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# Response inhibition in borderline personality disorder: event-related potentials in a Go/Nogo task

**M. Ruchsow[1], G. Groen[2], M. Kiefer[2], A. Buchheim[3], H. Walter[4], P. Martius[5], M. Reiter[2], L. Hermle[1], M. Spitzer[2], D. Ebert[1], M. Falkenstein[6]**

[1] Department of Psychiatry, Christophsbad Göppingen, Germany
[2] Department of Psychiatry, University of Ulm, Ulm, Germany
[3] Department of Psychotherapy and Psychosomatic Medicine, University of Ulm, Ulm, Germany
[4] Division of Medical Psychology, Department of Psychiatry, University of Bonn, Bonn, Germany
[5] Psychosomatic Hospital ''Klinik Höhenried'', Bernried, Germany
[6] Leibniz Research Centre for Working Environment and Human Factors Dortmund, Dortmund, Germany

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**Summary.** Borderline personality disorder (BPD) has been related to a dysfunction of anterior cingulate cortex, amygdala, and prefrontal cortex and has been associated clinically with impulsivity, affective instability, and significant interpersonal distress. We examined 17 patients with BPD and 17 age-, sex-, and education matched control participants with no history of Axis I or II psychopathology using event-related potentials (ERPs). Participants performed a hybrid flanker-Go/Nogo task while multichannel EEG was recorded. Our study focused on two ERP components: the Nogo-N2 and the Nogo-P3, which have been discussed in the context of response inhibition and response conflict. ERPs were computed on correct Go trials (button press) and correct Nogo trials (no button press), separately. Groups did not differ with regard to the Nogo-N2. However, BPD patients showed reduced Nogo-P3 amplitudes. For the entire group ($n = 34$) we found a negative correlation with the Barratt Impulsiveness Scale (BIS-10) and Becks's depression inventory (BDI).

The present study is the first to examine Nogo-N2 and Nogo-P3 in BPD and provides further evidence for impaired response inhibition in BPD patients.

**Keywords:** Response inhibition; event-related potentials; Nogo-N2; Nogo-P3; borderline personality disorder

## Introduction

Essential features of borderline personality disorder (BPD) are significant intra- and interpersonal distress, affective instability, and marked impulsivity (APA 1994). Among these, impulsivity has received special attention as one of the core symptoms of this disorder (Schmahl et al. 2002).

Correspondence: Martin Ruchsow, Department of Psychiatry, Christophsbad, Faurndauer Str. 6-28, 73035 Göppingen, Germany
e-mail: martin.ruchsow@christophsbad.de

Structural and functional imaging studies support the notion that BPD is associated with a dysfunctional frontolimbic network which consists of the anterior cingulate cortex (ACC), the orbitofrontal and dorsolateral prefrontal cortex, the hippocampus, and the amygdala (Lieb et al. 2004). Studies with fluorodeoxyglucose-positron-emission-tomography (FDG-PET) demonstrated altered baseline metabolism in prefrontal regions including the ACC (De la Fuenta et al. 1997; Juengling et al. 2003; Soloff et al. 2003). These brain areas have been suggested to play a role in dysfunctional serotonergic neurotransmission (Soloff et al. 2000), which has been associated with disinhibited impulsive reactions in patients with BPD (Hansenne et al. 2002).

According to Barratt (1985) impulsivity is not a unidimensional trait but rather consists of three factors: 1) a motor impulsiveness sub-trait (Mot) involving acting without thinking (''I act on the spur of the moment''), 2) a cognitive impulsiveness sub-trait (Cog) that involves fast cognitive decisions (''I make up my mind quickly''), and 3) a non-planning impulsiveness sub-trait (NP) that involves lack of ''futuring'' (prospective reasoning) which heavily relies on social conventions and norms (''I am more interested in the present than the future'').

In the laboratory, inhibition and impulsiveness can be assessed with the stop task (e.g. Logan et al. 1984) or with the Go/Nogo task (e.g. Pfefferbaum et al. 1985). While in

the stop task neurophysiological methods, like event-related potentials are problematic because of the overlap of response- and stop-signal ERPs, the Go/Nogo task yields a relatively simple ERP structure.

Two major ERP components have been described in Go/Nogo tasks. A first component, the N2 or (Nogo-N2), is seen as a phasic negative shift in Nogo compared to Go trials (e.g., Kopp et al. 1996; Kaiser et al. 2003). It can be observed with a peak latency between 250 and 350 msec post-stimulus with maximum over frontal scalp locations. The second component, the Nogo-P3, is a large positive deflection at about 300–600 msec post-stimulus which shows a frontocentral maximum in contrast to the parietal P3 or P3b which is seen in Go trials (Pfefferbaum et al. 1985; Strik et al. 1998). The P3b has been linked to response-related cognitive processes (e.g. Friedman et al. 1978; Falkenstein et al. 1994). Both Nogo-N2 and -P3 have been discussed controversially. The Nogo-N2 has been argued to reflect a cognitive top-down inhibition mechanism to suppress the incorrect tendency to respond operating at a processing stage prior to motor execution (Falkenstein et al. 1999; Kaiser et al. 2003; Kim et al. 2007). Contrary to that, Nieuwenhuis et al. (2003) and Donkers and van Boxtel (2004) suggested that the N2 mirrors conflict processing rather than response inhibition. A core assumption of this hypothesis is that Nogo ''responses'' compete and conflict with overt responses. The ''conflict approach'' was further corroborated by Jones et al. (2002) who demonstrated that a connectionist model implementing a conflict detection mechanism can account for behavioural and fMRI findings in Go/Nogo tasks. Moreover, the Nogo-N2 seems to be sensitive to manipulations of stimulus frequency as well as congruency effects resulting in different levels of response conflict (Bartholow et al. 2005). In summary, the Nogo-N2 appears to be related to conflict per se or rather the consequences of a conflict, namely inhibition or revision of inappropriate response tendencies.

The Nogo-P3 has been proposed to reflect response inhibition, as well (Herrmann et al. 2003). In several studies a clear relation of the Nogo-P3 to response inhibition (e.g. Bruin and Wijers 2002; Burle et al. 2004; Bekker et al. 2004; Smith et al. 2006) was shown. Because of its rather long latency with respect to the overt response in Go trials the Nogo-P3 may not reflect response inhibition itself, but rather its termination (e.g. Dimoska et al. 2006). Contrary to that Salisbury et al. (2004) and Verleger et al. (2006) suggested that an increased frontal P3 in Nogo trials cannot be attributed solely to response inhibition, but is due to or influenced by the absence of an overlap of movement-related potentials which rather influence the P3 on Go trials. However, the high amplitude of the Nogo-P3 in most studies (e.g., Falkenstein et al. 2002) makes its unlikely that it is solely related to slight variations of motor activity between Go and Nogo trials (Verleger et al. 2006). In summary, the vast majority of the current literature suggests that the Nogo-P3 is related to motor inhibition.

Source localizations of the Nogo-N2 and Nogo-P3 components in a Go/Nogo task revealed a bilateral source pair in the orbitofrontal and anterior cingulate cortex both for children (Jonkman et al. 2007) and adults (Bokura et al. 2001).

There are only few studies that have investigated N2 and P3 in subjects with psychiatric disorders. Kiehl et al. (2000) found alterations of N2 and P3 in individuals with schizophrenia and psychopathy. Also, a reduction of the N2 has been found in children with ADHD (e.g. Pliszka et al. 2000). As to BPD, there are altogether only very few neurophysiological studies. Using a two-tone discrimination task some studies found longer latencies and smaller amplitudes of the P3b (Blackwood et al. 1986; Kutcher et al. 1987), while Meares et al. (2005) found abnormally enhanced amplitudes of the P3a in BPD patients. The P3b is usually associated with controlled goal-directed processing whereas the P3a component is closely associated with prefrontal cortical mechanisms of automatic attention (Barceló et al. 2002). We (Ruchsow et al. 2006) and others (De Bruijn et al. 2006) have shown a reduction of error processing, as reflected in the Ne/ERN, in BPD.

The goal of the present study was to verify whether the impairment of response inhibition processes in BPD patients due to increased levels of impulsivity (Schmahl et al. 2002; Völlm et al. 2004) as shown on a behavioural level (Logan et al. 1984; Pfefferbaum et al. 1985) can also be demonstrated on a neurophysiological level, using ERPs. To this end, we used a speeded Go/Nogo task which requires the inhibition of pre-potent responses to one kind of stimuli (Nogo stimuli) and the execution of speeded responses to another kind of stimuli (Go stimuli). As Nogo-N2 and Nogo-P3 are generated in brain regions (Bokura et al. 2001; Jonkman et al. 2007) which are involved in the psychopathology and neurobiology of BPD (please see above) we expected reduced Nogo-N2 and Nogo-P3 amplitudes in BPD patients reflecting impaired response inhibition processes.

Assuming that Nogo-N2 amplitudes reflect response conflict rather than response inhibition we expected no between group differences in the present study as the number of incongruent trials (''high conflict trials'') and congruent trials (''low conflict trials'') were the same for both groups (see below).

## Materials and methods

### *Subjects*

One left-handed and sixteen right-handed (according to the Edinburgh Inventory, Oldfield 1971) participants (two male) served as control group (demographic data summarized in Table 1).

One left-handed and sixteen right-handed inpatients (one male) with borderline personality disorder (APA 1994) from the Psychiatric Hospital of the University of Ulm (Germany) and from the Psychosomatic and Psychiatric Hospital, Bad Wiessee (Germany) participated in the study. Diagnosis of BPD was established by using the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II; First et al. 1997). Severity of BPD was assessed with the Barratt Impulsiveness Scale, version 10 (BIS-10; Barratt 1985); severity of depression was assessed by the Beck's depression inventory (BDI; Beck et al. 1961). Before participating in the study, all control subjects received a clinical interview and were asked for history of Axis I or II psychopathology (''Did you ever contact a psychiatrist, psychotherapist or psychiatric hospital in your life?''). If this was the case they were excluded from the study. Subjects with history of electroconvulsive therapy (ECT), neurological or general medical disorders were excluded from the study, as well. Twelve of the patients were medicated. Medication was stable during the last two weeks before examination (for further details see Table 1).

Groups were matched for gender, age, and education (all $p$-values $>0.21$). BPD patients were significantly more depressed ($t_{32}=9.63$, $p<0.001$) and impulsive (Mot: $t_{32}=3.03$, $p=0.005$; Cog: $t_{32}=5.13$, $p<0.001$; NP: $t_{32}=2.21$, $p=0.03$) than control subjects.

After complete description of the study to the subjects, written informed consent was obtained. The study was approved by the local ethical committee and was conducted in accordance with the Declaration of Helsinki.

### *Measures*

Participants performed a hybrid flanker-Go/Nogo paradigm while a 64-channel EEG was recorded. Eight different letter strings (congruent: BBBBB, DDDDD, VVVVV, and UUUUU; incongruent: BBDBB, DDBDD, UUVUU, and VVUVV) were presented on a computer screen in randomized order. Subjects had to focus on the target letter in the middle of an array and had to press a right response key upon appearance of letters B or U (Go condition) and to withhold key press upon appearance of D or V (Nogo condition). The whole experiment consisted of 5 blocks with 120 trials each. The number of Go and Nogo trials was balanced (each trial type was presented 300 times). The four incongruent letter strings BBDBB, DDBDD, UUVUU, and VVUVV were presented 120 times each, whereas the four congruent letter strings BBBBB, DDDDD, VVVVV, and UUUUU were presented 30 times, each with a presentation time of 400 ms. The data were also used for analyzing errors in our previous study (Ruchsow et al. 2006). Subjects got feedback according to their performance 750 msec after key press. As feedback stimuli we used the German expressions for ''correct'' and ''false''. Severe time pressure was administered by asking the subjects to respond within a RT deadline. If subjects missed the RT deadline the feedback ''faster'' was presented. Feedback stimuli were presented for another 500 msec. The intertrial-interval was 2600 msec. Participants got a monetary reward, winning or losing a small amount of money each trial (five Euro-cent). However, instruction emphasized speed over accuracy. Before recording the EEG, subjects had a training period of 120–240 trials. RT deadlines were calculated individually by the mean RT of the training period minus 10 percent. Time windows ranged between 250 and 400 msec. Participants were seated in a comfortable chair in a sound-attenuating, electrically shielded booth. The whole experiment lasted about 2.5 h, including electrode placement, breaks and electrode removal.

Table 1. *Demographic parameters, sum scores of psychometric ratings, and medication (in mg) in patients with borderline personality disorder (BPD) and healthy controls*

| | Patients with BPD | Control subjects |
|---|---|---|
| N | 17 | 17 |
| Gender (f/m) | 16/1 | 15/2 |
| Age | 27.4 ± 6.7 | 27.8 ± 9.5 |
| Years of education | 10.9 ± 1.5 | 11.7 ± 2.0 |
| TCA | 159.5 ± 177.9 | 0 |
| CE | 35.6 ± 73.3 | 0 |
| Benz | 1.0 ± 3.3 | 0 |
| BIS-10; Mot | 26.8 ± 5.2 | 21.7 ± 4.5 |
| BIS-10; Cog | 30.4 ± 4.5 | 23.0 ± 3.9 |
| BIS-10; NP | 26.8 ± 5.7 | 23.0 ± 4.3 |
| BDI | 27.3 ± 9.8 | 3.7 ± 2.5 |

*TCA* Tricyclic antidepressants (reference substance: imipramine); *CE* chlorpromazine equivalents; *Benz* benzodiazepines (reference substance: diazepam); *BIS-10* Barratt Impulsiveness Scale, version 10; *Mot* motor impulsiveness sub-trait; *Cog* cognitive impulsiveness sub-trait; *NP* non-planning sub-trait; *BDI* Beck depression inventory.

EEG was recorded using the Easy-cap® system. Electrodes were positioned with equal distances. All electrodes were referenced to an electrode between Cz and FCz and re-referenced to average reference off-line. Eye movements were registered by vertical and horizontal EOG. Electrode impedances were measured at a standard frequency of 30 Hz and kept below 5 kΩ. The EEG was amplified by Neuroscan amplifiers (bandwidth DC-50 Hz; 50 Hz notch filter) and A/D converted with 12-bit resolution at a rate of 250 Hz and digitally low-pass filtered with 16 Hz and digitally high-pass filtered with 0.10 Hz. The EEG was baseline corrected to an interval between −150 msec and 0 msec before the onset of the stimuli (letter strings). Ocular artifacts were corrected by using a method proposed by Gratton et al. (1983). Stimulus-locked EEG-segments of 1000 msec were used in order to compute ERPs to correct Go and correct Nogo trials, separately.

For statistical analysis, electrodes were selected from nine different regions: left frontal (AF3, AF7, F3, F5), midline frontal (AFz, Fz), right frontal (AF4, AF8, F4, F6), left central (FC1, FC3, C1, C3), midline central (FCz, Cz), right central (FC2, FC4, C2, C4), left parietal (CP1, CP3, P1, P3), midline parietal (CPz, Pz), and right parietal (CP2, CP4, P2, P4).

Components of interest were the N2 and the P3 component. As the N2 was constrained to central scalp locations this ERP component was evaluated solely at these regions (please refer to: Holroyd 2004) whereas the P3 was analyzed at all nine regions (frontal, central, and parietal).

At frontal electrode sites P3 amplitudes were found to be polarity inverted as has been described in previous studies (Kiefer et al. 1998; Kaiser et al. 2003).

Analyzes were based on non-subtracted ERP data (''raw'' waves) of correct Go trials (button press) and correct Nogo trials (no button press). In order to determine relevant time windows peak analyses were performed for Nogo-N2 and Nogo-P3, separately. Nogo-N2 peaked at 246.2 ± 26.5 msec (Cz), Nogo-P3 peaked at 468.1 ± 87.5 msec (Fz), 392.4 ± 52.9 msec (Cz), and 430.8 ± 71.8 msec (Pz). In order to determine respective time-windows an ANOVA with repeated measures on ''region'' (4 levels: central N2, frontal P3, central P3, parietal P3) and ''group'' (2 levels: patients, controls) was performed which revealed a significant region effect ($F_{3,96}=81.60$, $p<0.001$). Post-hoc tests showed significant differences between all four regions (all $p$-values $<0.02$). Thus, the following time windows were selected for statistical analysis: 180–320 msec (central N2), 400–540 msec (frontal P3), 320–460 msec (central P3), and 360–500 msec (parietal P3).

For each time window an ANOVA with repeated measures on ''region'' (3 levels: right, midline, and left) and ''condition'' (2 levels: Nogo, Go) as within-subjects factors, and ''group'' (2 levels) as between-subjects factors were computed, while averaging across electrode positions within each of the different regions.

Significant region by condition by group interactions were analyzed by means of a different set of within regions ANOVAs on condition by group interactions, and further evaluated with Fisher LSD post-hoc tests (nominal level of $p < 0.05$).

For the sake of brevity, only effects involving "group" are reported.

Correlational analyses were performed in order to assess relationships between ERP components, BIS-10, BDI, age, and medication.

## Results

### *Behavioural data*

Given the task, correct responses on Go and Nogo trials were of interest. Mean numbers of analyzable EEG segments were $135.5 \pm 60.1$ (Go) and $200.1 \pm 75.5$ (Nogo) for healthy controls and $122.4 \pm 47.9$ (Go) and $160.5 \pm 77.3$ (Nogo) for BPD patients, respectively. An ANOVA with repeated measures on "condition" (Nogo, Go) and "group" (patients, controls) revealed neither a condition effect ($F_{1,32} = 2.39$, $p = 0.13$) nor a condition by group interaction ($F_{1,32} = 0.77$, $p = 0.39$).

In a second step groups were compared for error rates (incorrect Nogo trials: errors of commission) and reaction times (RTs) for correct Go trials and incorrect Nogo trials, separately. For healthy controls mean number of errors was $54.3 \pm 46.0$ corresponding to an error rate of 9.1%. BPD patients demonstrated a mean number of $72.7 \pm 66.9$ errors corresponding to an error rate of 12.1%. Error rates did not significantly differ between groups ($t_{32} = 0.93$, $p = 0.36$).

Mean RT was $230.3 \pm 27.6$ msec for correct Go trials and $226.8 \pm 34.2$ msec for incorrect Nogo trials in healthy subjects. In the patient group mean RTs were $221.9 \pm 36.2$ msec for correct Go trials and $216.5 \pm 36.8$ msec for incorrect Nogo trials. We performed an ANOVA for RTs with repeated measures on "condition" (2 levels) and "group" (2 levels) which revealed no significant between group differences (all $p$-values $> 0.37$).

### *Event-related potentials*

With regard to the central N2 there was neither a group effect ($F_{1,32} = 0.70$, $p < 0.41$) nor a condition by group interaction ($F_{1,32} = 0.27$, $p < 0.60$).

For the central P3 a significant condition by group ($F_{1,32} = 4.94$, $p < 0.03$) and a significant region by condition by group interaction ($F_{2,64} = 3.85$, $p < 0.03$) could be demonstrated. The triple interaction was further evaluated by within-region analyses of condition by group interactions which showed a significant interaction for left central ($F_{1,32} = 5.03$, $p = 0.03$) and midline central electrodes ($F_{1,32} = 5.26$, $p = 0.03$). For right central electrodes there was no significant condition by group interaction ($F_{1,32} = 2.21$, $p = 0.15$). Post-hoc tests revealed that the interactions were due to smaller Nogo-P3s in BPD patients than in controls (left central: $p < 0.005$; midline central: $p < 0.04$), whereas Go-P3s did not differ between groups (left central: $p = 0.50$; midline central: $p = 0.61$).

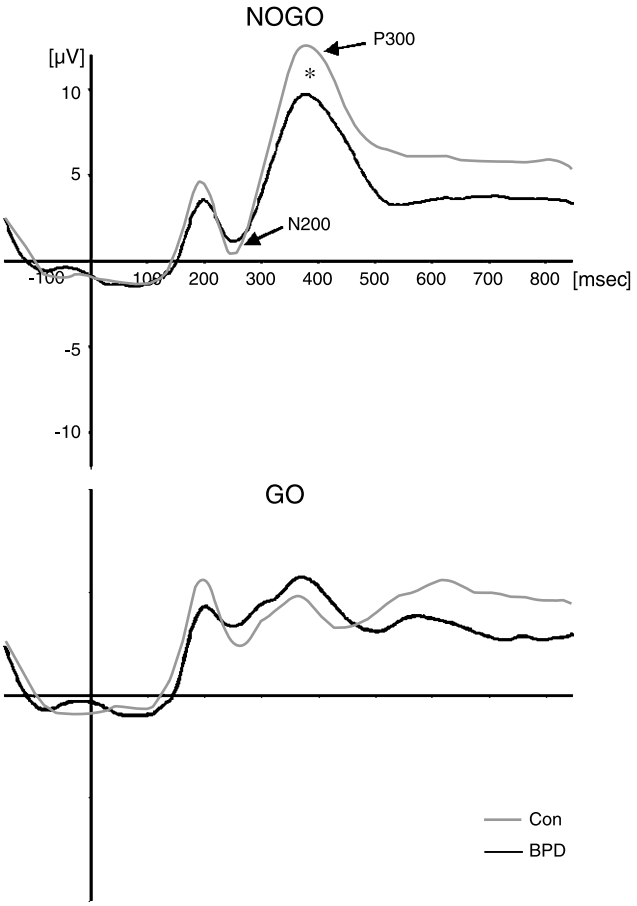


**Fig. 1.** Grand-averaged waveforms for patients with borderline personality disorder (BPD; *black*) and healthy controls (*grey*) at Cz. Nogo trials and Go trials are depicted separately. Potentials were collapsed across electrode positions within scalp regions

At frontal electrode sites we found a significant region by group interaction ($F_{2,64} = 4.23$, $p = 0.02$), which was due to regional differences (all $p$-values $< 0.01$) but not due to group differences (all $p$-values $> 0.29$) in post-hoc analyses (data not shown).

At parietal electrodes a significant between group effect ($F_{1,32} = 5.71$, $p = 0.02$) and a condition effect was detectable ($F_{1,32} = 61.30$; $p < 0.001$), but no interaction condition by group ($F_{1,32} = 0.98$; $p = 0.33$). The parietal P3 was overall more negative in the BPD group than in the control group, regardless of type of response, and Go-P3 ampli-

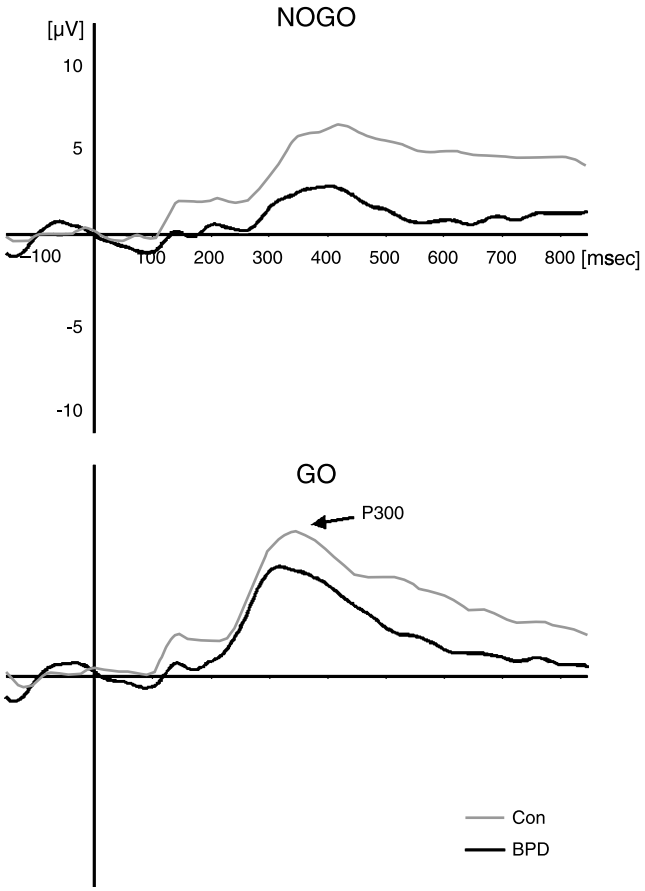


**Fig. 2.** Grand-averaged waveforms for patients with borderline personality disorder (BPD; *black*) and healthy controls (*grey*) at Pz. Nogo trials and Go trials are depicted separately. Potentials were collapsed across electrode positions within scalp regions

tudes were overall more positive than Nogo-P3 amplitudes for both groups.

### *Correlational findings*

We tested for correlations between left central and midline central Nogo-P3 amplitudes, psychometric data (BIS-10, BDI), age, and education.

We found a negative correlation between the Barratt Impulsiveness Scale, cognitive impulsiveness sub-trait and left central Nogo-P3 amplitudes for the entire group ($r = -0.41$; $p = 0.02$) but not for the two subgroups, separately (all $p$-values $> 0.27$).

Moreover, a negative correlation between the BDI and left central Nogo-P3 amplitudes could be demonstrated for the entire group ($r = -0.42$; $p = 0.01$) but not for subgroups, separately (all $p$-values $> 0.30$).

There were no significant correlations between Nogo-P3 amplitudes and age or medication (all $p$-values $> 0.12$).

## Discussion

In the present study we used a hybrid flanker-Go/Nogo paradigm in order to investigate Nogo-N2 and Nogo-P3 in BPD patients and control subjects without history of Axis I or II psychopathology. Most interestingly, in line with our prediction central Nogo-P3 amplitudes were found to differ significantly between groups: patients with BPD had reduced (''less positive'') Nogo-P3 amplitudes compared to controls. Importantly, this reduction of the P3 in patients at central electrode locations was specific for Nogo trials and absent in Go trials. Moreover, for the entire group ($n = 34$) there was a significant negative correlation between the cognitive impulsiveness sub-trait of the Barratt Impulsiveness Scale (BIS-10) and central Nogo-P3 amplitudes: the lower the amplitude of the central Nogo-P3 the higher cognitive impulsiveness. In addition, we found a negative correlation between Beck's depression inventory and central Nogo-P3 amplitudes. According to this consideration, Nogo-P3 amplitudes were reduced, because BPD patients were more severe depressed than controls. It is beyond the scope of the present paper to decide between these two alternatives which should be addressed by further ERP studies on patients with major depression.

With respect to error rates and reaction times there were no significant between group differences indicating comparable levels of performance in both groups. Consequently, ERP differences are most likely not due to differences in behavioural data.

Salisbury et al. (2004) suggested that the P3 reflects merely the absence of negative motor potentials in Nogo trials. Following this hypothesis, the P3 in Nogo trials is the true P3 which has a central maximum and which is only distorted in Go trials. With present data, this hypothesis seems unlikely. Our findings show, that the reduction of central Nogo-P3 amplitudes in BPD patients cannot be due to motor potentials because these are absent in both groups. If this reduction of the Nogo-P3 in the patients would merely reflect an unspecific reduction of the true P3, this reduction should be likewise visible for Go trials at central electrodes which is not the case. To explain the data pattern with the motor hypothesis one would have to assume that motor potentials are smaller in BPD compared to controls which would hence compensate for the reduction of the true P3 in the Go trials in BPD. This is highly unlikely. Hence the present data firstly show that there is a specific attenuation of the Nogo-P3 in Nogo trials, and that the motor hypothesis of the Nogo-P3 cannot easily explain our data. In summary, our findings support the view that the Nogo-P3 is related to response inhibition (Herrmann et al.

2003; Bruin and Wijers 2002; Burle et al. 2004), which is impaired in patients with BPD.

With regard to the Nogo-N2 component we could not find any between group differences. Consistent with our prediction these results might suggest that the Nogo-N2 has to be discussed in the context of response conflict rather than response inhibition. Interestingly, in a previous study with OCD patients using exactly the same Go/Nogo paradigm we found the opposite ERP pattern (Ruchsow et al. in press): OCD patients differed from controls with respect to Nogo-N2 amplitudes (OCD patients > controls) but not Nogo-P3 amplitudes. As the number of incongruent trials (and thereby the degree of response conflict) was the same in the present study and our previous (OCD) study one would expect identical ERP patterns in both studies.

Furthermore, present data shed some light on the ongoing discussion whether the N2 and the error (related) negativity reflect the same underlying process. The error negativity (Ne; Falkenstein et al. 1990) or error-related negativity (ERN; Gehring et al. 1990) is a negative deflection peaking between 100 and 150 msec after the onset of an erroneous response in various tasks like the Eriksen flanker paradigm and the Go/Nogo task (Scheffers et al. 1996). There was much controversy on the relationship of the Ne/ERN and the Nogo-N2 (e.g. Holroyd 2004). It has been suggested that these two ERP components reflect the same process, namely conflict processing (Yeung et al. 2004). The Ne/ERN was consistently found to be reduced in BPD patients compared to controls (de Bruijn et al. 2006; Ruchsow et al. 2006). In contrast, our present results, which are based on the same task as in our previous study (Ruchsow et al. 2006), show no reduction of N2 amplitudes in BPD patients compared to controls. This discrepancy suggests that Ne/ERN and Nogo-N2 do not mirror the same underlying process. Interestingly, using the same paradigm in patients with obsessive-compulsive disorder (OCD) we found an enhancement of both the Ne/ERN (Ruchsow et al. 2005) and the N2 (Ruchsow et al. in press), which would suggest the same underlying generator for the two ERP components. Unfortunately, the findings of the present study and our previous OCD studies (Ruchsow et al. 2005; Ruchsow et al. in press) cannot be compared directly, as control groups were different. Possibly, the psychopathological dimensions of impulsivity and compulsivity affect Nogo-N2 and Ne/ERN in a different manner. For example, N2 and Ne/ERN may contain a common component which is strongly affected in OCD, while in BPD only an Ne/ERN-specific subcomponent is affected. It seems reasonable to confirm this assumption by further ERP studies exploring other disorders of the impulsivity-compulsivity spectrum (Skodol and Oldham 1996).

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