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Electrophysiological correlates of error processing in borderline personality disorder

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Abstract

The electrophysiological correlates of error processing were investigated in patients with borderline personality disorder (BPD) using event-related potentials (ERP). Twelve patients with BPD and 12 healthy controls were additionally rated with the Barratt impulsiveness scale (BIS-10). Participants performed a Go/Nogo task while a 64 channel EEG was recorded. Three ERP components were of special interest: error-related negativity (ERN)/error negativity (Ne), early error positivity (early Pe) reflecting automatic error processing, and the late Pe component which is thought to mirror the awareness of erroneous responses. We found smaller amplitudes of the ERN/Ne in patients with BPD compared to controls. Moreover, significant correlations with the BIS-10 non-planning sub-score could be demonstrated for both the entire group and the patient group. No between-group differences were observed for the early and late Pe components. ERP measures appear to be a suitable tool to study clinical time courses in BPD.

Keywords: Evoked potentials (cognitive); Electrophysiology; Borderline personality disorder (BPD)

1. Introduction

Borderline personality disorder is characterized by a variety of symptoms like a pervasive pattern of instability of interpersonal relationships, self-image, and affects, recurrent suicidal behavior, affective instability and chronic feelings of emptiness (APA, 1994). Among these, increased impulsiveness has received special attention as one of the core symptoms of this disorder. There is still an ongoing debate on the neurobiological and neuropsychological mechanisms underlying BPD. Several positron emission tomography (PET) studies reported a hypometabolism in prefrontal cortical areas (De La Fuente et al., 1997; Soloff et al., 2003) which reflects either a diminished serotonergic turnover (Hansenne et al., 2002) and/or dopamine dysfunction (Friedel, 2004), and/or an interaction between these resulting in emotional dysregulation and impulsive behavior. In a magnetic resonance imaging study, Tebartz van Elst et al. (2003) found reduced volumes of the hippocampus, amygdala, and orbitofrontal, dorsolateral prefrontal, and anterior cingulate cortex in BPD patients. Moreover, BPD patients often suffer from impairments in decision-making and planning again pointing to deficits in a circuitry encompassing the frontal lobes (Bazanis et al., 2002).

According to Barratt (1985) impulsiveness 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’’). The Barratt impulsiveness scale, Version 10 (BIS-10) has been administered to psychiatric inpatients and could clearly differentiate antisocial and borderline personality disorder from other diagnostic categories, like major depression and schizophrenia (Barratt, 1985). The BIS-10 has also consistently been rated as a reliable and

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valid instrument for measuring impulsiveness in patients and normal controls (Patton et al., 1995; Bayle et al., 2000; Someya et al., 2001; Preuss et al., 2003; Spinella, 2004).

As cognitive processing related to impulsiveness is fast by definition, the high time resolution of event-related potentials (ERP) in the range of milliseconds makes them an appropriate tool to investigate a time sensitive phenomenon like this. In this context, recent ERP-studies proved their suitability in that they showed that the error negativity (Ne; Falkenstein et al., 1990) or error-related negativity (ERN; Gehring et al., 1990) mirrors impulsive responding, e.g. in an Eriksen flanker task (Dikman and Allen, 2000; Luu et al., 2000; Pailing et al., 2002). So in the present study we made use of ERP potentials to investigate error-processing in BPD patients, to test whether there are deviations from controls, and whether impulsivity may play a role mediating differences between patients and controls.

The ERN/Ne is a negative going ERP component peaking between 100 and 150 ms after the onset of electromyographic (EMG) activity associated with an erroneous response (Scheffers et al., 1996) in forced choice reaction time paradigms like the Eriksen flanker task or Go/Nogo tasks (Falkenstein et al., 1999). As the present study uses button presses (instead of EMG activity) in order to define an error, the ERN/Ne is expected to be found within 100 ms after an erroneous button press. A source analysis of the ERN/Ne scalp potential with brain electric source analysis (BESA; Scherg and Berg, 1990) pointed to neural generators in medial prefrontal areas, best to correspond to the anterior cingulate cortex (ACC; Dehaene et al., 1994; Ruchsow et al., 2002). The involvement of ACC in error processing has been confirmed by several fMRI studies (e.g. Carter et al., 1998). The ERN/Ne was originally interpreted as an error detection signal resulting from a mismatch between the representation of the correct response and the representation of the actual (false) response (Falkenstein et al., 1990; Gehring et al., 1993). Alternative accounts view the ERN/Ne as a brain potential reflecting the response evaluation process itself rather than the outcome of this process (Vidal et al., 2000). Rather contrary to these interpretations, Cohen and coworkers interpret the ERN/Ne to be associated with the detection of response conflict (Botvinick et al., 2001; Carter et al., 1998).

More recently, Holroyd and Coles (2002) proposed a theory combining elements of mismatch theory and reinforcement learning principles. According to their suggestion the ERN/Ne components are generated when a negative reinforcement learning signal is conveyed to the ACC via the mesencephalic dopamine system. This signal is used by the ACC to modify performance on the task at hand.

During an Eriksen flanker task, Luu et al. (2000) found large ERN/Ne amplitudes at the beginning of the session in college students who were high on negative affect (NA) and negative emotionality (NEM). Moreover, a shift of response patterns was found during the time course of the experiment. By means of a post-task questionnaire part of the subjects were reported to have been bored and dissatisfied with their performance resulting in motivational problems and disengagement from the task. When EEG data were re-analyzed for members of the high-NA and high-NEM groups with motivational problems the amplitude of the ERN/Ne decreased with the time-course of the entire experiment. This pattern of results was strikingly different from results of participants who were low on NA and NEM and showed unaltered ERN/Ne amplitudes throughout the whole experiment. Similarly, Dikman and Allen (2000) demonstrated that individuals low on socialization exhibit smaller ERN/Ne amplitudes during tasks which penalize error responses. In the same vein, Pailing et al. (2002) found smaller ERN/Ne peak amplitudes and higher error rates in subjects with a tendency towards impulsive responding (i.e. with a less controlled response strategy). They defined impulsiveness based on a linear regression from correct individual reaction times (RTs) on reaction times from erroneous responses. Mean residual scores were defined as mean difference of observed RTs minus predicted RTs for error trials. Less negative mean residual RTs were regarded as indicating a more cautious (controlled) response strategy whereas more negative residuals were interpreted to indicate a less controlled (i.e. more impulsive) response style. Furthermore, ERN/Ne latencies were positively related with percentage of errors suggesting that individuals with faster ERN/Ne components should have more opportunity to catch an erroneous intention before fully committing the response than individuals with slower ERN/Ne components (Pailing et al., 2002). On the opposite side of a possible impulsivity–compulsivity dimension, Gehring et al. (2000) have reported enhanced ERN/Nes in patients with obsessive–compulsive disorder. In these patients ERN/Ne amplitude was positively related with symptom severity. In contrast to these documented relations between impulsivity/compulsivity and ERN/Ne amplitude, Luu et al. (2000) did not find correlations between ERN/Ne amplitudes and measures of impulsivity in a sample of healthy controls. Perhaps, these discrepant findings are due to different psychometric measures of impulsivity and subject populations in these studies.

Another ERP component discussed in the context of error processing is the error positivity (Pe). The Pe is a slow positive wave with centro-parietal distribution which often follows the ERN/Ne in a time window between 300 and 500 ms after erroneous responses. The Pe can clearly be differentiated in time from the P300 (Falkenstein et al., 2000). A source localization analysis, using BESA revealed that the Pe consists of two components: an early Pe component with probable generators in the area of the caudal ACC and a late Pe component with probable generators in the area of the rostral ACC (Van Veen and Carter, 2002). The late Pe component was associated with awareness of erroneous responses, since it was more pronounced for perceived than for unperceived errors (Nieuwenhuis et al., 2001).

In the present study we expected that error monitoring processes as expressed in the ERN/Ne and Pe components differ between BPD patients and controls. Additionally, we tested whether impulsiveness in BPD patients might have an impact on the ERN/Ne amplitudes.