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Inattention in Bipolar and Unipolar Depression: Event-Related Potentials Associated with Performing a GoNogo Task

Yeong Suk Lee, MD, Young Joon Kwon, MD, PhD, Hwa Young Lee, MD, Ji Sun Kim, MD, PhD, Se-Hoon Shim, MD, PhD

Department of Psychiatry, Soonchunhyang University Cheonan Hospital, Cheonan, Korea

Background: This study aimed to examine the changes in Nogo N2 and P3 amplitudes in patients with bipolar affective disorder (BD) or major depressive disorder (MDD) and in healthy controls (HCs). The association between attention and Nogo N2 and P3 changes was also investigated.
Methods: The study included 30 participants with BD, 30 participants with MDD, and 30 HCs aged 19-60 years. They performed a GoNogo task while their electroencephalograms were recorded. Beck Depression Inventory and State-Trait Anxiety Inventory were used for evaluation. Furthermore, behavioral measures and GoNogo N2 and P3 amplitudes were compared between the three groups.
Results: Patients with BD or MDD exhibited a significantly poorer performance in Nogo accuracy than the HCs. Patients with BD or MDD showed significantly lower Nogo N2 amplitudes at the frontal, fronto-central, and central electrodes than the HCs. In patients with BD or MDD, the Nogo N2 amplitudes at the frontal or fronto-central electrode were positively correlated with state of anxiety scores and inattention.
Conclusion: These findings suggest that decreased Nogo N2 amplitudes in the frontal or fronto-central areas could be a biological marker for inattention during depressive episodes associated with BD or MDD.

Keywords Event-related potentials; Bipolar disorder; Major depressive disorder; Attention

INTRODUCTION

Bipolar affective disorder (BD) and major depressive disorder (MDD) have been regarded as representative mood disorders. BD is common in clinical psychiatric practice, and several studies have estimated its prevalence to range from 0.5% to 5% in community-based samples [1]. The global point prevalence of MDD, adjusting for methodological differences, was 4.7% (4.4%-5.0%) [2]. In Korea, the lifelong prevalence of mood disorders, including MDD and BD, accounts for 5.3%, making it the third highest among mental disorders after substance-related disorder and anxiety disorder [3].

Almost half of all patients with BD type I and approximately three quarters of those with BD type II will first have an episode of depression [4]. Because the first de-

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Correspondence: Ji Sun Kim, MD, PhD
Department of Psychiatry, College of Medicine, Soonchunhyang University Cheonan Hospital, 31 Suncheonhyang 6-gil, Dongnam-gu, Cheonan 31151, Korea
TEL +82-41-570-2280 **FAX** +82-41-592-3804 **E-mail** ideal91@hanmail.net **ORCID** https://orcid.org/0000-0003-2472-4591
Co-Correspondence: Se-Hoon Shim, MD, PhD
Department of Psychiatry, College of Medicine, Soonchunhyang University Cheonan Hospital, 31 Suncheonhyang 6-gil, Dongnam-gu, Cheonan 31151, Korea
TEL +82-41-570-3853 **FAX** +82-41-592-3804 **E-mail** shshim2k@daum.net **ORCID** https://orcid.org/0000-0002-3137-6591
Ji Sun Kim and Se-Hoon Shim contributed equally to this study.

pressive symptoms of the BD and the MDD are similar, long delays between symptom onset, treatment-seeking, and receipt of a bipolar diagnosis were common [5]. When BD has in essence occurred, the appropriate intervention in the early stage should prevent progress of the disease [6]. Misdiagnosis of BD as unipolar depression is a serious clinical problem [7]. Therefore, distinguishing between BD and MDD is paramount for clinicians in order to avoid risks of misdiagnosis and inappropriate medication treatments [8].

As part of this effort, multiple studies have tried to find out the clinical features for differential diagnosis between unipolar and bipolar depression [4,7]. Promising biomarkers to support the differential diagnosis between the two conditions has been needed.

Previous studies have reported that patients with BD and MDD did worse on attention-related tasks than did the healthy population. BD patients, even in the euthymic phase, were impaired in tasks of attentional set shifting, verbal memory and sustained attention [9]. Attentional impairment during the depressed phase of BD may be specific to effortful processing [10]. Sustained-attention deficit is present early in the course of the BD, but becomes more pronounced with repeated episodes [11]. The patients with MDD were impaired in the task of attentional-set shifting, requiring more trials to meet the criterion at the intradimensional stage of the task and being more likely to fail the task at the extradimensional shift stage than were controls [12]. The previous studies pointed out that patients with either BD or MDD had problems with attention in depression [10,12]. For the severity of impairment in attention, one study reported that the patients with BD had lower sustained attention than did those with MDD [11]. Another study has shown that MDD is not related to sustained attention rather than BD even HC [13] and suggested that bipolar depression could have much more impaired attention than dose unipolar depression.

Meanwhile, event-related potentials (ERP), in particular, is a brain-wave component that reflects cognitive function. Two constituents of the ERP, the N200 and P300 (N2 and P3), appear to be closely associated with the cognitive processes of selective attention [14]. Specifically, the GoNogo paradigm is a good way to see impaired attention [15]. Cognitive control related to GoNogo involves the ability to monitor conflicting response options and the subsequent decision to respond in a context-dependent manner [16]. Selective attention to relevant information and the inhibition of distracting information are involved in the process of cognitive control in the GoNogo paradigm [16]. There is electrophysiological evidence that action errors during a GoNogo task can result from sustained attention failures [17]. In the GoNogo paradigm, top-down attentional control plays a crucial role in inhibitory control for distracting stimuli [18]. There have been many previous studies related to the N2 and P3 ERP changes using the GoNogo paradigm and the cognitive dysfunction, including attention related to inhibition tasks in BD and MDD [19,20].

However, so far, no studies have compared the behavioral data and ERP in the GoNogo paradigm in patients with BD or MDD and with those in a healthy population.

Moreover, there has been no agreement between the existing studies.

Hence, we hypothesized that there was a significant difference in attentional behavioral data, such as task accuracy, false alarm rate, and reaction time, between the patients with BD or MDD and the healthy population. In addition, referring to a previous study in which patients with BD had lower sustained attention than did those with MDD, we hypothesized that the decline in attention appeared in the order of BD, MDD, and normal control. Then we also assumed that it would also appear in the Nogo N2 and P3 amplitudes in that order. Our aim in the study was to evaluate the attentional control reflected by GoNogo tasks and the differences in Nogo N2 and P3 amplitudes between patients with BD or MDD, and healthy controls (HCs), as well as the relationship between Nogo ERP and attentional dysfunction in the study population.

MATERIALS AND METHODS

1. Subject

The study participants with BD and MDD who visited the department of Psychiatry at Soonchunhyang University Cheonan Hospital were enrolled from January 2018 to February 2020. All cases were between the ages of 19 and 60. The patients with BD and MDD were diagnosed according to the criteria of the Diagnostic and Statistical Manual of Mental Disorder, 5th edition; these were ones who has been hospitalized or seen as outpatient because of having symptoms of depression [21]. The study was done with 30 patients with BD (10 male and 20 female,

with a mean age of 29.67±10.35 years) and 30 patients with MDD (14 male and 16 female, with a mean age of 33.07±13.38 years). Participants with any history of pregnancy, mental retardation, neurological or other severe medical diseases, a history of alcohol or substance abuse/dependence, or head trauma were excluded from the study by means of the initial screening interviews. The HC group consisted of 30 physically and mentally healthy volunteers (14 male and 16 female, with a mean age of 27.87±5.95 years) from the local community recruited through newspapers and posters. Each participant had normal hearing ability; 21 of the patients with BD were taking mood-stabilizing agents (lithium, valproate) with or without atypical antipsychotics (risperidone, quetiapine, aripiprazole, or olanzapine); and 16 of the patients with MDD were taking medications, such as selective serotonin reuptake inhibitors (escitalopram, and sertraline), a serotonin and norepinephrine reuptake inhibitor (duloxetine, venlafaxine, desvenlafaxine), or others (mirtazapine, trazodone, tianeptine). This study was approved by the Institutional Review Board and Ethics Committee of Soonchunhyang University Cheonan Hospital, and all experimental protocols were approved by the committee (2020-03-010). All participants received written and oral explanations regarding the study and provided written informed consent.

2. Clinical measures

To evaluate emotional functioning that is a depressive and anxiety symptom, we used the Beck Depression Inventory (BDI) and the State-Trait Anxiety Inventory (STAI). The BDI is a self-reporting examination developed to measure depression. It consists of 21 items, with each item's score ranging from 0 to 3, and the total score ranging from 0 to 63. The higher scores are positively correlated with a severe level of depression [22].

The STAI is a self-reporting examination developed to measure two types of anxiety. STAI consists of total 40 items (20 items each of the two type); each item's score ranges from 1 to 4. Higher scores are positively correlated with a higher level of anxiety [23].

3. EEG data acquisition and analysis

EEG was acquired using a NeuroScan SynAmps amplifier (Compumedicus USA, El Paso, TX, USA) with 64 Ag/AgCl electrodes mounted on a Quik Cap. Subjects were seated approximately 60 cm away from the computer screen in a relaxed sitting position in a silent room. Electrodes were placed as central (Cz) and frontal (Fz), and an earth electrode was placed fronto-parietal according to the extended 10-20 placement scheme. An electrode was placed infra-orbitally to monitor eye movement. Reference electrodes were placed at the mastoid, and the impedance was less than 5 kΩ. The band-pass filter was set at 0.1-100 Hz and sampled at 1,000 Hz. The procedure for the EEG acquisition followed our previous study [24].

The EEG data were preprocessed using CURRY 8 (Compumedics USA, Charlotte, NC, USA). Gross artifacts were rejected by visual inspection by a trained person. Eye-movement artifacts were removed using the mathematical procedure in the preprocessing software. Data were filtered using a 0.1-30 Hz band-pass filter and epoched from 100 ms pre-stimulus to 600 ms post-stimulus. These epochs were subtracted from the average value of the pre-stimulus interval for baseline correction. If any remaining epochs continued to have significant physiological artifacts (amplitude exceeding±75 μV) in any of the 62 electrode sites, they were excluded from further analysis. Only artifact-free epochs were averaged across trials and participants for ERP analysis. Based on the previous studies showing that Nogo ERP reflected behavioral inhibition, we included Nogo trials in ERP analysis.

4. Behavioral task paradigm

As stimuli for the GoNogo task, we applied the 'oddball paradigm' of auditory stimulation. ERPs were elicited binaurally through headphones. The subjects were instructed to press the spacebar as accurately and quickly as possible when the target tone appeared and not to respond when the non-target tone appeared. There were 400 trials, which consisted of Go (85% probability) and Nogo (15% probability) tones. The target tone (Nogo) was 1,500 Hz, and the nontarget tone (Go) was 1,000 Hz, with a 1,500-ms interval before the next trial. These stimuli were generated with E-prime software (Psychology Software Tools, Pittsburgh, PA, USA). In the Go condition, N200 (the most negative peak between 150 and 350 ms after stimulus onset, N2) and P300 (the most positive peak between 250 and 500 ms after stimulus onset, P3) were investigated at the Fz, fronto-central (FCz), Cz, and parietal (Pz) electrodes. Also, in the Nogo

condition, the N2 and the P3 were investigated at the Fz, FCz, Cz, and Pz electrodes. We focused on the changes of N2 and P3 at the frontal and FCz electrode, because BD and MDD have been regarded as a mental illness with frontal-lobe dysfunction, and the previous studies on BD or MDD patients generally showed changes of N2 and P3 in the FCz regions [20,25]. The time window we assumed was based on the previous studies [26]. To accumulate behavioral data, Go accuracy, Nogo accuracy, and reaction times were calculated based on the data of E-prime software. Nogo accuracy was calculated to find the false-alarm rate of responses to non-target stimuli.

5. Statistical analysis

For descriptive statistics, we used frequency distributions, continuous variables, arithmetic means, and standard deviation values. Three groups were compared using the chi-square test for discontinuous variables in differences in the demographic variables. For the continuous variables, after verifying the normality by means of the Shapiro-Wilks test, we evaluated whether there is a statistically significant difference using the parametric t-test or the non-parametric Mann-Whitney test. We used one-way analysis of variance (ANOVA) with a *post hoc* LSD test to compare the scores of psychological and behavioral data between the patients with BD or MDD and the HC groups. N2 and P3 amplitudes and latencies of patients and HCs were initially evaluated by using repeated measures ANOVA with electrodes (Fz, FCz, Cz, and Pz) as the within-subject factor and groups (BD, MDD, and HCs) as the between-subjects factor. The multivariate analysis of variance (MANOVA) and repeated measures ANOVA were used to control for education as covariates. We did a partial Pearson's correlation analysis between GoNogo ERP and psychological measures and behavioral data in patient groups with a 5,000-bootstrap resampling technique to correct for multiple correlations. We used a partial Pearson's correlation in patient groups to control for illness duration as a covariate. We additionally conducted multiple regression analysis for Nogo N2 amplitude and psychological data such as State Anxiety Inventory (SAI), Trait Anxiety Inventory (TAI), BDI which is different between the patients with BD or MDD and HCs. Comparisons were considered significant at $p<0.05$. For statistical analyses, we used IBM SPSS Statistics for Windows, Version 21.0 (IBM Co., Armonk, NY, USA).

RESULTS

1. Subjects

Table 1 shows the demographic and clinical symptom characteristics of the patients with BD and MDD and the HCs. There were no significant differences in the groups according to age and sex. The three group differed significantly in education ($p<0.001$), because the HC group had significantly more years of education than did the patient groups, as shown by chi-square tests and ANOVA with a *post-hoc* LSD test. The BD and MDD groups showed no significant difference in years of education. There were no significant differences in STAI state,

Table 1. Demographic and clinical symptoms characteristics in all participants

Variable	BD (n=30)	MDD (n=30)	HCs (n=30)	p-value	*Post-hoc* (LSD)
Age (y)	29.67±10.35	33.07±13.38	30.17±4.96	0.381	
Sex				0.483	
Male	10 (33.3)	14 (46.7)	14 (46.7)		
Female	20 (66.7)	16 (53.3)	16 (53.3)		
Education (y)	12.83±1.91	12.23±2.05	16.33±1.18	<0.001	a, b<c
Duration of illness (mo)	126.90±105.64	27.60±32.16		<0.001	a>b
STAI state	64.43±10.79	64.30±10.75	33.97±8.96	<0.001	a, b>c
STAI trait	65.37±7.97	63.40±10.76	36.17±8.81	<0.001	a, b>c
BDI	34.63±12.88	32.97±14.64	3.43±3.42	<0.001	a, b>c

Values are presented as mean±standard deviation or number (%).
BD, bipolar affective disorder; MDD, major depressive disorder; HC, healthy control; STAI, State-Trait Anxiety Inventory; BDI, Beck Depression Inventory.

STAI trait, or BDI between the BD and MDD groups.

2. Behavioral outcomes

Table 2 shows Nogo N2 and P3 behavioral outcomes. There was no significant difference in the Go accuracy between the patients with BD or MDD and HC groups (p=0.063). However, the BD group performed significantly worse than did the HCs (p=0.025). The three groups showed significant differences in the Nogo accuracy (p=0.001). The patients with BD or MDD performed significantly worse than did the HCs (BD, 90.07±12.24; MDD, 89.20±10.85; HC, 97.90±4.49). In the Go reaction time, there was no significant difference between the three groups (p=0.158). The BD or MDD groups showed a higher false-alarm rate than did the HCs (BD, 9.93±12.24; MDD, 10.80±10.85; HC, 2.10±4.50).

3. ERP

1) Amplitude

Table 3 shows the means and standard deviation for Nogo N2 and P3 amplitudes in the BD, MDD, and HC groups. The grand average of the Nogo ERPs at the Fz electrode for each group is shown in Fig. 1. The three groups showed significant differences in N2 amplitude at the frontal, FCz, and Cz electrodes (p=0.009, 0.004, 0.013). The BD and MDD groups had a significantly lower Nogo N2 amplitude at the frontal electrode than did the HC group (p=0.011, 0.002). The BD and MDD group had a significantly lower Nogo N2 amplitude at the FCz electrode than did the HC group (p=0.002, 0.002). And the BD or MDD group had a significantly lower Nogo N2 amplitude at the Cz electrode than did the HC group (p=0.005, 0.008). The three groups showed significant differences in P3 amplitude at the Pz

Table 2. Comparison of behavioral outcomes between patients with BD or MDD, and HCs

Variable	BD (n=30)	MDD (n=30)	HC (n=30)	p-value	*Post-hoc*, p-value		
					BD vs. MDD	BD vs. control	MDD vs. control
Go accuracy (%)	94.57±10.24	95.50±6.38	98.77±2.76	0.063	0.614	0.025	0.080
Nogo accuracy (%)	90.07±12.24	89.20±10.85	97.90±4.50	0.001	0.733	0.003	0.001
False alarm rate (%)	9.93±12.24	10.80±10.85	2.10±4.50	0.001	0.733	0.003	0.001
Reaction time (ms)	442.91±79.55	487.61±114.34	453.38±81.61	0.158	0.067	0.665	0.159

Values are presented as mean±standard deviation.
BD, bipolar affective disorder; MDD, major depressive disorder; HC, healthy control.

Table 3. Comparison of the amplitudes between patients with BD or MDD and HCs in Nogo condition

Variable	BD (n=30)	MDD (n=30)	HC (n=30)	p-value	*Post-hoc*, p-value		
					BD vs. MDD	BD vs. control	MDD vs. control
Amplitude (μV)							
N2 Fz	−3.94±2.73	−3.34±2.96	−5.45±3.71	0.009	0.330	0.011	0.002
N2 FCz	−3.48±2.95	−3.41±3.23	−5.37±3.39	0.004	0.702	0.002	0.002
N2 Cz	−2.01±2.62	−2.18±2.88	−3.29±3.00	0.013	0.929	0.005	0.008
N2 Pz	−0.06±2.41	−0.22±1.96	−0.10±2.36	0.817	0.916	0.530	0.611
P3 Fz	2.62±4.14	2.23±2.85	2.86±3.60	0.813	0.655	0.756	0.540
P3 FCz	5.41±5.23	3.94±4.16	5.20±4.06	0.350	0.190	0.840	0.254
P3 Cz	5.79±4.78	4.44±3.98	6.18±3.72	0.186	0.180	0.417	0.085
P3 Pz	5.22±3.52	5.03±3.17	7.22±4.52	0.019	0.675	0.011	0.008

Values are presented as mean±standard deviation.
BD, bipolar affective disorder; MDD, major depressive disorder; HC, healthy control; Fz, frontal; FCz, fronto-central; Cz, central; Pz, parietal.

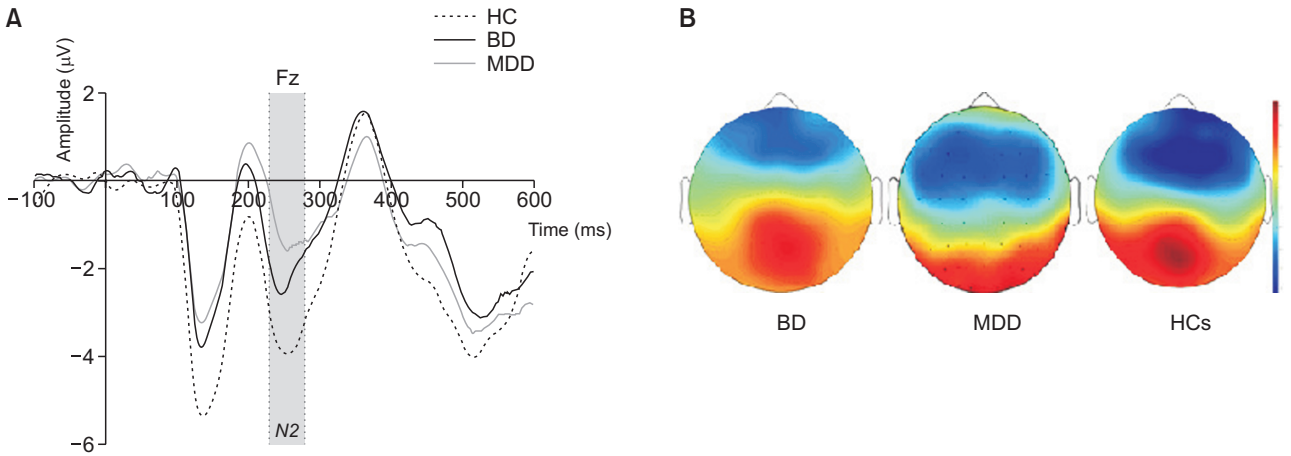

Fig. 1. (A) The grand average Nogo N2 and P3 waveforms, and (B) topographic maps of Nogo amplitudes at Fz in patients with BD or MDD and HCs. Fz, frontal; BD, bipolar affective disorder; MDD, major depressive disorder; HC, healthy control.

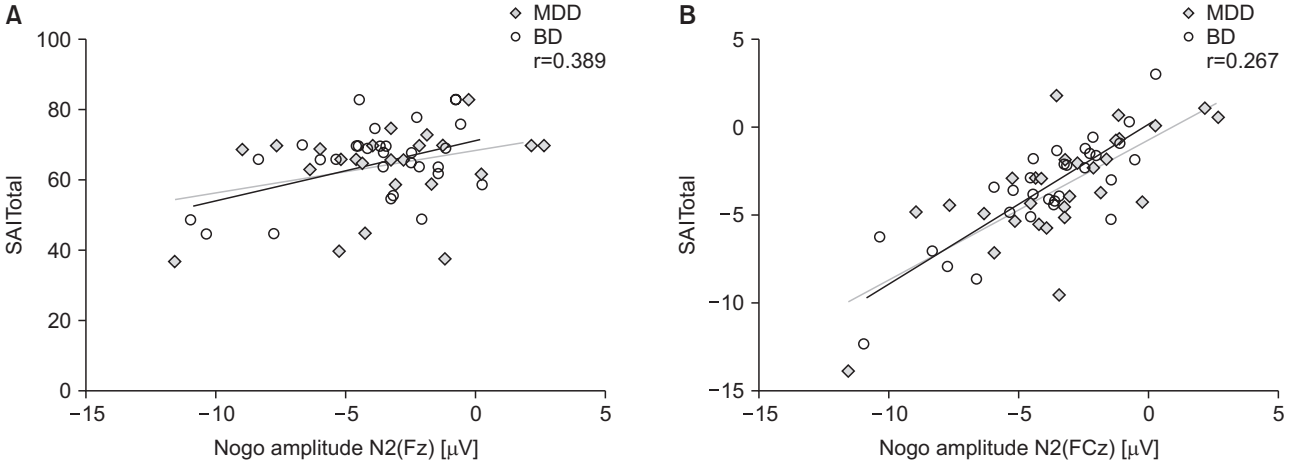

Fig. 2. (A) Scatter plots of Nogo N2 amplitude at Fz and SAI score change in patients with BD or MDD. (B) Scatter plots of Nogo N2 amplitude at FCz and SAI score change in patients with BD or MDD. MDD, major depressive disorder; BD, bipolar affective disorder; SAI, State Anxiety Inventory; Fz, frontal; FCz, fronto-central.

electrode (p=0.019). The BD group had a significantly lower Nogo P3 amplitude at the Pz electrode than did the HC group (p=0.011). The MDD group had a significantly lower Nogo P3 amplitude at the Pz electrode than did the HC group (p=0.008).

2) Correlations

In correlation analysis between the clinical symptoms and ERPs measures in the BD and MDD groups, Nogo N2 amplitudes at the frontal electrode were positively correlated with the SAI (*r*=0.389, p=0.003) (Fig. 2A). The Nogo N2 amplitudes at the FCz electrode were positively correlated with the SAI state (*r*=0.267, p=0.043) (Fig. 2B).

3) Multiple regression

Multiple regression analyses as Nogo N2 amplitude as a dependent variable showed significant association with SAI after adjusting age, sex and TAI, BDI at frontal electrode (R^2=0.213, standardized β=0.437, t=2.735, p=0.008). At FCz electrode, it also showed significant association with SAI after adjusting age, sex and TAI, BDI (R^2=0.095, standardized β=0.509, t=2.971, p=0.004).

DISCUSSION

In this study we aimed to identify the differences in attentional behavioral data and the Nogo N2, P3 ERP

by GoNogo paradigm between the BD, MDD, and HC groups. As we hypothesized, behavioral data such as Nogo accuracy differed between the three groups, but not in the order of BD, MDD, HC. In the patient groups, Nogo N2 amplitudes were significantly reduced in three sites, Fz, Fcz and Cz, compared to the normal group. For the Nogo P3 amplitudes, there was a difference in the Pz site, with BD and MDD significantly decreased compared to the normal group.

First, the patients with BD or MDD did significantly worse in the Nogo accuracy, false-alarm rate than did the HCs. False-alarm rate and commission error reflects poor motor-response inhibition, whereas reaction time and omission error reflects poor sustained and selective attention [27,28]. We found that there was a significant difference in false-alarm rate and commission rate (Nogo accuracy) between the patients with BD or MDD and the HCs. Previous studies had found that BD and MDD patients had a higher false-alarm rate than did the HCs [29,30]. Regarding the previous studies, our results suggest the possible difference in impulse control between BD, MDD and HCs. However, our study did not show any differences between BD and MDD.

Second, the patients with BD or MDD showed significantly lower Nogo N2 amplitudes in the Fz, FCz, or Cz electrodes than did the healthy population. The previous study showed that decreased N2 amplitudes in patients with BD reflected difficulties of the early stages of the inhibition and the impairment of the ability to control and monitor responses [19]. Moreover, there was previous evidence that the decreased N2 amplitudes was caused by the disruption in the response monitoring and control process for the patients with MDD [20]. As in the previous studies that showed decreased N2 amplitude, impaired attention in both patients with BD and those with MDD was also shown by decreased N2 amplitudes in this study, which also showed the changes of N2 amplitude in the frontal and FCz areas. There have been several previous reports that prefrontal dysfunctions appear in the depressed states [31-33].

Third, the Nogo P3 amplitudes of patients with BD and MDD were decreased in the Pz electrode compared to those of the healthy population. Since P3 reflects attention and working memory [34], our finding of difference of Nogo P3 amplitudes between the patients with BD or MDD and the HCs would reflect the differences of attentional function between the three groups, as is consistent with previous studies which suggested that P3 and temporo-parietal areas were involved [35]. Their conclusion was that integrity of the temporo-parietal junction is critical for P3.

Nogo N2 amplitudes in the frontal and FCz electrodes were positively correlated with SAI scores in the patient groups. The relationship between Nogo N2 amplitude and SAI was also significant in the multiple regression analysis. This discovery allowed us to think about the relationship between anxiety and decreased attention. Previous studies have shown that, when anxiety levels are very high, they could over-activate the orientating and alerting functions and reduce the capacity of attentional control [36]. Trait anxiety was related to deficiencies in the executive-control network, but state anxiety was associated with an over-functioning of the alerting and orienting networks [37]. The results are similar to the previous experiment's conclusions that N2 and P3 amplitudes were related to anxiety and cognitive self-evaluation [38]. According to the previous study, decreased N2 amplitudes reflected the difficulties of the early stages of the inhibition [19]; hence a high state of anxiety can be difficult to control because of anxiety in the early stage of inhibition. In our study, we found that the state of depression also has to do with the increase in state anxiety, which means a decline in attention, leading to a decline in N2 amplitude.

Meanwhile, unexpectedly, there was no difference between the Nogo amplitudes of patients with BD or MDD. N2 or P3 amplitude differences between the BD and MDD groups were not significant in the present study. The results of attentional behavioral tasks in the study also showed no significant differences between BD and MDD groups. Originally, we hypothesized that patients with BD would show more impairment of sustained attention than would the patients with MDD, considering the results of the previous study, which showed that the sustained-attention deficit in bipolar disorder may reflect an underlying predisposition to distractibility or poor attentional control, which in turn may lead to an inability to direct attention away from distracting stimuli/environmental events and may manifest as emotional lability [39]. Moreover, another previous study that showed much more impairment of sustained attention in bipolar depression used rapid visual processing computerized tasks [13]. It cannot be directly compared to the previous study, because there was no study to evaluate the electrophysiological differences between bipolar depression and unipolar depression. Although our

results were not consistent with most previous studies about the sustained attention, there was a previous study that showed no difference in attention between BD and MDD [40]. One previous study showed no difference of attention between bipolar depression and unipolar depression using digit span forward and backward [40]. For the study that showed much more impairment of sustained attention in bipolar depression used computerized tasks [13], no difference between BD and MDD in the study might have resulted from using a different task (GoNogo) to evaluate sustained attention. The GoNogo paradigm that we used might be different from the above computerized task, which might have been so easy that it might not have made a significant difference between the groups. However, the study also showed no significant difference in N2 and P3 amplitudes between the BD and MDD groups. Given the importance of biological measures to evaluate cognitive status in depression, our results should be focused and replicated in future studies. In addition, psychopharmacologic medication of the patients in the present study could have influenced the results; because we evaluated patients in a depressed state and ethical reasons should be considered, all kinds of medication were accepted in this sample of severely ill patients. Further study to verify the differences in attention and electrophysiological changes between BD and MDD patients with tasks of proper difficulty excluding the medication effects should be needed.

Our study has limitations. First, the patient groups and HCs could not be matched for education in spite of the correction of the demographic variables as covariates. The education of patient groups was significantly lower than that of the HC group. Although the difference in education between the study groups in this study might reflect clinical reality, further study using a matched population would be needed. Second, the patients with BD and MDD were taking medication at the time of testing. For the medication effects in N2 and P3 ERP, a further study to control medication effects will be needed. Third, the relatively small sample makes it difficult to generalize our findings. Because of the relatively small sample size, there might be no difference between the BD and the MDD ERPs. Further study to find neurophysiological biomarkers related to inattention in larger BD, MDD population with progressed behavioral tasks would be needed. Last, other clinical scales reflecting the characteristics of GoNogo paradigms such as impulsivity scales could not be used in the study. Future study will be needed using other clinical scales related to the paradigms.

Despite such limitations, to the best of our knowledge, this study is the first to explore electrophysiological differences between patients with BD or MDD and a HC group. Our study suggests that decreased Nogo N2 amplitudes in the frontal, FCz, and Cz area might be biological markers for inattention in mood disorders. In addition, the state of anxiety of a mood disorder could affect inattention and might present decreased Nogo N2 amplitudes in the depressed patients. Detection of an electrophysiological marker of inattention could support the effort of clinicians to provide proper assessment for first-experienced depressive symptoms.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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ORCID

Yeong Suk Lee
https://orcid.org/0000-0001-7927-5530
Young Joon Kwon
https://orcid.org/0000-0001-9340-0895
Hwa Young Lee
https://orcid.org/0000-0002-2749-6232
Ji Sun Kim
https://orcid.org/0000-0003-2472-4591
Se-Hoon Shim
https://orcid.org/0000-0002-3137-6591

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