0.293
O2 β1	11.90（5.60，14.20）	6.70（5.30，12.90）	0.422
O2 β2	5.00（3.30，10.90）	4.50（3.30，8.40）	0.549
F8 δ	17.70（9.70，31.0）	17.0（10.70，28.40）	0.872
F8 θ	11.10（9.20，16.40）	12.2（10.20，13.10）	0.759
F8 α	49.78±16.95	40.18±17.08	0.091
F8 α1	17.00（7.80，25.50）	23.50（11.80，45.30）	0.220
F8 α2	20.28±9.74	22.22±13.81	0.621
F8 β	13.40（8.50，20.50）	22.20（14.90，30.80）	0.040*
F8 β1	10.90（8.60，16.00）	8.00（4.80，11.20）	0.031*
F8 β2	9.70（5.10，14.30）	5.70（3.70，8.00）	0.054
T4 δ	15.30（12.40，31.90）	17.20（12.00，27.70）	0.827
T4 θ	12.40（9.90，18.10）	11.80（9.70，13.20）	0.530
T4 α	41.62±15.49	31.58±15.06	0.078
T4 α1	10.00（6.30，26.50）	11.30（7.80，26.50）	0.726
T4 α2	15.71±5.42	22.92±10.18	0.011*
T4 β	25.44±14.06	29.83±14.82	0.355
T4 β1	14.95±6.36	13.25±6.31	0.414
T4 β2	16.10（6.40，22.40）	9.20（5.60，16.20）	0.286
T6 δ	14.80（10.00，19.30）	18.70（10.80，23.40）	0.267
T6 θ	13.34±5.12	11.55±3.18	0.205
T6 α	51.28±16.76	44.41±15.36	0.196
T6 α1	16.80（10.20，31.20）	20.00（8.90，30.60）	0.930
T6 α2	23.01±10.07	29.18±14.16	0.130
T6 β	20.13±10.42	25.86±11.99	0.124
T6 β1	15.14±6.80	11.55±5.98	0.093
T6 β2	9.00（6.00，14.30）	8.30（4.20，11.50）	0.280

Analysis of relative energy values in each frequency band with left hemisphere brain region of PPPD patients.. * Represents a significant difference between groups（P＜0.05）. Values in the table are Mean (SD) for normally distributed variables;Median [Minimum, Maximum] for skewed variables.

(2) the fast/slow ratio of different brain regions on QEEG in PPPD patients

Compared with the control group, the fast/slow ratio in C4 (right central area) and P4 (right occipital area) in the PPPD group decreased, and the difference was statistically significant (P＜ 0.05) (see Table 4)

Table 4 Analysis of rapid-slow ratio of quantitative EEG in patients with PPPD

	PPPD group （n=25）	Control group (n=25)	r
FP1 ratio	1.67（1.30，3.95）	3.17（1.62，4.11）	0.215
FP2 ratio	2.58±1.95	3.28±2.22	0.308
F3 ratio	2.40（1.43，3.43）	2.78（1.78，4.37）	0.397
F4 ratio	2.59±1.60	3.77±2.25	0.070
C3 ratio	2.92（1.88，3.48）	3.74（2.50，4.43）	0.165
C4 ratio	2.35（2.00，3.20）	3.63（2.38，5.02）	0.020*
P3 ratio	2.95±1.61	4.11±2.13	0.067
P4 ratio	2.95（1.74，3.50）	3.74（2.55，6.75）	0.042*
O1 ratio	4.11±2.33	4.86±2.70	0.365
O2 ratio	3.09（1.88，6.40）	3.95（2.03，5.81）	0.648
F7 ratio	1.37(1.17，2.77)	2.21（1.25，3.39）	0.237
F8 ratio	2.44±1.42	2.45±1.49	0.986
T3 ratio	1.75（1.15，3.61）	2.48（1.07，4.31）	0.907
T4 ratio	2.05（1.16，3.31）	2.33（1.24，3.44）	0.483
T5 ratio	1.99(1.70,2.80)	2.76(1.93,5.80)	0.102
T6 ratio	2.85±1.93	3.00±1.79	0.754

The fast/slow ratio of different brain regions on QEEG in PPPD patients,compared with the control group, the fast/slow ratio in C4 (right central area) and P4 (right occipital area) in the PPPD group decreased Frequency band speed ratio:(α+β/δ+θ) . * represents a significant difference between groups （P＜0.05）. Values in the table are Mean (SD) for normally distributed variables; Median [Minimum, Maximum] for skewed variables

3 DISCUSSION

3.1 Quantitative EEG characteristics of PPPD patients

1) Clinical characteristics of PPDD patients

Our study found that 57.9% of PPPD patients were female. Additionally, 26.3% of the patients exhibited extroverted traits, while 42.1% presented with anxiety, and 36.8% experienced sleep disorders. These results are consistent with previous research (Chiarella et al., 2016). Importantly, our findings suggest that being female, introverted, and having anxiety and sleep disorders increase the likelihood of chronic

or recurrent symptoms following acute dizziness episodes, indicating the presence of chronic vestibular nerve disorders. Identifying and promptly treating these patients is crucial to significantly improve their quality of life.

2) QEEG band energy characteristics of PPPD patients

Most patients with PPPD experience sleep disorders, with anxiety being the prevalent emotional disorder. Studies indicate that patients with anxiety demonstrate a decrease in α wave activity, an increase in α wave frequency, and an increase in β wave activity (Forner-Phillips et al., 2020). Asymmetry between the left and right brain hemispheres is observed, with the left hemisphere primarily involved in logical thinking, memory, reasoning, and other cognitive processes, while the right hemisphere is more engaged in visual and spatial processing, as well as emotional expression. The involvement of the temporoparietal and inferior frontal cortex regions in vigilance and autonomic arousal suggests their significant role in regulating negative emotions such as depression and anxiety, thereby influencing behavior and responses (Cutrer and Baloh, 1992; Lee et al., 2018). Multiple studies have demonstrated asymmetry in frontal EEG α waves (Jacob et al., 2009; Li et al., 2020). Notably, Davidson et al. found that increased activity in the right frontal lobe is associated with anxiety(Wurthmann et al., 2017) , and Kropotov et al. in 2009, as well as KroFurthermore and Moscovitch et al. in 2011, observed α-wave asymmetry in the prefrontal lobe of patients with anxiety disorder, characterized by lower α-wave activity in the left hemisphere compared to the right hemisphere(Na et al., 2019; Schneider et al., 2018). Caviness et al. reported an increased relative energy value in the β band for patients with mood disorders(Fetz, 2007) .

The beta wave, the fastest brain wave observed in the electroencephalogram (EEG), indicates an aroused cortical state associated with mental stress and emotional excitement (Balaban, 2011). An increase in beta waves reflects heightened alertness and readiness to respond to changes in the external environment. It signifies a highly focused and tense state of attention in the left hemisphere and panic in the right hemisphere(Strube et al., 2021). Sleep disorders are commonly observed in most patients with PPPD, and prior EEG studies have demonstrated a significant and robust

increase in β band power during both wakefulness and sleep, extending to adjacent frequency bands, indicating persistent cortical hyperarousal (Del Campo-Vera et al., 2020).

Theta waves are more prevalent in the parietal and frontotemporal brain regions during fatigue states(McLoughlin et al., 2022) (Dias et al., 2022). Increased theta activity has been associated with daytime sleepiness and the use of hypnotics. A 2013 study demonstrated the generation of theta waves during emotion regulation (Dias et al., 2022).

This study aimed to compare the relative energy values of different frequency bands in PPPD patients and a healthy control group. The results revealed increased relative energy values in the α band of the right brain region, the β band of the left cerebral hemisphere, and the θ band in PPPD patients, except for O2 and F8. These changes may indicate alterations in functional connectivity within the brains of PPPD patients.

Further analysis suggests that these observed changes may be associated with anxiety, excessive vigilance in the vestibular and balance systems, and heightened visual dependence. However, additional studies are needed to comprehensively understand these phenomena, specifically investigating changes in α and β waves in the right and left hemispheres. These studies can illuminate potential discrepancies in activation levels across brain regions and in different disease states.

3)The characteristics of QEEG energy values in each brain region of PPPD patients

The prefrontal lobe and hippocampus play critical roles in regulating the stress response (Liu, 2020; Zhang, 2013). Disruption in this neural circuitry can lead to mental disorders like anxiety disorders and fear (Li et al., 2022). This study compared the relative energy values of PPPD patients to healthy controls. The results showed increased energy values in the α band in the right frontal (F4), right central (C4), and right parietal (P4) regions. Additionally, increased energy values were observed in the β1 band in the right frontal pole (FP2), right frontal (F4), and right anterior temporal (F8) regions, located in or near the frontal and temporal regions. Dysfunction in the frontal lobe can cause personality changes, emotional changes (e.g., depression and

anxiety), memory loss, and attention disorders(Zhang, 2005). Activation of the temporal lobe indicates excessive activation of the amygdala, responsible for emotion processing. Reduced alpha and theta activity in the frontal lobe is associated with impaired alertness. High anxiety is linked to heightened alertness, and during withdrawal emotions (e.g., fear and disgust), the right frontal and anterior temporal lobes are selectively activated. Therefore, individuals with anxiety are expected to exhibit increased activity in the right frontotemporal lobe (Coburn et al., 2006; Davidson, 1992). Studies have also shown increased activity in the right parietal lobe in individuals with anxiety(Coburn et al., 2006; Engels et al., 2007). In addition, increased arousal in individuals with depression and anxiety can explain the heightened activity in the right parietal lobe even during rest (Kesebir and Yosmaoğlu, 2018).

The prefrontal lobe and hippocampus play critical roles in regulating the stress response(Liu, 2020; Staab et al., 2014). Disruption in this neural circuitry can lead to mental disorders like anxiety disorders and fear(Li et al., 2022). This study compared the relative energy values of PPPD patients to healthy controls. The results showed increased energy values in the α band in the right frontal (F4), right central (C4), and right parietal (P4) regions. Additionally, increased energy values were observed in the β1 band in the right frontal pole (FP2), right frontal (F4), and right anterior temporal (F8) regions, located in or near the frontal and temporal regions. Dysfunction in the frontal lobe can cause personality changes, emotional changes (e.g., depression and anxiety), memory loss, and attention disorders(Zhang, 2005). Activation of the temporal lobe indicates excessive activation of the amygdala, responsible for emotion processing. Reduced alpha and theta activity in the frontal lobe is associated with impaired alertness. High anxiety is linked to heightened alertness, and during withdrawal emotions (e.g., fear and disgust), the right frontal and anterior temporal lobes are selectively activated. Therefore, individuals with anxiety are expected to exhibit increased activity in the right frontotemporal lobe(Davidson, 1992). Studies have also shown increased activity in the right parietal lobe in individuals with anxiety(Coburn et al., 2006; Engels et al., 2007). In addition, increased arousal in

individuals with depression and anxiety can explain the heightened activity in the right parietal lobe even during rest(Kesebir and Yosmaoğlu, 2018).

3.2 Summary and Limitations

In this study, quantitative electroencephalogram (QEEG) was used to analyze the changes of relative energy value and fast/slow ratio of each frequency band in different brain regions of PPPD patients, to explore the pathophysiological mechanism of PPPD, and to find quantitative electroencephalography-related biomarkers for PPPD patients. At the same time, the clinical characteristics of PPPD patients were analyzed by relevant symptom scales to comprehensively evaluate the clinical symptoms of PPPD patients. However, this study has the following shortcomings: 1. The sample size of this study is small, and subsequent studies need to expand the sample size to minimize errors. By grouping PPPD patients according to the severity of vertigo and regularly performing EEG examination, EEG changes in different disease states can be accurately evaluated, so as to better evaluate the therapeutic effect. (2) Quantitative EEG parameters selected in this study are few, and other relevant parameters can be combined to provide more complete data for evaluation in the future.

CONCLUSION

(1) Analysis of the clinical characteristics of PPPD patients found that PPPD patients were mostly female and introverted,majority patients had anxiety, sleep disorders, and varying degrees of fatigue.

(2)Quantitative EEG analysis suggested the possible role of anxiety, hypervigilance of vestibular and balance systems, and persistent high visual dependence in the disease mechanism of PPPD.

(3)Quantitative electroencephalogram (QEEG) was used to evaluate the electrophysiological characteristics of PPPD patients from multiple perspectives such as frequency, time and space.Therefore, this study provides new ideas for the application of biomarkers in PPPD patients.

DECLARATIONS

Ethical Approval

The studies involving human participants were approved by the Ethical Committee of Huadong Hospital of Fudan University. The patients/participants provided their written informed consent to participate in this study. Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

CONSENT TO PUBLICATION

All authors approved the publication.

DATA AVAILABILITY STATEMENT

Not applicable

CONFLICT OF INTEREST

The authors declare no competing interests.

FUNDING

This work was funded by Shanghai Municipal Key Clinical Specialty, grant number shslczdzk02801, the Shanghai Municipal Health Commission, grant number 2020YJZX0109.

ACKNOWLEDGMENTS

The author wishes to thank all the participants in this study.

AUTHOR CONTRIBUTION

ZL and DZ designed the study.ZL performed the research,analyzed the data and wrote the mazuscript.LZ,DZ and WW contributed to refining the ideas and carrying out additional analyses.All authors contributed to the article andapproved the submitted version.

REFERENCES

Adamec, I., Juren Measki, S., Krbot Skoric, M., Jazic, K., Crnosija, L., Milivojevic, I., and Habek, M. (2020). Persistent postural-perceptual dizziness: Clinical and neurophysiological study. J Clin Neurosci *72*, 26-30.

Balaban, C.D. (2011). Migraine, vertigo and migrainous vertigo: Links between vestibular and pain mechanisms. J Vestib Res *21*, 315-321.

Chiarella, G., Petrolo, C., Riccelli, R., Giofrè, L., Olivadese, G., Gioacchini, F.M., Scarpa, A., Cassandro, E., and Passamonti, L. (2016). Chronic subjective dizziness: Analysis of underlying personality factors. J Vestib Res *26*, 403-408.

Coburn, K.L., Lauterbach, E.C., Boutros, N.N., Black, K.J., Arciniegas, D.B., and Coffey, C.E. (2006). The value of quantitative electroencephalography in clinical psychiatry: a report by the Committee on

Research of the American Neuropsychiatric Association. J Neuropsychiatry Clin Neurosci *18*, 460-500.
Cousins, S., Kaski, D., Cutfield, N., Arshad, Q., Ahmad, H., Gresty, M.A., Seemungal, B.M., Golding, J., and Bronstein, A.M. (2017). Predictors of clinical recovery from vestibular neuritis: a prospective study. Ann Clin Transl Neurol *4*, 340-346.
Cutrer, F.M., and Baloh, R.W. (1992). Migraine-associated dizziness. Headache *32*, 300-304.
Das, S., Annam, C.S., Bakshi, S.S., and Seepana, R. (2023). Persistent positional perceptual dizziness in clinical practice: a scoping review. Neurol Sci *44*, 129-135.
Davidson, R.J. (1992). Anterior cerebral asymmetry and the nature of emotion. Brain Cogn *20*, 125-151.
Del Campo-Vera, R.M., Gogia, A.S., Chen, K.H., Sebastian, R., Kramer, D.R., Lee, M.B., Peng, T., Tafreshi, A., Barbaro, M.F., Liu, C.Y.*, et al.* (2020). Beta-band power modulation in the human hippocampus during a reaching task. J Neural Eng *17*, 036022.
Dias, C., Costa, D., Sousa, T., Castelhano, J., Figueiredo, V., Pereira, A.C., and Castelo-Branco, M. (2022). A neuronal theta band signature of error monitoring during integration of facial expression cues. PeerJ *10*, e12627.
Dieterich, M., Staab, J.P., and Brandt, T. (2016). Functional (psychogenic) dizziness. Handb Clin Neurol *139*, 447-468.
Engels, A.S., Heller, W., Mohanty, A., Herrington, J.D., Banich, M.T., Webb, A.G., and Miller, G.A. (2007). Specificity of regional brain activity in anxiety types during emotion processing. Psychophysiology *44*, 352-363.
Fetz, E.E. (2007). Volitional control of neural activity: implications for brain-computer interfaces. J Physiol *579*, 571-579.
Forner-Phillips, N.A., Mills, C., and Ross, R.S. (2020). Tendency to ruminate and anxiety are associated with altered alpha and beta oscillatory power dynamics during memory for contextual details. Cogn Affect Behav Neurosci *20*, 698-716.
Guerra-Jiménez, G., Arenas Rodríguez, A., Falcón González, J.C., Pérez Plasencia, D., and Ramos Macías, Á. (2017). Epidemiology of vestibular disorders in the otoneurology unit. Acta Otorrinolaringol Esp (Engl Ed) *68*, 317-322.
Indovina, I., Riccelli, R., Chiarella, G., Petrolo, C., Augimeri, A., Giofrè, L., Lacquaniti, F., Staab, J.P., and Passamonti, L. (2015). Role of the Insula and Vestibular System in Patients with Chronic Subjective Dizziness: An fMRI Study Using Sound-Evoked Vestibular Stimulation. Front Behav Neurosci *9*, 334.
Jacob, R.G., Redfern, M.S., and Furman, J.M. (2009). Space and motion discomfort and abnormal balance control in patients with anxiety disorders. J Neurol Neurosurg Psychiatry *80*, 74-78.
Kesebir, S., and Yosmaoğlu, A. (2018). QEEG in affective disorder: about to be a biomarker, endophenotype and predictor of treatment response. Heliyon *4*, e00741.
Lee, J.O., Lee, E.S., Kim, J.S., Lee, Y.B., Jeong, Y., Choi, B.S., Kim, J.H., and Staab, J.P. (2018). Altered brain function in persistent postural perceptual dizziness: A study on resting state functional connectivity. Hum Brain Mapp *39*, 3340-3353.
Li, K., Si, L., Cui, B., Ling, X., Shen, B., and Yang, X. (2020). Altered spontaneous functional activity of the right precuneus and cuneus in patients with persistent postural-perceptual dizziness. Brain Imaging Behav *14*, 2176-2186.
Li, N., Ren, W., and Li, Y. (2022). Research progress control anxiety behavior of the neural circuits. Anatomical study *44*, 492-496.

Liu, W. (2020). Neural circuitry from prefrontal cortex to amygdala mediates chronic stress-induced anxiety-like behavior in mice (Nanchang University).

McLoughlin, G., Gyurkovics, M., Palmer, J., and Makeig, S. (2022). Midfrontal Theta Activity in Psychiatric Illness: An Index of Cognitive Vulnerabilities Across Disorders. Biol Psychiatry *91*, 173-182.

Na, S., Im, J.J., Jeong, H., Lee, E.S., Lee, T.K., Chung, Y.A., and Song, I.U. (2019). Cerebral perfusion abnormalities in patients with persistent postural-perceptual dizziness (PPPD): a SPECT study. J Neural Transm (Vienna) *126*, 123-129.

Nigro, S., Indovina, I., Riccelli, R., Chiarella, G., Petrolo, C., Lacquaniti, F., Staab, J.P., and Passamonti, L. (2019). Reduced cortical folding in multi-modal vestibular regions in persistent postural perceptual dizziness. Brain Imaging Behav *13*, 798-809.

Passamonti, L., Riccelli, R., Lacquaniti, F., Staab, J.P., and Indovina, I. (2018). Brain responses to virtual reality visual motion stimulation are affected by neurotic personality traits in patients with persistent postural-perceptual dizziness. J Vestib Res *28*, 369-378.

Riccelli, R., Indovina, I., Staab, J.P., Nigro, S., Augimeri, A., Lacquaniti, F., and Passamonti, L. (2017). Neuroticism modulates brain visuo-vestibular and anxiety systems during a virtual rollercoaster task. Hum Brain Mapp *38*, 715-726.

Schneider, T.R., Hipp, J.F., Domnick, C., Carl, C., Büchel, C., and Engel, A.K. (2018). Modulation of neuronal oscillatory activity in the beta- and gamma-band is associated with current individual anxiety levels. Neuroimage *178*, 423-434.

Seemungal, B.M., and Passamonti, L. (2018). Persistent postural-perceptual dizziness: a useful new syndrome. Pract Neurol *18*, 3-4.

Sezier, A.E.I., Saywell, N., Terry, G., Taylor, D., and Kayes, N. (2019). Working-age adults' perspectives on living with persistent postural-perceptual dizziness: a qualitative exploratory study. BMJ Open *9*, e024326.

Staab, J.P. (2020). Persistent Postural-Perceptual Dizziness. Semin Neurol *40*, 130-137.

Staab, J.P., Rohe, D.E., Eggers, S.D., and Shepard, N.T. (2014). Anxious, introverted personality traits in patients with chronic subjective dizziness. J Psychosom Res *76*, 80-83.

Stephan, K.E., Manjaly, Z.M., Mathys, C.D., Weber, L.A., Paliwal, S., Gard, T., Tittgemeyer, M., Fleming, S.M., Haker, H., Seth, A.K., *et al.* (2016). Allostatic Self-efficacy: A Metacognitive Theory of Dyshomeostasis-Induced Fatigue and Depression. Front Hum Neurosci *10*, 550.

Strube, A., Rose, M., Fazeli, S., and Büchel, C. (2021). Alpha-to-beta- and gamma-band activity reflect predictive coding in affective visual processing. Sci Rep *11*, 23492.

Sun, L., and Xiang, K. (2020). A review on the alterations in the brain of persistent postural-perceptual dizziness patients and non-pharmacological interventions for its management. Rev Neurosci *31*, 675-680.

Waterston, J., Chen, L., Mahony, K., Gencarelli, J., and Stuart, G. (2021). Persistent Postural-Perceptual Dizziness: Precipitating Conditions, Co-morbidities and Treatment With Cognitive Behavioral Therapy. Front Neurol *12*, 795516.

Whitney, S.L., Alghwiri, A.A., and Alghadir, A. (2016). An overview of vestibular rehabilitation. Handb Clin Neurol *137*, 187-205.

Wurthmann, S., Naegel, S., Schulte Steinberg, B., Theysohn, N., Diener, H.C., Kleinschnitz, C., Obermann, M., and Holle, D. (2017). Cerebral gray matter changes in persistent postural perceptual dizziness. J Psychosom Res *103*, 95-101.

Yu, Y.C., Xue, H., Zhang, Y.X., and Zhou, J. (2018). Cognitive Behavior Therapy as Augmentation for Sertraline in Treating Patients with Persistent Postural-Perceptual Dizziness. Biomed Res Int *2018*, 8518631.

Zamyslowska-Szmytke, E., Politanski, P., and Jozefowicz-Korczynska, M. (2021). Dizziness Handicap Inventory in Clinical Evaluation of Dizzy Patients. Int J Environ Res Public Health *18*.

Zhang, L. (2005). The cognitive dysfunction and depression frontal lobe function defect. Abstract Chinese medicine internal medicine. Chinese medicine internal medicine, 660-663.

Zhang, Y. (2013). Not in the treatment of generalized anxiety disorder and panic disorder in patients with brain fear loop research (Central South University).

Corresponding Author:
Wenshi Wei
Department of Neurology, Huadong Hospital Affiliated to Fudan University, Shanghai, China
E-mail: 15221427798@163.com