Neural correlates of impulsive responding in borderline personality disorder: ERP evidence for reduced action monitoring

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Abstract

Patients with borderline personality disorder (BPD) are characterized by marked impulsive behaviour. The impulsive response style of patients with BPD may be associated with diminished action monitoring, which can be investigated by measuring the error-related negativity (ERN). The ERN is an ERP component generated in the anterior cingulate cortex (ACC) following erroneous responses. Behavioural and ERP measurements were obtained during performance on a speeded two-choice reaction task in a group of patients with BPD (N = 12) and in a group of age-matched controls (N = 12). The ERP results showed that ERN amplitudes were reduced for patients with BPD, as were the P300 amplitudes after late feedback. The behavioural results confirmed a more impulsive response style for the BPD group, reflected in larger RT differences between correct and incorrect responses and in an increase in erroneous responses to the easy congruent stimuli. Additionally, analyses on post-error congruency effects demonstrated that controls adjusted their behaviour following errors, but patients with BPD did not. The attenuated ERNs indicate reduced action monitoring in patients with BPD. This suggests that the ACC, or the action-monitoring network it is part of, is not functioning optimally. Due to this reduced action monitoring, patients with BPD do not learn from their errors as well as controls. Consequently, they do not adjust their behaviour when necessary and thus maintain their impulsive response style.

1. Introduction

Marked impulsivity is seen as one of the main characteristics of borderline personality disorder (BPD), together with rapidly changing mood states, aggressive behaviour, instability of interpersonal relationships, self-image, and affects. Impulsive behaviour may express itself in promiscuity, substance abuse, adverse financial behaviour, reckless driving, and binge eating. Suicidal behaviour and self-mutilation are also frequently related to impulsivity (American Psychiatric Association, 2000).

Although impulsiveness is an important clinical feature, the number of studies investigating its neural correlates is still relatively small. This may be explained by the lack of a unified definition, which is due to the wide range of behaviours in which the trait is present (see e.g. Eysenck, 1999). Moeller et al. (2001), in their review of the psychiatric aspects of impulsivity, concluded that a reliable definition should at least include the elements of the various behavioural models that have been developed based on findings from targeted laboratory tasks. These
elements are: (1) rapid, unplanned reactions to stimuli before complete processing of information, (2) decreased sensitivity to negative consequences of behaviour, and (3) lack of regard for long-term consequences. With the current study we aimed at investigating the neural correlates of the first two elements in patients with BPD.

Rapid, unplanned reactions and diminished sensitivity to resulting erroneous responses can be studied by means of electrophysiological measurements during a speeded forced-choice task. Especially the discovery of an event-related potential (ERP) component associated with error or conflict detection has given this type of action monitoring research an important impetus. This so-called error negativity (Falkenstein et al., 1991) or error-related negativity (ERN; Gehring et al., 1993) is characterized by a sharp negative deflection over fronto-centrally located electrodes appearing within 100 ms after an error has been made.

Source localization and fMRI studies have found the anterior cingulate cortex (ACC) as the most likely generator of the ERN (see e.g., Dehaene et al., 1994; Kiehl et al., 2000; Ullsperger and von Cramon, 2001), a finding that is in line with earlier studies demonstrating error-related activity in unit recordings from the ACC in monkeys (see e.g., Niki and Watanabe, 1979). The ACC is a mediofrontal brain structure known for its rich innervation from and to other regions of the brain and its rich concentration of different types of neurotransmitters like dopamine. The area is highly interconnected to the motor system, the limbic system, and to prefrontal regions. Because of these characteristics, the ACC has been described as the interface between cognition, motor control, and the drive of the organism (Paus, 2001).

Originally, the ERN was taken to be elicited by a mismatch, i.e., after the error detection system has failed to match a representation of the actual behaviour with a representation of the desired behaviour (see e.g. Falkenstein et al., 1991; Gehring et al., 1993). More recently, Holroyd and Coles (2002) extended this original interpretation in their so-called reinforcement-learning theory of the ERN. According to the theory, predictive error signals indicating whether events turn out to be worse than expected are carried to various brain areas by the dopamine system. These error signals are used to improve performance in order to prevent future errors. When a predictive error signal arrives at the ACC, the ERN is elicited. Alternative accounts refer to the ERN as the reflection of conflict that arises when two incompatible response tendencies are simultaneously activated (Botvinick et al., 2001; Cohen et al., 2000; Yeung et al., 2004). For the current study it is relevant that all three accounts agree that the ERN is generated in the ACC, that it reflects the outcome of an action-monitoring process, and that it is used to optimize performance in the future.

Differences in ERN amplitude have been observed in a variety of personality traits. Individuals low on socialization tend to exhibit smaller ERN amplitudes (Dikman and Allen, 2000), whereas individuals with greater negative affect (Luu et al., 2000) or those with obsessive-compulsive personality traits (Hajcak and Simons, 2002) show larger ERN amplitudes. Pailing et al. (2002) specifically investigated the relation between the ERN and impulsivity. In their study, subjects with large reaction-time differences between correct and incorrect responses had smaller ERN amplitudes. Because (overly) fast reaction times generally lead to more erroneous responses, these larger reaction-time differences were taken to reflect a more impulsive response style. This motivated us to investigate whether this line of reasoning could also be applied to patients with BPD.

PET and fMRI studies investigating neuropsychiatric disorders have demonstrated increased ACC activity in individuals with obsessive-compulsive disorder (OCD; see e.g., Adler et al., 2000; Ursu et al., 2003) and decreased ACC activity in patients with schizophrenia (Carter et al., 2001; Laurens et al., 2003). These differences in ACC activity were also reflected in action monitoring: enhanced ERN amplitudes were found in individuals with OCD (Gehring et al., 2000), whereas decreased ERN amplitudes were observed in patients with schizophrenia (Alain et al., 2002; Bates et al., 2002; Kopp and Rist, 1999; Mathalon et al., 2002).

With regard to BPD, a number of brain imaging studies examining patients have shown hypometabolism in prefrontal cortical areas (see e.g., De la Fuente et al., 1997; Goyer et al., 1994; Soloff et al., 2003). Recently, Tebartz van Elst et al. (2003) demonstrated volume loss of the right ACC in their BPD sample. The authors suggested that specifically this volume loss might differentiate BPD from other neuropsychiatric disorders.

In the present and to our knowledge the first such study in patients with BPD, we employed a speeded two-choice task while measuring ERN amplitudes. We predicted that patients with BPD would show increased impulsivity in different behavioural measures and reduced action monitoring as evidenced by smaller ERN amplitudes.

In order to examine the entire process of action monitoring from stimulus onset to feedback processing, we also examined two other ERP components known to be involved in action monitoring, namely the stimulus-locked N2 and the feedback-locked P300. The amplitude of the N2 is thought to reflect the monitoring of response conflict that arises from simultaneously active response tendencies as it is enlarged after incongruent stimuli compared to congruent ones (see e.g. Yeung et al., 2004). Consequently, the N2 is a reflection of a relatively early process. As we specifically anticipated group differences for the later processes directly related to erroneous responses, we did not expect to find any
2. Methods and materials

2.1. Participants

Twelve female patients (mean age = 29.3, SD = 4.5 years) with primary diagnosis BPD (according to DSM-IV) were recruited from two Dutch (inpatient and outpatient) psychiatry departments. An independent and trained physician confirmed the diagnostic criteria for BPD by use of the structural clinical interview for DSM-IV Axis II Personality Disorders (SCID-II; First et al., 1997). An extra clinical screening was performed to exclude current (comorbid) axis-I mood and psychotic disorders (except for dysthymic disorder).

Other exclusion criteria were neurological, ophthalmologic and vestibular disorders, as well as comorbidity with alcohol or substance dependence. Six patients used a selective serotonin reuptake inhibitor (SSRI), of whom one in combination with clozapine and one with cisaprid, and one patient used quetiapine only. The remaining five patients did not take any psychopharmacca. In the week prior to testing, the use of cannabis or any other illicit drug, or alcohol consumption exceeding more than 3 units per day, were further reasons for exclusion. In addition, all subjects were instructed to abstain from coffee and nicotine 24 h prior to testing. Note that on the day of the experiment, none of the subjects had used any psychotropic drug.

Twelve age-matched, healthy female controls (mean age = 26.0, SD = 6.8 years) were recruited by means of advertisements in a local paper. They had no prior history of psychiatric or neurological illness. In addition, they did not take any medication and adhered to the criteria set for alcohol and drug intake.

All participants gave written and informed consent and received financial compensation for their participation. The study was carried out in accordance with the latest version of the Declaration of Helsinki and was approved by the University Medical Centre’s ethical committee.

2.2. Task and procedure

Participants performed a speeded two-choice reaction task, the so-called ‘flankers’ task (Eriksen and Eriksen, 1974). Visual stimuli in this task are letter strings of five letters. The goal for participants is to respond as fast as possible to the central letter of the target string by pushing a button with their left or right index finger while ignoring the surrounding or flanking letters. The stimulus–response mappings are known to the subjects beforehand. Instructions may, for example, state that a left-hand response is required when the H is the central letter and a right-hand response when the central letter of the target string is an S. Four strings of letters are presented, two with five identical letters (HHHHH and SSSSS) and two including deviating flanking letters (SSHSS and HSHHH). When congruent stimuli are presented, i.e. where the flankers are identical to the central target letter, responding is relatively easy, as all visual information primes the correct response. In the case of different flankers surrounding the central target, the incongruent stimuli, responding is more difficult, as the visual information strongly induces the wrong response. Stimulus–response mappings were counterbalanced in both groups and equal emphasis was placed on speed and accuracy in the written and verbal task instructions.

Because previous ERN studies had demonstrated that accuracy could affect ERN amplitude (see e.g. Gehring et al., 1993), we first calculated individual reaction-time (RT) deadlines. This procedure, which had already been employed successfully in previous studies (see e.g., De Bruijn et al., 2004a), ensures similar performance levels for the two groups. The personal maximum RT sets the time-frame within which a subject should respond to avoid visual feedback indicating that the response was late. To this end, the subjects first performed a practice block of 60 trials. During the practice block, the initial RT deadline was set to a relatively liberal limit of 800 ms. After completion of the 60 practice trials, the participant’s average RTs and standard deviations (SDs) of the correct responses were computed. Subsequently, the RT deadline for each individual participant was determined by adding 0.5 SD to this mean RT.

The experimental phase consisted of five blocks of 100 trials each, i.e. 50 congruent and 50 incongruent stimuli, with a self-paced pause halfway each block. Subjects were first presented with a fixation point (lasting 100 ms). After 300 ms the stimulus (also lasting 100 ms) would appear. The next 900 ms the screen remained blank, after which the visual feedback stimulus (1000 ms) appeared. The visual feedback consisted of a yellow, a blue, or a red rectangle indicating whether the response had been correct, incorrect, or late, respectively. Responses were considered late when RTs exceeded the assigned deadline. The next trial was presented after an inter-trial interval of 100 ms. The total duration of the experimental phase was 35 min including breaks.
2.3. EEG recording

The electroencephalogram was recorded from four midline electrodes (Fz, FCz, Cz, Pz) mounted in an elastic electrode cap (Electrocap international). Electrodes were placed at locations in accordance with the international 10–20 system. All electrodes were referenced to the left mastoid, but were later off-line re-referenced to the average of the left and right mastoid. The vertical electro-oculogram (EOG) was recorded bipolarly from electrodes placed above and below the right eye. The horizontal EOG was also recorded bipolarly from electrodes lateral to both eyes. Electrode impedances were kept below 10 kΩ. The EEG and EOG signals were amplified using a time-constant of 8 s and were filtered off-line low-pass at 15 Hz. All signals were digitized with a sampling rate of 200 Hz.

2.4. Analyses

EOG artifact correction was carried out using the procedure proposed by Gratton et al. (1983). For both behavioural and ERP analyses all responses with reaction times faster than 150 ms (1.1%) were removed from the data sets. ERPs for correct and incorrect responses to incongruent stimuli were averaged separately off-line time-locked to response onset, starting 200 ms before and ending 500 ms after response onset relative to a 200 ms pre-response baseline. Incorrect responses to congruent stimuli were not analyzed as the number of trials was too small to obtain reliable ERPs. In addition, correct responses were averaged separately for congruent and incongruent stimuli time-locked to stimulus onset relative to a 200 ms pre-stimulus baseline. Finally, ERPs time-locked to feedback onset were averaged separately for feedback type time-locked to feedback onset, starting 200 ms before and ending 800 ms after feedback onset, also relative to a 200 ms pre-stimulus baseline.

ERN amplitude was determined on incorrect trials by subtracting the most negative peak in the 0–200 ms time-window after response onset from the most positive peak in the time-window starting 80 ms before and ending 80 ms after response onset at electrodes FCz and Cz, where maximal ERN amplitudes were expected.

N2 amplitude was determined on correct stimulus-locked ERPs as the difference between the most negative peak in the 200–350 ms time-window and its preceding positive peak at electrode FCz. P300 amplitude was defined as the most positive peak in the 300–800 ms time-window after feedback onset at electrode Pz.

Individual averages for RTs, amplitudes, and number of responses were entered in a repeated measures General Linear Model (GLM) with group (2 levels: BPD vs. control) as between-subject factor. Possible within-subject factors of the different GLMs were congruency (2 levels: congruent vs. incongruent), correctness (2 levels: correct vs. incorrect), feedback type (3 levels: correct feedback vs. incorrect feedback vs. late feedback), and post-correctness (2 levels: post-correct vs. post-error). The analyses on ERN amplitude also included the within-subject factor electrode (2 levels: FCz vs. Cz). Greenhouse–Geisser corrections were applied when appropriate, but uncorrected degrees of freedom values are given for ease of interpretation. Note that significant ERP outcomes were additionally analyzed for the medication-free subset of patients (N = 5) only, in order to exclude any possible effects that medication might have on the ERP measurements.

3. Results

3.1. Behavioural analyses

Fig. 1 shows the mean RTs of both groups. With regard to correct responses, the group RTs differed, with the BPD group overall responding more slowly (382 ms) than the control group [347 ms; \(F(1, 22) = 5.97, p = 0.023\)]. Usually, responses to congruent stimuli are given faster than responses to incongruent stimuli. This congruency effect was also present in the current data, as the RTs for correct responses were shorter following congruent (349 ms) than following incongruent stimuli [380 ms; \(F(1, 22) = 130.29, p < 0.001\)]. The interaction between congruency and group was not significant (\(F < 1\)).

As in similar studies, incorrect responses were faster (322 ms) than correct responses [365 ms; \(F(1, 22) = 96.26, p < 0.001\)]. However, the interaction with group revealed that this response effect was larger for the BPD group (56 ms) than for the control group [28 ms; \(F(1, 22) = 10.78, p = 0.003\)].
3.2. ERP analyses

3.2.1. Response-related ERN

As Fig. 2 shows, ERN amplitude was smaller for the BPD group (-6.18 μV) than for the control group [-10.30 μV; \( F(1, 22) = 4.91, p = 0.037 \)]. There was no main effect of electrode site, indicating that the ERN was equally large at both electrodes \( (F < 1) \). The interaction between electrode site and group was not significant either \( (F < 1) \). In addition, no differences were found for the peak latency of the ERN \( (F < 1) \). For both groups, the ERN peaked around 74 ms after response onset. Importantly, ERN amplitude was also reduced in the BPD group \( (-4.65 \mu V) \) when patients who used medication were excluded from the analysis \( [F(1, 15) = 6.42, p = 0.023] \).

3.2.2. Stimulus-related N2

Though Fig. 3 may suggest an overall difference in N2 amplitudes between the two groups, analyses demonstrated that the main effect of group was not significant \( (F < 1) \). As expected from previous studies, N2 amplitude was more negative for incongruent stimuli \( (-5.65 \mu V) \) than for congruent ones \( [5.12 \mu V; \ F(1, 22) = 4.83, \ p = 0.039] \). More importantly, the interaction between congruency and group showed that this N2 congruency effect did not differ between the two groups \( (F < 1) \).
3.2.3. Feedback-related P300

Fig. 4 shows the feedback-locked ERP waveforms. A main effect of feedback type was present, indicating that P300 amplitude was affected by feedback $[F(2, 44) = 41.31, p < 0.001]$. Simple contrasts referenced to ‘correct’ feedback (5.14 μV) showed that P300 amplitude was larger after ‘late’ feedback $[13.79 \mu V; F(1, 22) = 54.82, p < 0.001]$, but not different after ‘incorrect’ feedback $[4.84 \mu V; F < 1]$. Although there was no main effect for group ($F < 1$), the interaction between type of feedback and group was significant $[F(2, 44) = 10.04, p = 0.002]$. Here, simple contrasts referenced to correct feedback revealed that this was caused by smaller P300 amplitudes after late feedback for the BPD group (10.69 μV) compared to the control group $[16.88 \mu V; F(1, 22) = 10.73, p = 0.003]$. These effects on P300 amplitude were also present when patients who used medication were excluded from the analysis. Importantly, in these restricted analyses P300 amplitude after late feedback was also found to be smaller for the BPD group $[9.44 \mu V; F(1, 15) = 6.65, p = 0.021]$.1

3.3. Performance adjustments

As important as the ability to detect an error is the ability to adjust performance following an incorrect response to prevent similar errors in the future. One way to accomplish a successful performance adjustment in the flankers task is to reduce the interference effect of the flanking letters by focusing on the central target in the letter string. This type of performance adjustment can be investigated in more detail by examining behavioural congruency effects following incorrect and correct responses (see e.g., De Bruijn et al., 2004a; Ridderinkhof et al., 2002). Typically, standard congruency effects caused by slower responses to incongruent stimuli than to congruent ones are reduced following errors. This reduction reflects a change in response strategy after an incorrect response. The congruency effect is computed by subtracting RTs on congruent stimuli from RTs on incongruent stimuli following correct and incorrect responses. Note that only correct responses that were preceded by incongruent trials were included in the analyses. As can be seen in Fig. 5, the analyses only revealed a significant interaction between post-correctness and group $[F(1, 11) = 9.64, p = 0.005]$. This interaction indicated that a reduction of the congruency effect following errors was present for the control group (18 ms), but not for the BPD group (–12 ms).

1 Please note that group differences were specific to the P300 as they were not present with respect to the earlier N1 and P2 components (both $Fs < 1$).
In the current study, action-monitoring processes were investigated in a group of patients with BPD and in a matched control group. The results demonstrated that patients with BPD show reduced action monitoring, reflected in attenuated ERN amplitudes.

The present finding is in line with the impulsive response style characteristically associated with patients suffering from BPD. This marked impulsivity was also evident in different behavioural measures. The BPD patients showed (1) increased reaction-time differences between correct and incorrect responses, and (2) an increased number of erroneous responses to congruent stimuli. As a minimum stimulus-processing time is required to give the correct response in a flankers task, a larger reaction-time difference between correct and incorrect responses indicates that the incorrect response is given relatively too fast. This larger reaction-time difference for patients with BPD thus reflects an impulsive response style. The increased number of incorrect responses to congruent stimuli is of particular interest, because congruent trials require relatively little attention to be processed correctly. When subjects produce errors due to diminished attention processes, slower RTs are expected. However, our results showed that the incorrect responses to congruent trials were the fastest of all responses in both groups. Therefore, our RT findings

4. Discussion

In the current study, action-monitoring processes were investigated in a group of patients with BPD and
suggest that such erroneous responses are not simply caused by a loss of concentration, but that specifically these errors are related to an impulsive response style.

Group differences were also observed for the feedback-related ERPs. Compared to the controls, the BPD patients showed decreased P300 amplitudes for late feedback. P300 amplitude following feedback in a similar flankers task was previously shown to be larger when subjects attributed more meaning to the stimulus (De Bruijn et al., 2004b). Consequently, the current reductions in P300 amplitudes corroborate the view that the BPD patients attribute less meaning to this type of negative feedback compared to the control subjects. This implies that patients with BPD indeed show decreased sensitivity to the negative consequences of their behaviour (see Moeller et al., 2001). Note that the ERPs following correct and incorrect feedback were similar for both groups. Although incorrect feedback can also be regarded as a negative consequence of behaviour, differences in ERP amplitude between correct and incorrect feedback are not expected, since the error is already detected earlier, viz. at the moment of response onset (see Holroyd and Coles, 2002). Reductions in P300 amplitudes have previously been found in patients with BPD (e.g. Blackwood et al., 1986), but also in patients with schizophrenia (see e.g. Ford et al., 2001), and in criminal psychopaths (Kiehl et al., 1999). However, the present data suggest that the currently found reduction in P300 is not just a reflection of a general impairment in cognitive processing. The P300 was only reduced in the BPD group following late feedback, indicating that this reduction is specifically related to action monitoring.

Although patients were free from medication on the day of testing, it needs to be mentioned that the influence of medication cannot be ruled out entirely. More specifically, it is known that ERP components related to action monitoring may be affected by psychopharmacological compounds (see e.g. De Bruijn et al., 2004a; Zirnheld et al., 2004). Yet, we feel that any such effects are likely to be limited or non-existent as additional analyses showed that the effects demonstrating reduced action-monitoring processes remained present when patients who did use medication were excluded.

4.1. Learning from errors

The theories that attempt to explain the complex interplay between prefrontal, mediofrontal, and limbic areas all seem to agree that the ACC is specifically involved in monitoring and/or selection processes. Prefrontal areas are assumed to play an important role in controlling actions and in keeping task demands up to date. According to the reinforcement learning theory of the ERN (Holroyd and Coles, 2002), predictive error signals indicate whether ongoing events are worse or better than expected and are used to improve performance. In this interpretation, the core function of the ERN is to learn from errors to optimize response behaviour. In the theory, the ACC plays the role of a control filter that is trained through the predictive error signals to select appropriate motor controllers.

The current data suggest that patients with BPD are specifically impaired in this learning process. A forced two-choice task like the one we employed does not require much learning. After a short practice block, the stimulus–response mappings are clear to all subjects. The only aspect of the task that does involve learning through feedback is the exact timing of the response. Participants need to respond within a certain time window: when they respond faster, they tend to respond erroneously and when they respond slower, the visual feedback will signal that they responded late. Problems with this process of fine-tuning responses in the BPD group are supported by the increased RT differences between correct and incorrect responses and by the absence of performance adjustments. These findings suggest that patients with BPD have more difficulty determining the optimal moment to initiate their response. In the current task, this optimal moment can be derived from two different types of feedback.

First, there is the external feedback, in the form of visual stimuli indicating that the current response was given late. Although the BPD patients showed decreased sensitivity to this type of feedback in the ERP results, the proportion of late responses did not differ between the two groups. The absence of such behavioural effects with regard to late feedback was likely caused by the instructions that explicitly emphasized speed and accuracy. However, a post-hoc analysis did confirm some decreased sensitivity at the behavioural level by demonstrating that controls gave significantly more correct responses following late feedback (79%) than patients with BPD (72%; p = 0.036).

The second type of feedback is more important. This feedback is internal and is reflected by the ERN. The ERN is generated after errors due to premature responding. Thus, the internal feedback can be used to boost alertness for the subsequent trials or to delay the next response until all stimulus features have been processed. The absence of performance adjustments following errors in the BPD group suggests that, unlike control participants, patients do not use this internal feedback to optimize the moment of response initiation.

Taken together, the increased impulsive response style employed by patients with BPD results in premature responding, i.e. before all the stimulus information has been adequately processed and evaluated. This impulsive response style is reflected in the BPD group in the increased RT differences between correct and incorrect responses and in the higher error rate on congruent trials. When patients with BPD make an error,
their ERN amplitudes are reduced compared to the control group. As the ERN is the result of action-monitoring processes, the reduced ERN amplitudes are an indication that the ACC or the action-monitoring network is part of is not functioning optimally in patients with BPD. Normally, error signals enable learning reflected in adaptations in behaviour necessary to prevent future errors. Diminished error signals will thus lead to the absence of such adaptations. In the current data, this is evident in the lack of performance adjustments in the BPD group. Finally, when responses are given before the stimulus is sufficiently processed, evaluation of a feedback stimulus that is presented even later in time is expected to be minimal. This is reflected in the reduced P300 amplitudes following late feedback in the BPD group. Late feedback provides valuable information on the timing of the response and is apparently not processed adequately. As a result, the entire action-monitoring process, i.e. from stimulus processing until adjusting performance, is affected in the BPD group. Consequently, patients with BPD do not learn from their errors as well as controls, thus maintaining their impulsive response style.

4.2. Affective evaluation of errors

A complementary explanation for the reduced ERN amplitudes is related to an assumed functional subdivision of the ACC. The caudal part of the ACC has strong connections to the dorsolateral prefrontal cortex and is involved in executive functions and motor control and is therefore termed the cognitive ACC. The rostral part of the ACC mainly projects to the limbic system and is thought to be involved in the emotional regulation of executive control, hence it is termed the affective ACC (for a review see Bush et al., 2000).

The affective role of the rostral ACC in action monitoring has been emphasized in an fMRI study by Laurens et al. (2003), in which error processing was investigated in schizophrenia. Laurens and colleagues demonstrated that patients with schizophrenia are specifically characterized by decreased rostral ACC activity during the commission of errors. As disturbances in affect and motivation are common and persistent symptoms of schizophrenia, the authors concluded that this reduced ACC activity leads to disturbances in a subjective affective error assessment process.

With regard to BPD, affective instability is, along with marked impulsivity, a main characteristic of the disorder. This is also evident in the problems patients with BPD have to contend with on a daily basis, which are mostly affective in nature rather than cognitive. Therefore it may be possible that, similar to patients with schizophrenia, the reduced action monitoring in our BPD group is mainly caused by a disturbance in the activity of the rostral part of the ACC, leading to a relative diminution of the affective response associated with the committed error. Future investigations using neuroimaging techniques with a higher spatial resolution like fMRI could explore whether a specific part of the ACC is responsible for reduced action monitoring in patients with BPD.

5. Conclusions

The current study demonstrated impulsivity-related reductions in action monitoring in a group of patients with BPD. The results indicate that patients with BPD do not learn from their errors as well as controls. Consequently, the patients do not adjust their behaviour following errors optimally, thus maintaining their impulsive response style. Although the present results warrant replication, in the near future this new insight may be applied to help explain the etiology and pathophysiology of BPD and foster the prognosis of treatments.

References


