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Borderline Personality and Bipolar Disorders Cannot Be Differentiated Electrophysiologically

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Abstract

Objectives. Certain studies have claimed that borderline personality disorder (BPD) could be evaluated as a subtype of bipolar disorder (BD), whereas others have argued that BPD should be regarded as an independent disorder because of its distinct clinical features. The aim of this study was to investigate if there was a difference between these 2 disorders biologically based on EEG recordings. *Methods*. A total of 111 subjects (11 healthy, 25 BPD, 75 BD) who had resting EEG recordings were included. The EEGs were analyzed to compute absolute power values. *Results*. One-way analysis of variance results revealed statistically significant differences among the 3 groups on 55 out of 229 EEG variables. However, post hoc analysis indicated that all of the significant changes were between healthy and patient groups and no significant differences were found between 2 clinical groups. *Conclusion*. The findings suggested that these 2 clinical entities are biologically similar; however, further research should be performed to explain the basis clinical differences between the 2 disorders.

Keywords

bipolar disorder, borderline personality disorder, diagnosis, EEG

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Background

Many studies have focused on the debate whether borderline personality disorder (BPD) should be conceptualized as a part of bipolar disorder (BD). The issue remains a controversy. Some studies have claimed that BPD could be accepted as a subtype of BD as the diagnostic criteria of the 2 disorders are similar,[1-4] whereas have others suggested that BPD should be evaluated as an independent disorder from BD[5-9] because of certain clinical features that distinguish 2 disorders.[10]

The controversy regarding the clinical features remains unresolved. However, another method to explore if the 2 disorders represent the same entity is to compare neuroimaging findings. Sripada and Silk,[11] in a review of functional neuroimaging studies, pointed out to both similarities and differences in brain areas affected in these 2 disorders. Structural neuroimaging studies report a decrease in volume of the prefrontal cortex in both disorders.[12] Nonetheless, nonoverlapping alterations have also been identified. Patients with BD have increased or normal volumes of amygdala and hippocampus, whereas patients with BPD demonstrate a decrease in volume of these brain regions.[13]

In positron emission tomography (PET) studies, researchers reported that BPD was mostly associated with altered frontal lobe metabolism.[11] Soloff et al[14] identified via PET that BPD patients displayed diminished response to serotonergic stimulation in the prefrontal cortex. In a later study, Soloff et al[15] reported that frontal hypometabolism in BPD is mainly related to increased impulsivity. According to the study results, impulsive behavior in BPD might be related to diminished glucose absorption in medial orbital frontal cortex. In another PET study, Boen et al[16] demonstrated that both BD and BPD disorders are associated with altered metabolism in insula. However, BPD and BD groups mainly differ in terms of metabolic activity in hypothalamus, midbrain striatum and cerebellum.

EEG studies on BPD also revealed controversial results. For instance, Ogiso et al[17] did not report any EEG abnormalities specific to BPD. Another study reported a greater incidence of epileptiform EEG abnormalities in borderline patients as compared with depressed individuals.[18] Berchio et al[19] conducted a study to determine alterations of face and gaze perception in

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BPD. The main result of this study was that women with BPD displayed alterations in perception of neutral gaze. According to EEG results, anterior cingulate cortex and prefrontal regions were aberrantly activated in BPD group during the face encoding process in comparison with the control group. In another study, Flasbeck et al[20] reported no significant EEG alterations that in BPD patients; however, right frontal EEG asymmetry correlated with alexithymia scores. Beeney et al[21] measured EEG asymmetry of patients with BPD and major depressive disorder after a social rejection challenge. According to the results, BPD patients had a higher left cortical activation, while patients with major depressive disorder exhibited higher right cortical activation. Another study investigated the differences of EEG-vigilance in BPD, obsessive-compulsive disorder and healthy controls.[22] According to results, BPD patients exhibited significantly lower EEG vigilance than the other groups.

Numerous studies reveal that there are prominent EEG alterations in BD. A study demonstrated that patients with BD and attention deficit/hyperactivity disorder (ADHD) had higher absolute theta power as compared with the control group during rest.[23] Another main characteristic of BD is likely to be the dysfunction of the brain connectivity. This indicates an alteration in synchronization of brain networks.[24] The decreased synchronization for BD patients in the alpha band was most prominent in the frontocentral and centroparietal connections. In addition, a decrease in alpha and an increase in beta band were also reported for BD.[25]

In light of the neuroimaging findings, one might argue that although clinical similarities exist between the 2 disorders, there are certain biological differences. One shortcoming of the previous neuroimaging studies is that they rarely compared BD and BPD groups directly with each other. In light of the relevant literature, the current study was designed to compare EEG findings in these 2 disorders. The main goal was to assess whether these 2 disorders are biologically identical or not. To the best of our knowledge, there have been no studies that compared EEG findings in these 2 disorders directly.

Methods and Materials

Participants

This retrospective study included 111 subjects (11 healthy, 25 BPD, 75 BD) who had EEG recordings and were recruited for electrophysiological analysis. All the subjects were recruited from the outpatient clinic database (see Table 1 for demographic information).

All patients were medication free for at least 1 month at the time of quantitative EEG (qEEG) acquisition. The diagnosis was made based on *DSM-5* (*Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition*) criteria. Patients with comorbid organic epilepsy, organic mental disorders, history of head injury and mental retardation, neurological diseases, or any other medical illnesses were excluded from the study.

qEEG Variables. All qEEG data were recorded in a quiet, dimly lit room. Patients sat calmly with eyes closed during the recording time of 7 minutes. In total, 19 electrodes were placed on the scalp, based on the international 10-20 system. Linked mastoid electrodes (A1-A2) were used for reference during acquisition. The data-sampling rate was 500 Hz and the acquired signals were band-pass filtered at 0.15 to 70 Hz and notch filtered at 50 Hz. Data artifacts were eliminated manually off-line for each patient. Data were averaged across the recording epochs (2 seconds) for each electrode, and the absolute power (percentage of total power) was computed for the following bands: delta (1-4 Hz), theta (4-7 Hz), alpha (8-12 Hz), alpha1 (8-10 Hz), alpha2 (10-12 Hz), beta (12-25 Hz), beta1 (12-15 Hz), beta2 (15-18 Hz), beta3 (18-25 Hz), high beta (25-30 Hz), gamma (30-50 Hz), gamma1 (30-35 Hz), gamma2 (35-40 Hz), high gamma (40-50 Hz). Neuroguide Deluxe v.2.5.1 (Applied Neuroscience, Largo, and FL) software was used for qEEG analysis.

Table 1. Demographic Information of the Participants.

(a): Gender Characteristics of the Study Population: Number

	Male	Female	Total
Borderline	7	18	25
Bipolar	33	42	75
Healthy	5	6	11
Total	45	66	111

(b) Mean and Standard Deviation of Age in the Study Population

	Male	Female	Total
Mean age			
Borderline	30.57	24.72	26.36
Bipolar	34.06	33.17	33.56
Healthy	33.80	40.67	37.55
Total	33.49	31.55	32.33
Standard deviation of age			
Borderline	9.14	5.11	6.82
Bipolar	11.12	9.23	10.05
Healthy	15.25	12.58	13.60
Total	11.14	9.75	10.33

Statistical Analysis

Statistical analysis was conducted by using SPSS version 24. Age and sex characteristics were analyzed with 2-way analysis of variance (ANOVA). Since the qEEG data were highly skewed, natural log-transformation was applied. One-way ANOVA was run with significance threshold set to .05. After finding the difference in variances among three groups, Tamhane's T2 was used for post hoc analysis.

Results

Results revealed that there was no significant difference among the groups regarding age and sex characteristics. 55 of 229 qEEG variables exhibited significant difference among the between group differences. Results showed that there was a

significant difference among the 3 groups for delta frequency in FP1, F3, F4, Fz, C3, Cz, P3, P4, Pz, O1, O2, F7, F8, T3, and T4 channels. Besides, in theta frequency a significant difference was observed between the 3 groups in FP1, P3, P4, Pz, O1, F7, T3, and T4 channels. In addition, there were significant group differences in following beta bands-channel pairs: FP1 Beta, FP2 Beta, C4 Beta, P3 Beta, P3 Beta 3, O1 Beta, O1 Beta 1, O1 Beta 2, O1 Beta 3, F7 Beta, F7 Beta 1, F7 Beta 2, F7 Beta 3, Pz High Beta, O1 High Beta, F7 High Beta, C3 High Beta. For Gamma band, group differences were detected at the following channels: C3 Gamma, C3 High Gamma, C3 Gamma 1, C4 High Gamma, Cz Gamma 2, P3 Gamma 1, P3 Gamma 2, F7 Gamma, F7 High Gamma, F7 Gamma 1 and F7 Gamma 2 channels. Finally, in Alpha band we detected group differences at following channels: C4 2, P3, T3 and T3 Alpha (see Table 2).

When 2 clinical groups were compared with post hoc analysis, no statistically significant difference between bipolar and borderline populations was observed. However, both groups were significantly different from the healthy population within those 55 variables (see Table 2). Table 3 illustrates the average values of the 3 groups.

Discussion

The main finding of the study was that although both BD and BPD are dissimilar from the healthy controls, they could not be differentiated from each other electrophysiologically. When group differences were examined in detail, it was observed that both groups had greater power in fast and slow oscillations as compared to the controls. This may indicate that these disorders could be biologically placed into the same category.

In this study, we used qEEG to explore biological differences between two conditions. qEEG is widely used in psychiatry as a biological marker and studies focus on absolute power values, source localization, evoked potentials and connectivity measures to identify disorder specific markers.[26] In depression, several studies showed alterations in alpha and theta band; however, research also suggests that qEEG can be used to find biomarkers predicting treatment response.[27] In addition, an altered theta/beta ratio is used to assist diagnosis in ADHD after several studies showing that this ratio is elevated in ADHD[28] (but also see Arns et al[29]). Besides depression and ADHD, studies suggest that qEEG variable can also be useful in several other psychiatric disorders: For instance, we showed that qEEG gamma band power could be used to evaluate insight level in schizophrenia[30] and in another study qEEG was used to determine subtypes of patients with obsessive-compulsive disorder.[31] These evidence suggest that qEEG could be evaluated as a reliable method to determine biological underpinnings of psychiatric disease.

Taken together, it can be said that the 2 entities are biologically similar but clinically different from each other. In practice, the differential diagnosis of the 2 disorders is solely based on reported symptoms. While the 2 disorders are quite similar in terms of symptoms, certain features such as presence of hypomania help to differentiate them from each other.[32] Differentiating the 2 disorders seems to be important from a

Table 2. Post Hoc Results of Tamhane's T2 for EEG Variables.

Analysis of Variance		Post Hoc (Tamhane's T2)		
Absolute Power (AP)-Channel-Band	*P*			*P*
AP-FP1-Delta	.047	BD	BPD	1.000
			Healthy	.032
AP-FP1-Theta	.046	BD	BPD	1.000
			Healthy	.013
AP-FP1-Beta 1	.036	BD	BPD	.811
			Healthy	.006
AP-FP2-Beta	.029	BD	BPD	.771
			Healthy	.005
AP-F3-Delta	.046	BD	BPD	.997
			Healthy	.006
AP-F4-Delta	.011	BD	BPD	.209
			Healthy	.001
AP-Fz-Delta	.011	BD	BPD	.748
			Healthy	.001
AP-C3-Delta	.018	BD	BPD	.597
			Healthy	.000
AP-C3-High Beta	.041	BD	BPD	.492
			Healthy	.038
AP-C3-Gamma	.036	BD	BPD	.868
			Healthy	.024
AP-C3-High Gamma	.018	BD	BPD	.701
			Healthy	.023
AP-C3-Gamma1	.034	BD	BPD	.884
			Healthy	.020
AP-C4-High Gamma	.000	BD	BPD	.712
			Healthy	.000
AP-C4-Alpha2	.000	BD	BPD	.832
			Healthy	.000
AP-C4-Beta3	.000	BD	BPD	.972
			Healthy	.000
AP-Cz-Delta	.000	BD	BPD	.929
			Healthy	.000
AP-Cz-Gamma 2	.000	BD	BPD	.850
			Healthy	.000
AP-P3-Delta	.000	BD	BPD	.999
			Healthy	.000
AP-P3-Theta	.000	BD	BPD	.999
			Healthy	.000
AP-P3-Alpha	.000	BD	BPD	.810
			Healthy	.000
AP-P3-Beta	.000	BD	BPD	.938
			Healthy	.000
AP-P3-Beta3	.000	BD	BPD	.925
			Healthy	.000
AP-P3-Gamma1	.041	BD	BPD	.868
			Healthy	.041
AP-P3-Gamma 2	.018	BD	BPD	.714
			Healthy	.036
AP-P4-Delta	.007	BD	BPD	.159
			Healthy	.000
AP-P4-Theta	.019	BD	BPD	.575
			Healthy	.004

(continued)

Table 2. (continued)

Analysis of Variance		Post Hoc (Tamhane's T2)		
Absolute Power (AP)-Channel-Band	*P*			*P*
AP-Pz-Delta	.005	BD	BPD	.392
			Healthy	.000
AP-Pz-Theta	.018	BD	BPD	.926
			Healthy	.003
AP-Pz-High Beta	.013	BD	BPD	.389
			Healthy	.045
AP-O1-Delta	.001	BD	BPD	.116
			Healthy	.000
AP-O1-Theta	.013	BD	BPD	.377
			Healthy	.010
AP-O1-Beta	.012	BD	BPD	1.000
			Healthy	.000
AP-O1-High Beta	.008	BD	BPD	.901
			Healthy	.005
AP-O1-Beta 1	.035	BD	BPD	.925
			Healthy	.002
AP-O1-Beta 2	.042	BD	BPD	.992
			Healthy	.005
AP-O1-Beta 3	.012	BD	BPD	.982
			Healthy	.001
AP-O2-Delta	.031	BD	BPD	.617
			Healthy	.001
AP-F7-Delta	.002	BD	BPD	.540
			Healthy	.001
AP-F7-Theta	.006	BD	BPD	.302
			Healthy	.003
AP-F7-Beta	.002	BD	BPD	.394
			Healthy	.016
AP-F7-High Beta	.022	BD	BPD	.408
			Healthy	.022
AP-F7-Gamma	.033	BD	BPD	.929
			Healthy	.030
AP-F7-High Gamma	.016	BD	BPD	.674
			Healthy	.011
AP-F7-Beta 1	.001	BD	BPD	.129
			Healthy	.022
AP-F7-Beta 2	.002	BD	BPD	.475
			Healthy	.019
AP-F7-Beta 3	.022	BD	BPD	.717
			Healthy	.029
AP-F7-Gamma 1	.047	BD	BPD	.932
			Healthy	.040
AP-F7-Gamma 2	.015	BD	BPD	.932
			Healthy	.017
AP-F8-Delta	.030	BD	BPD	.991
			Healthy	.009
AP-T3-Delta	.023	BD	BPD	.341
			Healthy	.004
AP-T3-Theta	.003	BD	BPD	.060
			Healthy	.003
AP-T3-Alpha	.021	BD	BPD	.857
			Healthy	.033
AP-T3-Alpha 1	.022	BD	BPD	.854
			Healthy	.032
AP-T4-Delta	.033	BD	BPD	.532
			Healthy	.000
AP-T4-Theta	.037	BD	BPD	.279
			Healthy	.003

Abbreviations: BD, bipolar disorder; BPD, borderline personality disorder.

Table 3. Back-Transformed Mean Values of the 3 Groups.

Absolute Power (AP)-Channel-Band	BD	BPD	Healthy
AP-FP1-Delta	82.43	84.88	34.96
AP-FP1-Theta	23.12	22.84	10.73
AP-FP1-Beta 1	3.76	3.2	1.98
AP-FP2-Beta	9.62	8.82	6.04
AP-F3-Delta	36.68	35.52	20.31
AP-F4-Delta	34.51	25.85	16.41
AP-Fz-Delta	37.14	32.09	18.02
AP-C3-Delta	27.92	23.55	14.53
AP-C3-High Beta	1.32	1.11	0.81
AP-C3-Gamma	0.71	0.63	0.38
AP-C3-High Gamma	0.06	0.05	0.02
AP-C3-Gamma1	0.56	0.51	0.3
AP-C4-High Gamma	1.22	1.04	0.9
AP-C4-Alpha2	6.47	7.28	4.48
AP-C4-Beta3	3.92	3.75	2.86
AP-Cz-Delta	33.08	29.66	25.06
AP-Cz-Gamma 2	0.17	0.14	0.1
AP-P3-Delta	28.66	21.5	14.69
AP-P3-Theta	13.54	10.81	7.58
AP-P3-Alpha	24.96	29.15	17.29
AP-P3-Beta	12.85	12.84	8.62
AP-P3-Beta3	4.43	4.44	2.93
AP-P3-Gamma1	0.52	0.46	0.3
AP-P3-Gamma 2	0.16	0.13	0.08
AP-P4-Delta	35.51	24.26	13.54
AP-P4-Theta	16	13.25	7.99
AP-Pz-Delta	40.67	30.93	15.46
AP-Pz-Theta	21.59	19.4	9.86
AP-Pz-High Beta	1.46	1.25	0.96
AP-O1-Delta	32.4	20.9	10.64
AP-O1-Theta	14.36	10.51	6.04
AP-O1-Beta	10.86	10.92	5.767
AP-O1-High Beta	1.23	1.12	0.62
AP-O1-Beta 1	4.54	4.14	2.32
AP-O1-Beta 2	2.18	2.43	1.34
AP-O1-Beta 3	3.87	4.17	2.09
AP-O2-Delta	36.98	27.95	14.46
AP-F7-Delta	46.21	36.38	17.57
AP-F7-Theta	12.43	9.81	6.4

(continued)

Table 3. (continued)

Absolute Power (AP)-Channel-Band	BD	BPD	Healthy
AP-F7-Beta	6.67	5.69	3.96
AP-F7-High Beta	1.27	1.03	0.73
AP-F7-Gamma	0.77	0.72	0.41
AP-F7-High Gamma	0.08	0.06	0.02
AP-F7-Beta 1	2.14	1.72	1.26
AP-F7-Beta 2	1.64	1.42	0.97
AP-F7-Beta 3	2.9	2.58	1.8
AP-F7-Gamma 1	0.6	0.56	0.33
AP-F7-Gamma 2	0.19	0.18	0.09
AP-F8-Delta	38.23	35.27	14.28
AP-T3-Delta	23.01	15.54	8.9
AP-T3-Theta	6.18	4.03	2.83
AP-T3-Alpha	4.58	4.07	2.25
AP-T3-Alpha 1	2.57	2.28	1.19
AP-T4-Delta	23.49	16.76	8.39
AP-T4-Theta	5.9	4.34	2.98

Abbreviations: BD, bipolar disorder; BPD, borderline personality disorder.

clinical point as the treatment differs. However, lack of a biological marker makes differentiation quite difficult. On the other hand, one should also acknowledge that other neuroimaging techniques such as PET, structural magnetic resonance imaging, and so on can prove that there are biological differences between BD and BPD.

The main limitation of this study was unequal sample size in groups as well as relatively small sample size particularly in the control group. Unequal sample sizes resulted from the retrospective nature of the study. However, further studies, with larger and equal sample sizes, may be necessary to confirm the findings. Another limitation is that we only used one EEG metric (ie, absolute power). Using several different metrics such as indices of connectivity and asymmetry could have identified markers differentiating 2 disorders. Our reason for using only absolute power was that it is the most commonly used EEG metric in qEEG studies. However, future studies should explore whether other EEG metrics could differentiate BD and BPD. If the hypothesis that the 2 disorders are biologically identical is confirmed, then BPD disorder patients should be included in the BD spectrum is confirmed. In that case, the search for biomarkers should target identifying outcomes such as occurrence of mania rather than targeting the differential diagnosis. To the best of our knowledge, this is the first EEG study comparing BD and BPD head-to-head.

Authors' Note

The study protocol was approved by the Uskudar University Ethics Committee. Mehmet Guven Gunver is now affiliated with Department of Biostatistics, Uskudar University, Istanbul, Turkey.

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Author Contributions

Mehmet Kemal Arıkan - Data collection, interpretation of data, writing of the report.
Mehmet Güven Günver - Statistical analysis, writing the report.
Barış Metin - Interpretation of data.
Nevzat Tarhan - Interpretation of data, funding.

Declaration of Conflicting Interests

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