The impact of perfectionism and anxiety traits on action monitoring in major depressive disorder

Didier L. Schrijvers · Ellen R. A. De Bruijn · Marianne Destoop · Wouter Hulstijn · Bernard G. C. Sabbe

Abstract  Perfectionism and anxiety features are involved in the clinical presentation and neurobiology of major depressive disorder (MDD). In MDD, cognitive control mechanisms such as action monitoring can adequately be investigated applying electrophysiological registrations of the error-related negativity (ERN) and error positivity (Pe). It is also known that traits of perfectionism and anxiety influence ERN amplitudes in healthy subjects. The current study explores the impact of perfectionism and anxiety traits on action monitoring in MDD. A total of 39 MDD patients performed a flankers task during an event-related potential (ERP) session and completed the multidimensional perfectionism scale (MPS) with its concern over mistakes (CM) and doubt about actions (DA) subscales and the trait form of the State Trait Anxiety Inventory. Multiple regression analyses with stepwise backward elimination revealed MPS-DA to be a significant predictor ($R^2$:0.22) for the ERN outcomes, and overall MPS ($R^2$:0.13) and MPS-CM scores ($R^2$:0.18) to have significant predictive value for the Pe amplitudes. Anxiety traits did not have a predictive capacity for the ERPs. MPS-DA clearly affected the ERN, and overall MPS and MPS-CM influenced the Pe, whereas no predictive capacity was found for anxiety traits. The manifest impact of perfectionism on patients’ error-related ERPs may contribute to our understanding of the action-monitoring process and the functional significance of the Pe in MDD. The divergent findings for perfectionism and anxiety features also indicate that the wide range of various affective personality styles might exert a different effect on action monitoring in MDD, awaiting further investigation.

Keywords  Major depression · Anxiety · Perfectionism · Error-related negativity · Error positivity

Introduction

Affective personality and temperament styles have been demonstrated to be involved in the neurobiology and clinical presentation of major depressive disorder (MDD). With regard to the symptomatology, a substantial body of evidence has been published demonstrating a positive association between severity of depressive symptoms and levels of perfectionism as well as anxiety features (Enns and Cox 1999; Huprich et al. 2008; Coryell et al. 1992; Parker et al. 1999; Clark and Watson 1991). Also, evidence from neuroendocrine, neuroanatomic, molecular and genetic studies has been published demonstrating common neurobiological factors for affective personality styles and major depression, pointing to a link between the pathophysiology of those personality traits and the pathophysiology of MDD (Foster and MacQueen 2008).

During the last decade, a large number of clinical studies have used electrophysiology to obtain better insight into the underlying neural mechanisms of various psychiatric
disorders (for an overview: see Ullsperger 2006; Olvet and Hajcak 2008). The error-related negativity (ERN), also known as the error negativity (Ne), is an event-related potential (ERP) that appears as a negative deflection and peaks approximately 50–100 ms after subjects have made a mistake (Gehring et al. 1993; Falkenstein et al. 1990). The Ne/ERN is known to be generated in the dorsal anterior cingulate cortex and is assumed to reflect dopaminergic activity related to the continuous evaluation and adjustment of ongoing actions (Dehaene et al. 1994; Ullsperger and Von Cramon 2001; Holroyd and Coles 2002; Jocham and Ullsperger 2009). The latter processes, also known as action monitoring, are an important aspect of our cognitive control mechanisms, as they enable fast and flexible adjustments to ongoing changes in our environment. Specifically, these adequate behavioural adjustments are often disturbed in various psychiatric disorders. Hence, measuring the Ne/ERN in psychiatric disorders allows for a detailed investigation of possible disturbances in the neural processes underlying action monitoring. The Ne/ERN is typically followed by error positivity (Pe), a slow positive wave with a centroparietal scalp distribution appearing approximately 200–400 ms after the onset of the erroneous response (Falkenstein et al. 1991) that is suggested to reflect conscious error awareness and subjective, affective evaluation of the error (Falkenstein et al. 2000; Overbeek et al. 2005).

Affective temperament styles have been demonstrated to influence Ne/ERN amplitudes in healthy controls. Trait features of affective distress, worry, negative affect, obsessive–compulsivity, anxiety and perfectionism have been documented to enhance Ne/ERN amplitudes or ACC activity in non-clinical subjects (Compton et al. 2007; Pieters et al. 2007; Hajcak et al. 2004a; Hajcak and Simons 2002; Paulus et al. 2004; Santesso et al. 2006). Accordingly, it has been described that perfectionistic subjects engage in hypervigilant monitoring of outcomes and selectively attend to failure (Shafran et al. 2002). Ne/ERN enhancements have also been observed in clinical samples with high levels of anxiety or perfectionism such as in obsessive–compulsive patients (Gehring et al. 2000; Johannes et al. 2001a; Hajcak and Simons 2002; Ursu et al. 2003; Ladouceur et al. 2006).

More recently, different research groups have investigated the Ne/ERN in MDD. Enhanced Ne/ERNs, but unchanged Pes, were observed in mildly to moderately depressed patients (Chiu and Deldin 2007; Holmes and Pizzagalli 2008). Since high levels of affective distress (such as symptoms of depression, obsessive compulsiveness and perfectionism, worry and anxiety traits, as well as experiences of negative affect) have been suggested to be characteristic for both affective and anxiety disorders (Clark and Watson 1991), it even has been assumed that the above-mentioned enhanced Ne/ERNs are not a function of either anxiety or depression specifically, but are related to the underlying high negative affect only (Hajcak et al. 2004a). Accordingly, some authors have suggested that Ne/ERN alterations might reflect an endophenotype for internalizing disorders such as depression and anxiety disorders (Olvet and Hajcak 2008). However, our research group demonstrated unchanged Ne/ERN amplitudes in a severely depressed sample and a clear association between the degree of psychomotor retardation and the level of attenuation on the Ne/ERN (Schrijvers et al. 2008a, b). We attributed the absence of Ne/ERN enhancements in our severely depressed patient sample to the attenuating effects of symptoms that are typical in severe depression, such as apathy, anhedonia and psychomotor retardation. We, moreover, found decreased Pe amplitudes in severely depressed patients with and without psychomotor retardation (Schrijvers et al. 2008a, b).

Hence, features of affective distress such as traits of anxiety and perfectionism play an important role in the clinical manifestation and pathophysiology of MDD, but also substantially influence the action-monitoring process in both clinical and nonclinical populations. Therefore, the aim of the current study is to investigate the impact of levels of anxiety and perfectionism on the action-monitoring process in severely depressed patients. Such an observable impact on the error-related ERPs in severe MDD could contribute to a better understanding of the previously reported ERP divergences between mild to moderate and severe MDD. Since perfectionism encompasses several dimensions, we will not only focus on ‘overall’ perfectionism when investigating the impact on the Ne/ERN, but also on several relevant subdimensions of the perfectionism construct. Based on earlier reports on the effects of high affective distress in clinical and non-clinical subjects, we expected depressed patients with high subjective levels of perfectionism and trait anxiety to generate larger Ne/ERN amplitudes than their counterparts reporting lower levels. In view of the limited literature on the impact of affective distress on Pe amplitudes, our investigations of this aspect should be seen as explorative.

### Methods

#### Subjects

We found 39 inpatients with MDD from four Belgian tertiary-care psychiatric hospitals willing to participate in our study. All had a DSM-IV-TR diagnosis of a major depressive (single or recurrent) episode. Patients with concurrent diagnoses of schizophrenia, psychoactive substance misuse or neurological disorders (e.g. dementia,
stroke, Parkinson’s disease, head injury or epilepsy) were excluded. Two patients were additionally diagnosed with a post-traumatic stress disorder, and two with chronic fatigue syndrome. All except one were on antidepressant medication with some taking a combination of antidepressants and low doses of benzodiazepines (n = 11) and/or antipsychotics (n = 13). Table 1 gives an overview of the medication taken by each patient. To assess depression severity, an experienced clinician (D.S.) administered the 17-item Hamilton Depression Rating Scale (HDRS; Hamilton 1960) prior to the ERP assessment.

All patients were native Dutch speakers who gave their informed consent after the nature of the study had been fully explained to them. The study was carried out consistent with the latest version of the Helsinki Declaration and was approved by the medical ethics committees of the participating clinics.

**Questionnaires**

The participants completed a Dutch version of the Multidimensional Perfectionism Scale (MPS; Soenens et al. 2005) and the Trait form of the State-Trait Anxiety Inventory (STAI-T; Spielberger et al. 1970; Van der Ploeg et al. 1980) within 2 days after the Ne/ERN assessment.

Besides providing an overall assessment of perfectionism, the MPS also gauges six core dimensions of perfectionism: personal standards (setting high standards for self-evaluation), concern over mistakes (reflecting negative reactions to mistakes), doubts about action (the tendency to doubt one’s abilities), organization (the importance placed on orderliness), parental expectations (the belief that one’s parents set very high goals) and parental criticism (the perception that one’s parents were overly critical; Frost et al. 1990). Participants were asked to rate each item on a five-point Likert-type scale (1 = not at all true; 5 = completely true) to indicate how they generally feel.

Only the overall MPS scores and the subdimensions, personal standards (MPS-PS), concern over mistakes (MPS-CM) and doubt about actions (MPS-DA), were entered into the statistical analyses. We chose not to enter the other perfectionism dimensions (organization, parental expectations, parental criticism) in the statistical model, since we did not expect these to affect the action-monitoring process.

The STAI-T is a 20-item self-report measure evaluating the tendencies to perceive stressful situations as dangerous or threatening and to respond to such situations with increased state anxiety, reflecting anxiety proneness. Previous research has shown that the STAI-T is a good indicator of general distress and negative affect (Nitschke et al. 2001). Participants were asked to rate each item on a four-point Likert-type scale (1 = ‘almost never’; 4 = ‘almost always’) as an indication of how they generally feel.

| Subject number | Medication (dosage in mg/day) |
|----------------|-----------------------------|
| 1              | Venlafaxine (225), Prazepam (10) |
| 2              | Venlafaxine (150), Lormetazepam (2) |
| 3              | Venlafaxine (150), Trazodone (100) |
| 4              | Venlafaxine (150), Mirtazapine (30), Prothipendyl (80) |
| 5              | Paroxetine (20), Mirtazapine (15), Quetiapine (500) |
| 6              | Escitalopram (10), Clonazepam (0,5) |
| 7              | Venlafaxine (150), Trazodone (150), Clorazepate (30) |
| 8              | Venlafaxine (225), Mirtazapine (30), Quetiapine (400) |
| 9              | Clomipramine (150), Mirtazapine (30), Lormetazepam (2) |
| 10             | Dosulepine (150) |
| 11             | Escitalopram (10), Mirtazapine (30), Amisulpride (800) |
| 12             | Paroxetine (10), Quetiapine (300), Lorazepam (3) |
| 13             | Escitalopram (10), Trazodone (100), Risperidone (2), Alprazolam (2) |
| 14             | Mirtazapine (30) |
| 15             | Fluoxetine (20), Trazodone (200), Amisulpride (50) |
| 16             | Mirtazapine (30), Alprazolam (0,5) |
| 17             | Venlafaxine (150), Amisulpride (50) |
| 18             | Venlafaxine (225), Trazodone (100) |
| 19             | / |
| 20             | Venlafaxine (75) |
| 21             | Fluvoxamine (100), Aripiprazole (10) |
| 22             | Escitalopram (10), Trazodone (100) |
| 23             | Paroxetine (30), Quetiapine (200) |
| 24             | Escitalopram (10), Trazodone (50) |
| 25             | Venlafaxine (225), Mirtazapine (30), Risperidone (2) |
| 26             | Escitalopram (10) |
| 27             | Escitalopram (10), Trazodone (50), Risperidone (2) |
| 28             | Escitalopram (10), Quetiapine (100) |
| 29             | Venlafaxine (150) |
| 30             | Paroxetine (40), Mirtazapine (30), Clonazepam (0,5) |
| 31             | Paroxetine (20), Mirtazapine (30), Trazodone (25) |
| 32             | Escitalopram (20), Trazodone (100) |
| 33             | Paroxetine (40) |
| 34             | Escitalopram (20), Trazodone (100) |
| 35             | Escitalopram (20), Trazodone (100), Clorazepate (10) |
| 36             | Escitalopram (20) |
| 37             | Fluvoxamine (400), Mirtazapine (30) |
| 38             | Paroxetine (40), Trazodone (100) |
| 39             | Escitalopram (20) |

Mean values for medication (standard deviation) were as follows: antidepressants (reference substance: imipramine): 223 mg (103); neuroleptics (reference substance: chlorpromazine): 261.5 mg (197); benzodiazepines (reference substance: diazepam): 8.7 mg (5.4)
Method and design

All participants performed a standard Eriksen flankers task (Eriksen and Eriksen 1974) in which they have to respond by pressing a button with either their left or their right index on the central letter (H or S) in a congruent (SSSSS or HHHHH) or incongruent (SSHSS or HSHSH) letter string. In the task instructions, equal emphasis was placed on speed and accuracy and the stimulus–response mappings were counterbalanced.

Because previous Ne/ERN studies had demonstrated that accuracy could affect Ne/ERN amplitudes (see e.g. Gehring et al. 1993), individual reaction-time (RT) deadlines were calculated first to ensure that error production would be similar in the patient group (De Bruijn et al. 2006b). This personal maximum RT comprises the interval that is needed to respond in order to avoid feedback indicating that the response was too late. To set their RTs, all subjects first performed a 60-trial practice block following verbal instructions with the initial RT deadline being set to a relatively liberal limit of 800 ms. After completion, the participants’ 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 six blocks of 100 trials (50 congruent/50 incongruent) with a self-paced pause halfway through each block. Verbal encouragements were given to keep performance accuracy at around 80–90%.

Subjects were first presented with a fixation point (lasting 100 ms). After 300 ms, the stimulus (also lasting 100 ms) appeared. For the next 900 ms, the screen remained blank, after which the visual feedback stimulus was shown (1,000 ms). The next trial was presented following a 100-ms intertrial interval. The visual feedback consisted of a yellow, blue or red rectangle indicating whether the response had been correct, incorrect or late, respectively. Responses were considered late when RTs exceeded the assigned deadline. The experimental phase lasted around 40 min including pauses.

EEG recording

The electroencephalogram (EEG) was recorded from 23 tin electrodes mounted in an elastic electrode cap (Electrocap International). Electrodes were placed at 3 midline (Fz/Cz/Pz) and 20 lateral (FP1-2/F7-8/F3-4/FC5-6/T3-4/C3-4/CP5-6/T5-6/P3-4/O1-2) locations in accordance with the extended 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 the right mastoid. The ground electrode was placed at the forehead. 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 bandpass filter between 0.02 and 30 Hz and digitized at 250 Hz.

Analyses

To eliminate EOG artefacts, we followed the procedure proposed by Gratton et al. (1983). For both behavioural and ERP analyses, all responses with RTs faster than 150 ms (1.1%) were removed from the data sets. For averaging, a minimum of 15 erroneous trials (on a total of 600 trials) were necessary. Correct and incorrect ERPs were separately averaged off-line, time locked to response onset, starting 200 ms before and ending 500 ms after response onset relative to a baseline from −200 to 0 ms preceding the response. Correct responses were additionally averaged separately for congruent and incongruent stimuli time locked to stimulus onset relative to a 200-ms pre-stimulus baseline.

For incorrect trials, Ne/ERN amplitudes were determined 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 electrode Cz and Fz, where maximal Ne/ERN amplitudes were expected (De Bruijn et al. 2004, 2006b; Schrijvers et al. 2008a, b). Since the Pe is a slow positive wave component without a clear peak, as was confirmed by visual inspection at the single-subject level, Pe amplitudes on incorrect trials were defined as the mean average amplitude in the 200–400 ms time window after response onset at electrodes Cz and Pz (Mathalon et al. 2002; Ullsperger and Szymanowski 2004).

Statistical analyses were performed using SPSS 14.0. Normality of the questionnaire data was tested with the Kolmogorov–Smirnov test. Repeated measures (RM) general linear model (GLM) software was used to examine the ERP variables in the total patient sample. For the overall GLM RM analysis, correctness [correct vs. incorrect responses] and electrode [Ne/ERN: (Fz, Cz); Pe: (Cz, Pz)] were entered as within-subject factors. Bivariate Pearson correlation analyses were conducted to explore the associations between the HDRS, MPS, STAI-T scores and the ERP variables. Subsequently, multiple linear regression with stepwise backward elimination was applied to closely investigate the impact of the perfectionism and anxiety features on the ERP amplitudes. Separate analyses will be conducted for each dependent variable, i.e. the Ne/ERN and Pe amplitudes, and the behavioural variables. Two models were used: in the first model, overall MPS and.
STAI-T scores were entered as covariates. In the second model, the scores for the DA, CM and PS MPS subdimensions were entered as covariates. In addition, the total HDRS scores were also entered as predictor in each model, since depression severity might also affect the ERP amplitudes. Given the explorative character of these analyses, the backward option of the linear regression method was applied. To investigate the possible effects of medication on the ERP amplitudes, additional Pearson correlation analyses were computed between, on the one hand, mean standardized dosages of the different types of medication and, on the other, ERP amplitudes.

Results

Clinical, ERP and behavioural variables

All patients [mean age: 39 years, SD = 11; male/female ratio: 13/26] were severely depressed as reflected in the mean total HDRS scores (25.9; SD = 5.7). According to the Belgian education system, level of education was subdivided into low (=1), average (=2) and high (=3) level, with a mean level of 2.03 (SD = 0.74) for the present sample. Mean overall MPS and total STAI-T scores were 83.1 (SD = 24.5) and 46.6 (SD = 5.6), respectively. All scores were relatively high compared to the values reported previously for several healthy control populations (e.g. Frost and Steketee 1997; Saboonchi and Lundh 1997). All data of the (subscales of the) clinical questionnaires and scales were normally distributed.

Mean amplitudes and latencies in the total patient cohort were −8.07 μV (SD: 5.57) and 80 ms (SD: 44) for the Ne/ERN and 4.82 μV (SD: 6.32) and 239 ms (SD: 91) for the Pe. RM GLM analyses in the overall sample revealed main effects of correctness for the Ne/ERN [F(1,38) = 22.15, p < 0.001] as well as the Pe [F(1,38) = 33.9, p < 0.001].

Table 2 Bivariate Pearson correlations between the total scores on the Hamilton depression rating scale (HDRS), the overall multidimensional perfectionism scores as well as its subscales concern over mistakes (MPS-CM), doubt about actions (MPS-DA), personal standards (MPS-PS), the trait scores (STAI-T) of the state-trait anxiety inventory and the ERP amplitudes at Fz and Pe amplitudes at Pz for all patients (n = 39)

|        | HDRS   | Overall MPS | MPS-PS | MPS-CM | MPS-DA | STAI-T | ERP (Fz) | Pe (Pz) |
|--------|--------|-------------|--------|--------|--------|--------|----------|---------|
| HDRS   | 1      | 0.075       | −0.01  | 0.16   | −0.02  | 0.23***| 0.06     | −0.03   |
| Overall MPS | ×     | 1           | 0.86***| 0.90***| 0.54***| 0.28*  | −0.19    | 0.36*   |
| MPS-PS | ×      | ×           | 1      | 0.75***| 0.36*  | 0.31*  | −0.089   | 0.212   |
| MPS-CM | ×      | ×           | ×      | 1      | 0.48***| 0.31*  | −0.09    | 0.43**  |
| MPS-DA | ×      | ×           | ×      | ×      | 1      | 0.06   | −0.47**  | 0.18    |
| STAI-T | ×      | ×           | ×      | ×      | ×      | 1      | −0.14    | 0.25*   |
| ERP (Fz) | ×     | ×           | ×      | ×      | ×      | ×      | 1        | −0.003  |
| Pe (Pz) | ×      | ×           | ×      | ×      | ×      | ×      | ×        | 1       |

# p < 0.1; * p < 0.05; ** p < 0.01; *** p < 0.001

The main effect of electrode approached significance for the Ne/ERN [Fz: −6.81 μV, Cz: −6.15 μV; F(1,38) = 3.44, p < 0.1], but not for the Pe [F < 1]. The correctness by electrode interaction was significant for the Ne/ERN [F(1,38) = 5.72, p < 0.05], but not for the Pe [F(1,38) = 1.32, p = 0.3]. As in previous studies, responses to congruent stimuli [427 ms] were faster than responses to incongruent stimuli [464 ms; F(1,38) = 7.72, p < 0.01], as were the RTs for incorrect [396 ms] responses relative to those for the correct ones [426 ms; F(1,38) = 66.04, p < 0.001]. Patients responded correctly in 77.6% (SD: 7.1) of all trials and were too late in 16.2% (SD: 7.3), whereas 6.2% (SD: 2.8) of all responses were incorrect. Congruent trials resulted in more erroneous and late responses [5.4 and 9.8%, respectively] than congruent trials [1.7%; F(1,38) = 61.54, p < 0.001 and 4.1%; F(1,38) = 95.17, p < 0.001, respectively]. Moreover, the analyses of the RTs following incorrect and correct responses yielded an additional main effect for post-correctness [F(1,38) = 53.15, p < 0.001]: responses following errors [496 ms] were more protracted than those following correct responses [449 ms], reflecting a behavioural adjustment known as post-error slowing (Rabbitt 1966).

No significant correlations between the standardized medication dosages and ERP amplitudes were found for antidepressant dosages (all r values < 0.23, all p values > 0.18) or for neuroleptic (all r values < 0.26, all p values > 0.42) and benzodiazepine dosages (all r values < 0.33, all p values > 0.23).

Effects of perfectionism and anxiety on action monitoring

Correlation analyses

The correlations between the respective dependent (Ne/ERN-Fz, Pe-Pz) and independent variables (total HDRS,
overall MPS, STAI-T, MPS-DA, MPS-CM, MPS-PS) are depicted in Table 2. Overall, the MPS and the MPS subdimension scores were intercorrelated. A significant negative correlation was found between the Ne/ERN and MPS-DA scores. The Pe amplitude correlated positively with the overall MPS and MPS-CM scores (significant) as well as with the STAI-T scores (nearly significant). No correlations were observed between MPS-CM, MPS-DA or STAI-T scores and any of the behaviour variables (reaction time, percentage of correct or incorrect responses, degree of post-error slowing). In addition, it is worth noting that HDRS scores correlated with the proportion of late Pe (0.132, p = 0.094) and correct responses (r = −0.29, p < 0.05) as well as with the mean RT (r = 0.30, p < 0.05), but not with any of the ERP variables.

Regression analyses

Ne/ERN Given the reported nearly significant main effect of electrode in favour of the Ne/ERN at Fz, we chose to enter the amplitude of the Ne/ERN at Fz as a dependent variable in the regression model. In the first step, overall MPS and STAI-T scores as well as total HDRS scores were entered as independent variables. The regression analyses could not show a significant impact of overall MPS scores (p = 0.23), STAI-T (p = 0.613) or total HDRS scores (p = 0.557) on the Ne/ERN amplitudes (see Table 3).

In a second analysis, MPS-DA, MPS-CM, MPS-PS and HDRS scores were entered as covariates. This analysis revealed the MPS subdimension DA to be a significant predictor for the Ne/ERN outcomes in our depressed sample: the coefficient of determination \( R^2 \) for this model was 0.222, which implied that approximately 22% of the variability of Ne/ERN amplitudes could be explained by this specific MPS subdimension (see Table 3). The other independent variables, i.e. MPS-CM (p = 0.27), MPS-PS (p = 0.786) and HDRS (p = 0.931) were automatically removed from the backward regression analysis.

Error positivity Because no significant main effect of electrode was found for the Pe amplitudes and Pe has repeatedly been demonstrated to be most pronounced at Pz (Overbeek et al. 2005), we chose to enter the amplitudes at electrode Pz in our final analyses.

Similar to the Ne/ERN analyses reported above, model 1 encompasses overall MPS, STAI-T and HDRS scores as independent variables. After backward elimination, overall MPS scores appeared to have a significant predictive value for the Pe amplitudes: as listed in Table 3, approximately 13% of the variability in Pe amplitudes can be explained by the amount of overall perfectionism (p < 0.05). STAI-T (p = 0.669) and HDRS scores (p = 0.317) were removed from this analysis.

In model 2, MPS-CM, -PS, -DA and HDRS scores were entered. This model revealed that especially MPS-CM has a predictive capacity of 18.5% for the Pe values (see Table 3). The other variables did not reach significance (MPS-DA: p = 0.62; MPS-PS: p = 0.269; HDRS: p = 0.474).

Behavioural measures Similar regression analyses were also conducted to search for a possible impact of perfectionism or anxiety features on the behavioural measures. The above-mentioned independent variables were entered into four separate analyses with the following dependent variables: reaction time, proportion of correct responses, proportion of errors and amount of post-error slowing. These analyses demonstrated that none of the perfectionism or anxiety variables executed a significant impact on any of the behavioural measures (all p values >0.12).

Median split analyses

Finally, for a visual demonstration of the data, we conducted an additional median split analysis, based on the results of the linear regression analyses. For the Ne/ERN amplitudes, the patient group was divided into a group of

---

**Table 3** Backward stepwise multivariate regression model for the error-related negativity (at Fz) and the error positivity (at Pz)

| Ne/ERN (Fz) | R² | B value | β value | p value | 95% Confidence interval for B |
|-------------|----|---------|---------|---------|-----------------------------|
| Model 1     | Overall MPS | 0.038 | -0.038 | -0.195 | 0.23 | -0.102; 0.026 |
| Model 2     | MPS-DA | 0.222 | -0.562 | -0.471 | 0.002 | -0.913; -0.212 |

| Pe (Pz) | R² | B value | β value | p value | 95% Confidence interval for B |
|---------|----|---------|---------|---------|-----------------------------|
| Model 1 | Overall MPS | 0.132 | 0.094 | 0.363 | 0.023 | 0.014; 0.174 |
| Model 2 | MPS-CM | 0.185 | 0.293 | 0.430 | 0.006 | 0.088; 0.499 |

Outcome measures (dependent variables) included determinants for each model (independent variables). R², B value (unstandardized coefficient) and β value (standardized coefficient) for each determinant and p value (significance); the last column represents the corresponding 95% confidence interval for each predictor. The excluded determinants for each model are not described in this table.

**Overall MPS**, overall score of the multidimensional perfectionism scale; **MPS-DA**, doubts about action subdimension of the MPS; **MPS-CM**, concern over mistakes subdimension of the MPS.
patients with high ($N = 20$, mean age = 40) and a group of patients with low scores ($N = 19$, mean age = 39) on the MPS-DA subscale. We did the same for the Pe amplitudes by dividing the patient group according to the MPS-CM scores (high scores: $N = 20$, mean age = 38; low scores: $N = 19$, mean age = 41). Figure 1 depicts the Ne/ERN grand averages of the high and low MPS-DA subgroups, and the Pe grand averages of the high and low MPS-CM grand averages. This figure clearly illustrates the demonstrated effects of MPS-DA and CM scores on,
respectively, the Ne/ERN and Pe amplitudes in our patient group. We also compared the respective MPS-DA and MPS-CM subgroups with regard to the mean standardized medication dosages to rule out a possible impact of medication on the observed differences: no differences were found between the MPS-CM subgroups ($F < 1$ for mean antidepressant, neuroleptic and benzodiazepine doses) or between the MPS-DA subgroups (antidepressants: $F = 1.30$, $p = 0.26$; neuroleptics: $F = 2.2$, $p = 0.12$; benzodiazepines: $F < 1$).

**Discussion**

The aim of the current study was to investigate the impact of perfectionism and anxiety traits on action monitoring in major depression, given on the one hand the involvement of perfectionism and anxiety in the pathophysiology and clinical manifestation of MDD, and on the other the reported impact of both affective personality features on the Ne/ERN. Concerning perfectionism, several features had a substantial effect on the error-related ERPs: the MPS-Doubt about actions subscale demonstrated a clear impact on the Ne/ERN, whereas overall perfectionism and its subscale concern over mistakes were found to affect Pe amplitudes. Surprisingly, anxiety traits did not affect the error-related ERPs. Moreover, neither perfectionism nor anxiety features influenced the behavioural action-monitoring measures.

**Perfectionism**

**Ne/ERN**

The current study provided support for an important effect of perfectionistic doubt about actions on the Ne/ERN: patients who displayed more doubts about their responses had significantly larger Ne/ERN amplitudes than those who were less doubtful. This MPS subdimension has been linked to ‘obsessional-like’ thinking. It reflects the extent to which people doubt their ability to accomplish tasks and thus expresses to what extent subjects are insecure about their performance and try to reach optimal results (Frost et al. 1990). These MPS-DA findings are generally in line with most studies investigating affective personality styles in healthy volunteers, mostly reporting enhanced Ne/ERNs or ACC activity in cohorts of college students scoring high on affective distress, worry, negative affect, anxiety and perfectionism (Hajcak et al. 2003, 2004a; Pailing and Segalowitz 2004). Note that also patients with obsessive–compulsive disorder have been demonstrated to manifest prominent Ne/ERN enhancements (Gehring et al. 2000; Johannes et al. 2001a).

The current results shed new light on the divergent Ne/ERN findings in major depression. As mentioned in “Introduction”, enhanced Ne/ERNs were previously observed in mild to moderately depressed community-dwelling patients (Chiu and Deldin 2007; Holmes and Pizzagalli 2008). For severely depressed inpatients, unchanged Ne/ERN amplitudes were reported, whilst patients with manifest psychomotor retardation, apathy and anhedonia showed attenuated Ne/ERNs (Schrijvers et al. 2008a, b, 2009). In the light of the current discussion, it is important to notice that our overall patient sample was severely depressed (as reflected in the mean total HDRS scores) and that our current findings thus underpin that also in severely depressed patients, perfectionism traits substantially affect the Ne/ERN. But why, then, were these enhancing effects not translated into heightened Ne/ERN signals in our earlier severely depressed sample (Schrijvers et al. 2008a, b)? In a previous study in MDD, we suggested that any Ne/ERN-enhancing effects might have been overruled by the attenuating effects of symptoms typical for severe depression, such as apathy, anhedonia and psychomotor retardation. Hence, the attenuating effect of the above-mentioned symptoms that are typical for severe depression could have been neutralized by the enhancing effect of the perfectionism style, leading to unchanged Ne/ERN amplitudes in severely depressed patients compared to healthy controls.

Second, we would like to mention the involvement of an individual’s personality or temperament in the aetiology and development of depressive disorder. Melancholic features have been demonstrated to be more prevalent in severe than in mild to moderate depression (Parker et al. 2000; Benazzi 2002). Whereas melancholia is considered to be a biological disorder with personality playing only a minor role in its aetiopathogenesis, personality styles such as anxious worrying, perfectionism, self-criticism and sensitivity to rejection are much more involved in the development and clinical picture of non-melancholic or mild to moderate depression (Parker and Manicavasagar 2005). Hence, we argue that the disparate Ne/ERN findings in severe and mild to moderate depression might, at least partially, be the reflection of differences in depressive subtypes and symptom profiles. Further research, also in mild to moderate depression, is needed to confirm this suggestion.

**Error positivity**

Furthermore, the current study demonstrated an apparent relation between the overall MPS scores and the MPS concern over mistakes dimension, and the Pe amplitudes in our severely depressed sample: patients who scored high on the total MPS and MPS-CM subscale had increased Pe.
amplitudes compared to patients who scored low. The CM subscale reflects negative reactions to mistakes, a tendency to interpret mistakes as equivalent to failure and a tendency to believe that one will lose the respect of others following failure.

Consistent with our findings, enhanced Pe components have earlier been found to be associated with higher levels of parent-reported obsessive–compulsive behaviours in a sample of nonclinical 10-year-old children (Santesso et al. 2006). Conversely however, Hajcak et al. (2004a) reported smaller error positivities for severely affectively distressed undergraduates than for those reporting low affective distress, and Santesso et al. (2005) found no impact of neuroticism on the Pe in healthy 10-year-old children. These inconsistent results clearly stress the need for future research that has to look more closely into the link between affective personality styles and the Pe. Nevertheless, a direct comparison of ours and the mentioned studies calls for caution since they all investigated very diverse, nonclinical samples, whereas our results were obtained in a cohort of depressed patients. Additionally, the different studies investigated a variety of affective personality styles, which might have a different impact on the error-related ERPs.

With regard to the functional significance of the Pe, the so-called affective-processing hypothesis as well as the behaviour-adaptation and error-awareness hypotheses have been proposed (Falkenstein et al. 2000; Overbeek et al. 2005). The affective-processing hypothesis states that the Pe reflects an emotional error-assessment process, which is modulated by the individual significance of an error (Falkenstein et al. 2000) and our Pe findings fit in well with this model. Indeed, the MPS-CM subdimension mainly mirrors the negative thoughts associated with the commission of an error, such as with the items, ‘how others think about me’ and ‘making an error is similar to total failure’. Accordingly, Frost and Trepanier (1997) reported high-scoring MPS-CM subjects to be more bothered by their mistakes and to react with more negative affect than subjects with low MPS-CM scores. The effect of these negative reactions may be even more pronounced in MDD due to the negative mood state inherent to the disorder. The relationship we observed between high MPS-CM scores and more pronounced Pe amplitudes therefore seems to support the suggestion that the Pe could be the reflection of the emotional corollaries related to erroneous events.

In general, it can be noted that the clinically observable contrast between more perfectionistic and more impulsive patients is also extrapolated to the error-related ERPs. Whereas the current and other clinical and nonclinical studies revealed an enhancing impact on the Ne/ERN of levels of perfectionism and other features of negative affect, studies on clinical and nonclinical populations with high levels of impulsivity, such as those with ADHD and borderline personality disorder, have demonstrated a negative relationship between impulsivity and Ne/ERN and Pe amplitudes (Ruchstow et al. 2006; de Bruijn et al. 2006b; Olvet and Hajcak 2008; Herrmann et al. 2010). Hence, the contrast between the enhancing impact of perfectionism and the diminished Ne/ERNs reported for highly impulsive subjects could reflect the opposite relationship between features of perfectionism and impulsivity, which are both traits related to behavioural disinhibition.

Furthermore, it needs to be mentioned that, rather than a single neurotransmitter, extensive interactions between serotonin, norepinephrine and dopamine are responsible for the complex pathogenesis of MDD, with the importance of dopamine increasing once melancholic features become manifest (Schrijvers et al. 2008a, b). With regard to the pathophysiology of the action-monitoring process, a pivotal role has been dedicated to dopamine, although recent studies also point to an impact of serotonin and norepinephrine (see Jocham and Ullsperger 2009). Features of perfectionism have previously been linked to a hyperactivity of the serotonergic system (Steiger et al. 2004). Hence, as both dopamine and serotonin dysregulations with their accompanying melancholic and perfectionism features, respectively, exert an impact on the action-monitoring process in MDD, the current findings appear to underline the complex interactions between the above-mentioned neurotransmitter mechanisms in the pathophysiology of MDD. However, these speculations need to be confirmed by future neurochemical studies.

Trait anxiety

With regard to the STAI scores, the regression analyses did not show a substantial effect of the measured anxiety traits on any of the ERP amplitudes. This is at odds with our initial research hypothesis, and is not in line with our findings on the perfectionism subdimensions. Given the repeatedly reported effect of anxiety on the Ne/ERN in healthy volunteers and anxiety being a frequent symptom during a depressive episode, we would have expected an observable impact of anxiety features on the Ne/ERN in severe depression.

A possible explanation for the divergent perfectionism and anxiety findings could be that the assessed trait anxiety features are more closely linked to the state-dependent depressive and anxiety symptoms than we previously expected, as can be seen in the nearly significant positive correlation between the HDRS and STAI-trait scores (but not MPS scores; see Table 2). It has indeed been suggested that the items of the trait scale may reflect depression rather than anxiety, going beyond the measurement of ‘pure’ anxiety (Bieling et al. 1998). Previously, it has also been
demonstrated that state anxiety features do not influence the Ne/ERN (Moser et al. 2005).

Furthermore, it has been reported that melancholic symptoms in MDD are associated with blunted ACC activity (Davidson et al. 2002; Mayberg 2003) and that trait anxiety features are linked with ACC hyperactivity (Paulus et al. 2004). It can be speculated that this blunted ACC state wipes out all additional effects on the ACC, such as the enhancing influence of anxiety traits, obstructing any enlarging impact on the Ne/ERN.

Behavioural measures

Although we found Ne/ERN and Pe differences in our MDD patients to be related to several aspects of perfectionism, we did not find corresponding differences in their performance on the flankers task. This divergence between ERP and behavioural measures is consistent with previous affective distress studies (Luu et al. 2000; Hajcak and Simons 2002; Hajcak et al. 2003, 2004a; Paulus et al. 2004; Moser et al. 2005; Ladouceur et al. 2006) and rules out the possibility that behavioural differences could explain the ERP differences that we and others observed (Hajcak et al. 2004b; Yeung 2004). Hajcak et al. (2003, 2004a) suggested the dissociation between performance and self-reported emotional reactions to be responsible for these findings. They proposed that pathological perfectionism and anxiety reflect unnecessary emotional reactions that are typically not associated with measurable performance differences. It is also noteworthy that the flankers task is a perceptually and conceptually straightforward test, implying that it does not place sufficient demands on processing resources for complex executive-control strategies or behavioural adjustments to be required for an adequate performance. It is probable that a more complex experimental task or real-world conditions would elicit corresponding differences in error-related ERPs and performance measures in high- and low-scoring subgroups.

Limitations and future research

Since several studies reported an impact of benzodiazepines and antipsychotics (but not antidepressants) on the Ne/ERN, the current study is limited in that all but one of our patients were on psychotropic medication at the time of the ERP recording session (Johannes et al. 2001b; De Bruijn et al. 2004, 2006a; Schrijvers et al. 2008a, b). However, additional analyses of the medication status did not reveal a large impact of the respective agents on the ERPs, indicating that the patients’ treatment regimens did not substantially affect the overall outcomes and conclusions. Moreover, our patient sample was highly representative of hospitalized MDD patients, most of whom were treated with a combination of different types of psychotropic drugs.

In the current paper, only perfectionism and trait anxiety features were investigated. However, the various affective personality styles appear to have a different effect on the ERP amplitudes in MDD. Therefore, to obtain a more complete picture of the impact of all these personality styles on action monitoring, additional personality inventories assessing, amongst others, obsessive–compulsive, worry or neuroticism features should be investigated. In addition, the use of more objective, clinician-rated scales for assessing these features would be needed to complement the self-report questionnaires mostly used in this type of research.

Finally, our sample mainly comprised severely depressed inpatients. Comparative studies that directly chart the effects of affective personality styles on action monitoring in a group with severe and a group with mild to moderate MDD could contribute to a further comprehension of the current knowledge of the Ne/ERN in MDD and of the impact of these personality styles in the pathophysiology of MDD.

The divergent results, i.e. the observed impact of perfectionism and the absence of an impact of anxiety on the action monitoring, clearly indicate the complex processes underlying the cognitive evaluation processes in MDD. Thus, the current results indicate that not all of the wide range of affective personality styles such as negative affect, neuroticism, obsessive–compulsive features, anxiety and perfectionism have the same effect on error-related ERPs in MDD. Moreover, it has already been demonstrated that other factors such as biological, environmental and genetic factors also exert, to a larger or lesser extent, an influence on action-monitoring processes (Anokhin et al. 2008; Holroyd and Coles 2002; Jocham and Ullsperger 2009). Besides that, MDD remains a very heterogeneous disorder with differences in the symptom profile or severity and underlying pathophysiology (such as neurotransmitter deficits) for each depressive subtype (Parker and Manicavasagar 2005). These differences and the possible impact of a lot of other factors might also contribute to the current divergent ERP results in mild to moderate and severe MDD (Chiu and Deldin 2007; Holmes and Pizzagalli 2008; Schrijvers et al. 2008a, b). The present and previous findings clearly show the complexity of this disease and its underlying cognitive processes, in which all the above-mentioned factors probably play a role, most of them interacting with each other.

Acknowledgments The authors would like to thank all participants for their cooperation, and Sara Vermeylen and Yvonne Maas for their help in administering the tests.
References

Anokhin AP, Golosheisky S, Heath AC (2008) Heritability of frontal brain function related to action monitoring. Psychophysiology 45:524–534

Benazzi F (2002) Psychomotor changes in melancholic and atypical depression: unipolar and bipolar-II subtypes. Psychiatry Res 112:211–220

Bieling PJ, Antony MM, Swinson RP (1998) The state-trait anxiety inventory, trait version: structure and content re-examined. Behav Res Ther 36:777–788

Chiu PH, Deldin PJ (2007) Neural evidence for enhanced error detection in major depressive disorder. Am J Psychiatry 164:608–616

Clark LA, Watson D (1991) Tripartite model of anxiety and depression: psychometric evidence and taxonomic implications. J Abnorm Psychol 100:316–336

Compton RJ, Carp J, Chaddock L, Quandt LC, Ratliff JW, Hulstijn W (2006b) Neural correlates of impulsive responding in healthy volunteers. Brain Res 1105:122–134

Davidson RJ, Pizzagalli D, Nitschke JB, Putnam K (2002) Depression: perspectives from affective neuroscience. Ann Rev Psychol 53:545–574

De Bruijn ERA, Hulstijn W, Verkes RJ, Ruigt GSF, Sabbe BGC (2004) Drug-induced stimulation and suppression of action monitoring in healthy volunteers. Psychopharmacology (Berl) 177:151–160

De Bruijn ERA, Sabbe BGC, Hulstijn W, Ruigt GSF, Verkes RJ (2006a) Effects of antipsychotic and antidepressant drugs on action monitoring in healthy volunteers. Brain Res 1105:122–129

De Bruijn ERA, Grootens KP, Verkes RJ, Buchholz V, Hummelen JW, Hulstijn W (2006b) Neural correlates of impulsive responding in borderline personality disorder: ERP evidence for reduced action monitoring. J Psychiatr Res 40:428–437

Dehaene S, Posner MI, Tucker DM (1994) Localisation of a neural system for error detection and compensation. Psychol Sci 5:303–305

Enns MW, Cox BJ (1999) Perfectionism and depression symptom severity in major depressive disorder. Behav Res Ther 37:783–794

Eriksen BA, Erriksen CW (1974) Effects of noise letters upon the identification of a target letter in a non-search task. Percept Psychophys 16:143–149

Falkenstein M, Hohnsbein J, Hoormann J, Blanke L (1990) Effects of errors in choice reaction tasks on the ERN under focused and divided attention: psychophysiological brain research. Tilburg University Press, Tilburg

Falkenstein M, Hohnsbein J, Hoormann J, Blanke L (1991) Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. Electroencephalogr Clin Neurophysiol 78:447–455

Falkenstein M, Hoormann J, Christ S, Hohnsbein J (2000) ERP components on reaction errors and their functional significance: a tutorial. Biol Psychol 51:87–107

Foster JA, MacQueen G (2008) Neurobiological factors linking personality traits and major depression. Can J Psychiatry 53:6–13

Frost RO, Steketee G (1997) Perfectionism in obsessive—compulsive disorder patients. Behav Res Ther 35:291–296

Frost RO, Trepanier KL (1997) Self-monitoring of mistakes among subjects high and low in perfectionistic concern over mistakes. Cogn Ther Res 21:209–222

Frost R, Marten P, Lahert C, Rosenblate R (1990) The dimensions of perfectionism. Cogn Ther Res 14:449–468

Gehring WJ, Goss B, Coles MG, Meyer DE, Al E (1993) A neural system for error detection and compensation. Psychol Sci 4:385–390

Gehring WJ, Himle J, Nisenson LG (2000) Action-monitoring dysfunction in obsessive—compulsive disorder. Psychol Sci 11:1–6

Gratton G, Coles MG, Donchin E (1983) A new method for off-line removal of ocular artifact. Electroencephalogr Clin Neurophysiol 55:468–484

Hajcak G, Simons RF (2002) Error-related brain activity in obsessive—compulsive undergraduates. Psychiatry Res 110:63–72

Hajcak G, McDonald N, Simons RF (2003) Anxiety and error-related brain activity. Biol Psychol 64:77–90

Hajcak G, McDonald N, Simons RF (2004a) Error-related psychophysiology and negative affect. Brain Cogn 56:179–189

Hajcak G, Vidal F, Simons RF (2004b) Difficulties with easy tasks: ERN/Ne and stimulus component overlap. In: Ullsperger M, Falkenstein M (eds) Errors, conflicts, and the brain: current opinions on performance monitoring. MPI for Human Cognitive and Brain Sciences, Leipzig, pp 204–211

Hamilton M (1960) A rating scale for depression. J Neurol Neurosurg Psychiatry 23:56–62

Herrmann M, Mader K, Schreppel T, Jacob C, Heine M, Boreatti-Hümmle A, Ehlis A, Scheuerpfug P, Pauli P, Fallgatter A (2010) Neural correlates of performance monitoring in adult patients with attention deficit hyperactivity disorder (ADHD). World J Biol Psychiatry 11:457–464

Holroyd CB, Coles MG (2002) The neural basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. Psychol Rev 109:759–790

Huprich SK, Porcerelli JP, Keaschuk R, Binienda J, Engle B (2008) Depressive personality disorder, dysthymia, and their relationship to perfectionism. Depress Anxiety 25:207–217

Jocham G, Ullsperger M (2009) Neuropharmacology of performance monitoring. Neurosci Biobehav Rev 33:48–60

Johannes S, Wieringa BM, Nager W, Rada D, Dengler R, Emrich HM, Munte TF, Dietrich DE (2001a) Discrepan target detection and action monitoring in obsessive—compulsive disorder. Psychiatry Res Neuroimag 108:101–110

Johannes S, Wieringa BM, Nager W, Dengler R, Munte TF (2001b) Oxazepam alters action monitoring. Psychopharmacology (Berl) 155:100–106

Johannes S, Wieringa BM, Nager W, Rada D, Dengler R, Emrich HM, Munte TF, Dietrich DE (2001a) Discrepan target detection and action monitoring in obsessive—compulsive disorder. Psychiatry Res Neuroimag 108:101–110

Johannes S, Wieringa BM, Nager W, Dengler R, Munte TF (2001b) Oxazepam alters action monitoring. Psychopharmacology (Berl) 155:100–106

Ladouceur CD, Dahl RE, Birmaher B, Axelson DA, Ryan ND (2006) Increased error related negativity (ERN) in childhood anxiety disorders: ERP and source localization. J Child Psychol Psychiatry 47:1073–1082

Lau P, Collins P, Tucker DM (2000) Mood, personality and self-monitoring: negative affect and emotionality in relation to frontal lobe mechanisms of error monitoring. J Exp Psychol Gen 129:43–60

Mathalon DH, Fedor M, Faustman WO, Gray M, Askari N, Ford JM (2002) Response-monitoring dysfunction in schizophrenia: an event-related brain potential study. J Abnorm Psychol 111:22–41

Open Access This article is distributed under the terms of the Creative Commons Attribution Noncommercial License which permits any noncommercial use, distribution, and reproduction in any medium, provided the original author(s) and source are credited.

Perfectionism, anxiety and action monitoring in depression
Mayberg HS (2003) Modulating dysfunctional limbic-cortical circuits in depression: towards development of brain-based algorithms for diagnosis and optimised treatment. Br Med Bull 65:193–207
Moser JS, Hacajk G, Simons RF (2005) The effects of fear on performance monitoring and attentional allocation. Psychophysiology 42:261–268
Nitschke JB, Heller W, Imig JC, McDonald RP, Miller GA (2001) Distinguishing dimensions of anxiety and depression. Cogn Ther Res 25:1–22
Olvet DM, Hajcak G (2008) The error-related negativity (ERN) and psychopathology: toward an endophenotype. Clin Psychol Rev 28:1343–1354
Overbeek TJM, Nieuwenhuis S, Ridderinkhof KR (2005) Dissociable components of error processing: on the functional significance of the Pe Vis-à-vis the ERN/Ne. J Psychophysiol 19:319–329
Pailing PE, Segalowitz SJ (2004) The error-related negativity as a state and trait measure: motivation, personality, and ERPs in response to errors. Psychophysiology 41:84–95
Parker G, Manicavasagar V (2005) Modelling and managing the depressive disorders: a clinical guide. Cambridge University Press, Cambridge
Parker G, Wilhelm K, Mitchell P, Austin MP, Roussos J, Gladstone G (1999) The influence of anxiety as a risk to early onset major depression. J Affect Disord 52:11–17
Parker G, Roy K, Hadzi-Pavlovic D, Mitchell P, Wilhelm K, Menkes DB et al (2000) Subtyping depression by clinical features: the Australian database. Acta Psych Scan 101:21–28
Paulus MP, Feinstein JS, Simmons A, Stein MB (2004) Anterior cingulate activation in high trait anxious subjects is related to altered error processing during decision making. Biol Psychiatry 55:1179–1187
Pieters GLM, de Bruijn ERA, Maas Y, Hulstijn W, Vandereycken W, Paulus MP, Feinstein JS, Simmons A, Stein MB (2004) Anterior cingulate activation in high trait anxious subjects is related to altered error processing during decision making. Biol Psychiatry 55:1179–1187
Pieters GLM, de Bruijn ERA, Maas YJ, Vancoillie P, Hulstijn W, Sabbe BGC (2005) ERP correlates of error processing in borderline personality disorder. Psychophysiology 42:261–268
Saboonchi F, Lundh L-G (1997) Perfectionism, self-consciousness and anxiety. Pers Individ Dif 22:921–928
Santesso DL, Segalowitz SJ, Schmidt LA (2005) ERP correlates of error monitoring in 10-year olds are related to socialization. Biol Psychol 70:79–87
Santesso DL, Segalowitz SJ, Schmidt LA (2006) Error-related electrocortical responses are enhanced in children with obsessive—compulsive behaviors. Dev Neuropsychol 29:431–445