Action monitoring and perfectionism in anorexia nervosa

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Abstract

To study action monitoring in anorexia nervosa, behavioral and EEG measures were obtained in underweight anorexia nervosa patients \((n=17)\) and matched healthy controls \((n=19)\) while performing a speeded choice-reaction task. Our main measures of interest were questionnaire outcomes, reaction times, error rates, and the error-related negativity ERP component. Questionnaire and behavioral results indicated increased perfectionism in patients with anorexia nervosa. In line with their perfectionism and controlled response style patients made significantly less errors than controls. However, when controlling for this difference in error rates, the EEG results demonstrated a reduced error-related negativity in the patient group. These seemingly contradictory outcomes of improved performance and reduced error monitoring are discussed in relation with indications of anterior cingulate cortex hypoactivity in anorexia nervosa patients.

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1. Introduction

Anorexia nervosa (AN) is characterized in DSM-IV as demonstrating severe weight loss, fear of gaining weight, preoccupation with body appearance, and amenorrhea. Perfectionism is considered to be a key characteristic in AN and to play a role in the etiology and maintenance of the disorder (see e.g. Franco-Paredes, Mancilla-Diaz, Vazquez-Arevalo, Lopez-Aguilar, & Alvarez-Rayon, 2005; Shafran, Cooper, & Fairburn, 2002; Shafran & Mansell, 2001). For example, AN patients score higher on perfectionism, even after long-term weight recovery (Bastiani, Rao, Weltzin, & Kaye, 1995; Casper, 1990; Srinivasagam et al., 1995) and they employ a slow but accurate response style compared to healthy controls (Holliday, Tchanturia, Landau, Collier, & Treasure, 2005; Kaye, Bastiani, & Moss, 1995).

Perfectionist subjects engage in hypervigilant monitoring of outcomes and selectively attend to failure (Shafran et al., 2002). Bulik and co-workers (2003) showed that elevated concern over mistakes is specifically associated with AN. In general, optimal performance requires minimizing the number of errors by continuously monitoring one’s actions and their outcomes.

Action monitoring can be investigated in more detail by examining the so-called ‘error-related negativity’ or ERN (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The ERN is a negative response-locked event-related potential (ERP) component elicited immediately following an error. Originally, the ERN has been interpreted as the outcome of a generic error-detection mechanism, where the error signal is used as input for a remedial action system, enabling performance adjustments for correction or prevention of future...
errors (see e.g. Coles, Scheffers, & Holroyd, 2001). Alternatively, the ERN has been described as the reflection of response conflict that arises when two incompatible response tendencies are simultaneously activated (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Yeung, Cohen, & Botvinick, 2004). A third interpretation of the ERN attributes a more central role to affective or motivational processes in action monitoring, arguing that the ERN (additionally) reflects an affective evaluation of the error (De Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe, 2005; Gehring & Willoughby, 2002; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003; Pailing, Segalowitz, Dywan, & Davies, 2002; Yeung, 2004). Several source localization studies on ERP data (see e.g. Dehaene, Posner, & Tucker, 1994; Van Veen & Carter, 2002; Holroyd, Dien, & Coles, 1998) and functional Magnetic Resonance Imaging (fMRI) studies (see e.g. Carter et al., 1998; Kiehl, Liddle, & Hopfinger, 2000; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Ullsperger & von Cramon, 2001) have indicated the anterior cingulate cortex (ACC) as the likely generator of the ERN. All theories seem to agree that the ERN reflects the outcome of an action-monitoring process in which the signal is used to adjust behavior to improve performance and prevent future errors. In speeded choice-reaction tasks, errors are usually the result of premature responding. A possible performance adjustment is then to slow down on the trial following an error, a phenomenon known as post-error slowing (Rabbitt, 1966).

Alterations in action-monitoring are associated with differences in the response style people employ. Enhanced ERN amplitudes were for instance found in normal subjects with a controlled response style (Pailing et al., 2002), and in non-clinical subjects with obsessive-compulsive characteristics (Hajcak & Simons, 2002). In line with this, patients suffering from obsessive-compulsive disorder (OCD) also showed enhanced ERN amplitudes (Gehring, Himle, & Nisenson, 2000; Johannes et al., 2001). These increased ERN amplitudes coincide with positron emission tomography (PET) and fMRI studies demonstrating increased ACC activity in OCD patients (Adler et al., 2000; Ursu, Stenger, Shear, Jones, & Carter, 2003). Importantly, reviews on the phenomenological and neurobiological links between OCD and AN, and data from family and genetic studies seem to confirm that perfectionism is a, possibly genetically transmitted, common vulnerability factor for AN and OCD (see e.g. Anderluh, Tchanturia, Rabe-Hesketh, & Treasure, 2003; Bulik & Tozzi, 2004; Halmi et al., 2000; Monteleone, Brambilla, Bortolotti, & Maj, 2000). However, neuroimaging studies in the resting condition in AN, rather suggest hypoperfusion in the ACC (Delvenne et al., 1995; Naruo et al., 2001; Takano et al., 2001) for a review, see (Frank, Bailar, Henry, Wagner, & Kaye, 2004), which persists after weight gain (Kojima et al., 2005).

The aim of the current study is to investigate action monitoring in AN patients and in matched controls in more detail. In addition to ERP and behavioral measurements of a speeded choice-reaction task, we obtained results from questionnaires to assess perfectionism. Based on previous research (Bastiani et al., 1995; Casper, 1990; Srinivasagam et al., 1995), we expected questionnaire outcomes and behavioral measures to demonstrate increased perfectionism and a more controlled response style in the AN patients. With respect to the ERN analyses two possible outcomes were predicted. The first prediction was based on the earlier findings of enhanced ERN amplitudes in patients with OCD. When the controlled response style of patients with AN coincides with increased action monitoring as issued by the ACC, larger ERN amplitudes were also expected for this patient group. On the other hand, when perfectionism is not associated with increased action monitoring as issued by the ACC, we expected smaller ERN amplitudes compared to controls. Such reduction in ERN amplitude would be in line with studies demonstrating ACC hypoactivity in AN patients.

2. Materials and methods

2.1. Participants

Thirty-six participants (17 AN patients and 19 controls, matched for sex, age, and educational level) took part in the study. Patients were hospitalized in a specialized treatment center for eating disorders during a 12-month period. Inclusion criteria for the patients were: a diagnosis of AN-restricting subtype according to DSM-IV criteria, a body mass index (BMI) below 17.5 kg/m², and absence of psychotic or substance-related disorder. All eligible patients were approached, and none of them refused to participate. Controls were age-matched healthy volunteers showing no indications of an eating disorder during a short interview and on two self-reporting measures (EDI and EDES; see next paragraph) and who had a BMI above 19 kg/m². During the interview, controls were also screened for a history of severe head injury, substance abuse and psychiatric referral, which were used as exclusion criteria. All participants gave written informed consent before participation and the study was approved by the ethical committee of the University Center in Kortenberg.

2.2. Clinical assessment

Patients and controls were measured and weighed on the day of testing. Seven patients were taking psychotropic medication: five received selective serotonin reuptake inhibitors (SSRIs), four received a short-acting benzodiazepine as a sleep inducer. The patients did not take this medication within 24 h before testing. All participants completed the following self-report questionnaires: the Symptom Checklist (SCL-90; Derogatis, 1977) and the Beck depression inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) were used to assess a wide array of psychiatric symptoms; The eating disorder inventory (EDI; Garner, Olmsted, & Polivy, 1983) and the eating disorder evaluation scale (EDES; Vandereycken, 1993) were used to assess perfectionism.
assessed psychological and clinical aspects directly related to eating disorders; the multidimensional perfectionism scale (MPS; Frost, Marten, Lahert, & Rosenblate, 1990) provided an overall assessment of perfectionism as well as 6 core dimensions of perfectionism. This last scale has been proven to discriminate successfully between subjects with and without AN (Srinivasagam et al., 1995). AN patients scored significantly higher than normal controls on the MPS in a 6- to 24-month follow-up study (Sutandar-Pinnock, Blake, Carter, Olmsted, & Kaplan, 2003). Limited research on the psychometric qualities of (3 sub-scales) of the Dutch translation of the MPS indicates that this translation has similar psychometric qualities as the original version (Soenens, Vansteenkiste, Luyten, Duriez, & Goossens, 2005).

2.3. Design and procedure

Participants performed the so-called flankers task (Eriksen & Eriksen, 1974) in which they had to respond with either their left or right index finger to the central letter (H or S) in a congruent (HHHHH and SSSSS) or incongruent (HHSHH and SSHSS) letter string. Equal emphasis was placed on speed and accuracy in the written and verbal task instructions.

Because previous ERP 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 Brujin, Hulstijn, Verkes, Ruigt, & Sabbe, 2004), ensures similar performance levels of 75–80% accuracy. 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 six blocks of 100 trials (i.e. 50 congruent and 50 incongruent) with a self-paced pause halfway through each block. After each block, participants received feedback on performance. Verbal encouragements were given to keep performance accuracy around 75–80%. Each trial began with the presentation of a fixation point for 100 ms, followed 300 ms later by the stimulus also lasting 100 ms. During the following 900 ms the screen remained blank, after which a visual feedback stimulus appeared for 1000 ms. The next trial was presented after an inter-trial interval of 100 ms. The feedback was a yellow, a blue, or a red rectangle indicating whether the response had been correct, incorrect, or too late, respectively. The total duration of the experimental phase was around 40 min including breaks.

2.4. EEG recording

The electroencephalogram (EEG) was recorded from 27 tin electrodes mounted in an electrode cap (Electrocap International) referenced to the average of both mastoids. Electrodes were placed at midline (Fpz/AFz/Fz/FCz/Cz/Pz/Oz) and lateral (FP1–2/F7–8/F3–4/FC5–6/T3–4/C3–4/ CP5–6/T5–6/P3–4/01–2) locations. The electro-oculogram (EOG) was recorded from electrodes placed above and below the right eye, and from electrodes placed lateral to both eyes. All electrode impedances were kept below 5 kΩ. The EEG and EOG signals were amplified using a band-pass filter between .02 and 30 Hz and digitized at 200 Hz.

2.5. Analyses

EOG artifact correction was carried out using the procedure proposed by Gratton, Coles, and Donchin (1983). ERPs for correct and incorrect responses were averaged separately off-line time-locked to response onset, starting 200 ms before and ending 500 ms after response onset relative to a 100 ms pre-response baseline. ERN amplitude was determined on the averages per individual participant for incorrect responses by subtracting the most positive peak in the time window starting 80 ms before and ending 80 ms after response onset from the most negative peak in the 0–200 ms time window after response onset at electrode Cz, where ERN amplitude was maximal.

The P3 component was defined as the most positive peak in the 300–800 ms time window after stimulus onset at electrodes Fz, FCz, Cz, and Pz on correct trials only. The Pe component was defined as the mean amplitude of the difference wave (incorrect minus correct response) in the 200–400 ms time window after response onset at electrodes Fz, FCz, Cz, and Pz.

Individual averages for RTs and amplitudes were entered in a repeated measures ANOVA with group (controls vs. AN) as between-subject factor. Possible within-subject factors were congruency (congruent vs. incongruent) and correctness (correct vs. incorrect). Since error rates differed between groups (see Section 3) and might potentially influence RTs and ERN amplitudes, Pearson correlation coefficients were used to determine any relationship between error rates and these measures. When a relation was found, the percentage of errors was introduced as a covariate in ANCOVAs with ERN amplitudes and RTs as dependent variables, to control for this possible confounder in the group comparisons. Similarly, correlation coefficients between depression (BDI) scores and ERN amplitudes were calculated to determine possible effects of depression on the ERN.

ANOVA were conducted with the proportions of the different types of responses (i.e. correct, error, too late) as dependent variables and group as factor. Post-error slowing was quantified as the difference in RT between correct responses following incorrect responses and correct responses following correct responses. Please note that late
### Table 1
Clinical variables (standard deviation) of restricting AN patients and controls

<table>
<thead>
<tr>
<th></th>
<th>AN (n = 17)</th>
<th>Controls (n = 19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>20.2 (3.8)</td>
<td>20.5 (3.6)</td>
</tr>
<tr>
<td>Duration of illness (years)</td>
<td>3.7 (3.4)</td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>40.4 (6.5)</td>
<td>60.1 (5.4)</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.7 (0.1)</td>
<td>1.7 (0.1)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>14.5 (1.2)</td>
<td>21.9 (2.0)</td>
</tr>
<tr>
<td>MPS overall perfectionism</td>
<td>80.0 (31.7)</td>
<td>51.3 (13.2)</td>
</tr>
<tr>
<td>EDI total score</td>
<td>66.7 (40.1)</td>
<td>17.0 (12.4)</td>
</tr>
<tr>
<td>BDI total score</td>
<td>28.2 (13.1)</td>
<td>4.0 (3.6)</td>
</tr>
<tr>
<td>SCL-90 total score</td>
<td>246.7 (90.6)</td>
<td>126.9 (22.8)</td>
</tr>
<tr>
<td>SCL-90 anxiety</td>
<td>270.0 (12.1)</td>
<td>144.4 (3.2)</td>
</tr>
<tr>
<td>SCL-90 agoraphobia</td>
<td>14.8 (7.4)</td>
<td>8.1 (2.0)</td>
</tr>
<tr>
<td>SCL-90 depression</td>
<td>52.6 (19.3)</td>
<td>22.5 (6.0)</td>
</tr>
<tr>
<td>SCL-90 somatization</td>
<td>30.1 (13.4)</td>
<td>17.4 (4.2)</td>
</tr>
<tr>
<td>SCL-90 inadequacy</td>
<td>26.8 (10.2)</td>
<td>13.2 (3.6)</td>
</tr>
<tr>
<td>SCL-90 distrust interpersonal sensitivity</td>
<td>51.0 (18.2)</td>
<td>25.4 (6.3)</td>
</tr>
<tr>
<td>SCL-90 hostility</td>
<td>13.2 (5.8)</td>
<td>8.7 (4.1)</td>
</tr>
<tr>
<td>SCL-90 sleep problems</td>
<td>9.3 (4.4)</td>
<td>4.9 (2.8)</td>
</tr>
<tr>
<td>SCL-90 additional</td>
<td>21.8 (8.6)</td>
<td>12.3 (3.0)</td>
</tr>
<tr>
<td>EDES adjustment</td>
<td>12.8 (5.1)</td>
<td>20.0 (3.9)</td>
</tr>
<tr>
<td>EDES sexuality</td>
<td>5.4 (4.3)</td>
<td>15.5 (2.5)</td>
</tr>
<tr>
<td>EDES bulimia</td>
<td>15.9 (4.3)</td>
<td>16.4 (1.4)</td>
</tr>
<tr>
<td>EDES preoccupation</td>
<td>5.0 (4.7)</td>
<td>20.3 (4.4)</td>
</tr>
<tr>
<td>EDES body dissatisfaction</td>
<td>13.2 (7.7)</td>
<td>7.5 (6.6)</td>
</tr>
<tr>
<td>EDES perfectionism</td>
<td>5.9 (5.8)</td>
<td>1.2 (1.7)</td>
</tr>
<tr>
<td>EDES interpersonaldistrust</td>
<td>7.2 (4.7)</td>
<td>1.1 (2.6)</td>
</tr>
<tr>
<td>EDES introceptive awareness</td>
<td>8.7 (4.7)</td>
<td>0.6 (1.3)</td>
</tr>
<tr>
<td>EDES maturity fears</td>
<td>7.1 (6.5)</td>
<td>2.9 (2.4)</td>
</tr>
<tr>
<td>MPS overall perfectionism</td>
<td>80.0 (31.7)</td>
<td>51.3 (13.2)</td>
</tr>
<tr>
<td>MPS concern over mistakes</td>
<td>28.3 (12.9)</td>
<td>14.2 (4.4)</td>
</tr>
<tr>
<td>MPS personal standards</td>
<td>22.8 (8.5)</td>
<td>16.0 (4.8)</td>
</tr>
<tr>
<td>MPS parent expectations</td>
<td>8.2 (4.5)</td>
<td>8.4 (3.7)</td>
</tr>
<tr>
<td>MPS parental criticism</td>
<td>7.7 (4.2)</td>
<td>5.2 (2.2)</td>
</tr>
<tr>
<td>MPS double of actions</td>
<td>13.2 (5.6)</td>
<td>7.5 (2.7)</td>
</tr>
<tr>
<td>MPS organization</td>
<td>23.7 (5.5)</td>
<td>19.9 (5.2)</td>
</tr>
</tbody>
</table>

* a p ≤ 0.05.
* b p ≤ 0.01 (t-test between groups).

But correct responses were also included in the post-error slowing analyses, as slowing down after an error often results in a late response due to use of the strict RT dead-

### Table 2
Behavioral analyses

<table>
<thead>
<tr>
<th></th>
<th>AN (n = 17)</th>
<th>Controls (n = 19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correct</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RT (ms)</td>
<td>361 (32)</td>
<td>391 (36)</td>
</tr>
<tr>
<td>Proportion (%)</td>
<td>90.90 (3.48)</td>
<td>72.41 (7.19)</td>
</tr>
<tr>
<td>Incorrect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RT (ms)</td>
<td>337 (60)</td>
<td>330 (42)</td>
</tr>
<tr>
<td>Proportion (%)</td>
<td>2.90 (2.61)</td>
<td>9.69 (5.01)</td>
</tr>
<tr>
<td>Too late</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proportion (%)</td>
<td>6.20 (2.91)</td>
<td>17.90 (6.57)</td>
</tr>
</tbody>
</table>

Overall RTs and proportions for the different types of responses to congruent and incongruent stimuli for the AN and the control group. Standard deviations are given in parentheses.

### 3. Results

#### 3.1. Clinical variables and questionnaires

Table 1 presents mean age, BMI, and scores on clinical rating scales for both groups. AN patients scored significantly higher on MPS overall perfectionism and on all MPS subscales except parent expectations.

#### 3.2. Behavioral results: reaction times, proportions of error responses, and performance adjustments

Reaction times are summarized in Table 2. There was no significant group difference for reaction times \([F(1, 34) = 1.48, p = .232]\) and also the mean individual reaction-time deadlines did not differ between the two groups (control group: 478 ms; AN group: 490 ms; \(F(1,34) < 1, p = .426\)). Incorrect responses were faster (328 ms) than correct responses [366 ms; \(F(1,34) = 68.48, p < .001\)] and congruent stimuli (338 ms) were responded to faster than incongruent ones [356 ms; \(F(1,34) = 29.68, p < .001\)]. Interestingly, the three-way interaction between group, congruency, and correctness was significant \((F (1,34) = 4.26, p = .047)\). Post hoc tests revealed that a congruency effect for incorrect responses was present for the control group \([19 ms; t = -3.22, p = .004]\), but not for the AN group \([-7 ms; t = .65, p = .526]\). Significant negative correlations were found between error rates and RTs (control group: \(r = -0.685, p = .001\); AN group: \(r = -0.425, p = .088\)). Therefore, error rate was introduced as a covariate in an ANCOVA with RT as dependent variable. Again, no significant difference in RT was found between groups: \(F(1,33) < 1, p = .728\).

The analyses of the proportions of incorrect and too late trials showed only a significant group difference for the amount of incorrect responses. More errors were made by...
the control group (11.00%, range: 4–31%) compared to the AN group (6.29%, range: 1–12%; F(1, 34) = 7.95, p = .008).

As depicted in Fig. 1, the control group displayed significantly post-error slowing [20 ms; t = 3.07, p = .007], while this effect was only marginally significant for the AN group [11 ms; t = 2.00, p = .063].

Finally, analyses on the proportion of correct and incorrect responses immediately after an error demonstrated that AN patients followed an error significantly less often by another error (4.73%) than controls did [11.27%; F(1, 34) = 8.66, p = .006]. The proportions of correct responses following an error [AN: 76.46%, controls: 74.24%; F(1, 34) = .40, p = .532] or of too late responses following an error [AN: 18.82%, controls: 14.49%; F(1, 34) = 3.25, p = .080] did not differ between the two groups.

3.3. ERP results

Fig. 2 shows the response-locked ERPs for correct and incorrect responses for the control and the AN group. For both groups an ERN is present after incorrect responses. Analyses showed that there was no difference in ERN amplitude between the control (−11.69 μV) and the AN group (−8.32 μV; F(1, 34) = 2.11, p = .156). However, as the use of the RT deadline did not provide similar error rates for the two groups, we additionally controlled for this difference in accuracy. Indeed, as can be seen in Fig. 3, ERN amplitudes correlated significantly with error rates in both groups: r = .432 (p = .042) for AN patients and r = .444 (p = .028) for controls. Therefore, the error rate was introduced as a covariate in the comparison of ERN amplitudes between groups, to correct for the significantly lower percentage of errors in the AN group. The ANCOVA for ERN amplitude, with error rate as a covariate, yielded a significant group effect [F(1, 33) = 7.793; p = .009], with a smaller ERN amplitude in the AN patients (−6.77 μV) than in the controls (−13.08 μV).

As BDI scores differed significantly between groups, Pearson correlation coefficients between BDI scores and ERN amplitudes were calculated. No significant correlations emerged: r = .442 (p = .076) for AN patients and r = −.176 (p = .472) for controls.

Importantly, an additional analysis demonstrated that the ERN effect was not caused by benzodiazepine use, as ERN amplitudes remained significantly smaller in AN patients than controls when patients using benzodiazepines (n = 4) were excluded from the analyses [ERN amplitude (non-benzodiazepine) AN = −7.45 μV; F(1, 29) = 4.370, p = .045].

Analyses of the stimulus-locked P3 component and the response-locked error positivity (Pe) did not reveal any significant main effects for group (P3: F(1, 34) = 1.87, p = .179; Pe: F < 1), nor any significant interactions with group (both Fs < 1).

3.4. Correlation analyses

The error rate did not correlate significantly with MPS-perfectionism neither in the control group (r = −.109, p = .328), nor in the AN group (r = .213, p = .206). Correlations between ERN amplitudes and MPS-overall perfectionism scores were significant for controls (r = −.430; p = .033), but not for AN patients (r = .109; p = .389).

4. Discussion

To our knowledge, the present study is the first to investigate the relation between perfectionism and (electrophysiological measures of) action monitoring in patients with AN. In line with previous studies, questionnaire data showed that restricting AN patients score higher than
controls on most dimensions of perfectionism. Patients committed less errors than controls, while no differences in RT emerged between the groups. Although questionnaire and behavioral outcomes provide evidence for an increased perfectionist response style in AN patients, these results do not coincide with enhanced ERN amplitudes. On the contrary, when ERN amplitudes are corrected for error rates by introducing this variable as a covariate in the comparison of ERN amplitudes between groups, the AN patients show significantly smaller ERN amplitudes than normal controls. In the correlation analyses, the expected correlation between MPS perfectionism and ERN amplitude emerged in the control group, but not in the AN group. As predicted, AN patients scored considerably higher on self-report measures of perfectionism, including the MPS subscale 'concerns over mistakes', which seems particularly relevant to action monitoring with three items specifically referring to the effect of mistakes on self and others. An increased perfectionist response style in the AN patients is also supported by the finding that patients committed significantly less errors than controls. While in previous studies the use of the RT deadline was successful in maintaining error rates comparable over groups or conditions (e.g. De Bruijn et al., 2006; De Bruijn et al., 2004), it seems more difficult to elicit errors in AN patients than in controls. Hence, the lower error rate likely reflects a more controlled
response style. Second, post hoc tests revealed that the significant three-way interaction between correctness, congruency and group was caused by the absence of a congruency effect for incorrect responses in the AN group. Fast incorrect responses to congruent stimuli specifically result from impulsive responding. Therefore, the absence of these fast incorrect responses in AN patients is indicative of their perfectionist response style.

The finding that the control group showed post-error slowing, while this performance adjustment is only marginally significant in the AN group, could be interpreted as a reflection of diminished action monitoring in AN patients. However, the proportion analyses of responses following errors contradicts such interpretation. Compared to controls, AN patients gave less erroneous responses following an incorrect response and also showed a tendency to respond more often too late immediately following an error.

No significant correlation between error rates and MPS perfectionism scores emerged in neither of the groups. This outcome reflects both the complexity of the (multidimensional) perfectionism concept (Dunkley, Blankstein, Masheb, & Grilo, 2006; Hewitt, Flett, Besser, Sherry, & McGee, 2003) and the difference between self-report measures, indicating how participants view themselves, and objective measures, reflecting behavior (Butler & Montgomery, 2005).

Error rates significantly correlated with ERN amplitudes in both groups, higher ERN amplitudes being associated with lower error rates, a finding that has been reported by others as well (see e.g. Santesso, Segalowitz, & Schmidt, 2005). A possible interpretation of this relationship is that subjects with a low error rate are more focused on optimal performance. Other analyses did not reveal a significant correlation between ERN amplitudes and BDI scores as a measure of depression in either group. Therefore, it seems unlikely that in the current experiment ERN amplitudes were affected by group differences in severity of depression.

The expected correlation between MPS perfectionism and ERN amplitude emerged in the control group, but not in the AN group. In the AN patients, their perfectionist trait, resulting in decreased error rates, could possibly be associated with a condition that counteracts the increased ERN amplitudes. This condition could for instance be related to the underweight state of the patients in our study. On a neural level, the finding of attenuated ERN amplitudes in AN patients, suggests that, during error detection, the ACC is less activated in patients with AN compared to controls. Although this interpretation may seem in conflict with increased perfectionism and a more controlled response style at first, it is in line with findings of neuroimaging studies in the resting condition in AN, suggesting hypoperfusion in the ACC (Delvenne et al., 1995; Kojima et al., 2005; Naruo et al., 2001; Takano et al., 2001). Similarly, Ferro et al. (2005) conclude from a single photon emission computed tomography (SPECT) study in AN patients during the execution of a Stroop interference task that their results are suggestive of a blunted cingulate function in AN patients in relation to executive function.

Hence, the current study suggests that hypoactivity of the ACC, as currently supported by reduced ERN amplitudes, does not necessarily lead to diminished task performance. A possible explanation for this intact performance may be related to recruitment of other brain areas in order to, for instance, increase cognitive control. This explanation of hyperactivity of alternative brain regions, is supported by at least one study in AN, which showed hypoperfusion of the ACC in combination with hyperperfusion in the amygdala–hippocampus complex (Takano et al., 2001). With respect to healthy subjects, Colcombe et al. (2004) recently demonstrated in an fMRI study using a similar flankers task that elderly healthy subjects with higher fitness showed an increased functioning of the frontal and parietal network. Importantly, this increased recruitment in frontal and parietal regions, associated with attentional selection and response conflict resolution, was associated with a reduced activity in the ACC. An advantage of activating the frontal and parietal network may be that errors are better prevented, so that there is less need for fast adaptive actions issued by the ACC.

With respect to the ERP outcomes, the fact that part of the AN group was medicated should be mentioned as a limitation of the present study. The benzodiazepines (half-life values under 12 h) and SSRIIs (half-life values under 28 h) were omitted the day before testing, meaning that no sleep inductor was taken within at least 36 h of testing. However, we acknowledge that this medication-free period may not rule out possible influences of medication entirely. Previous research has shown that psychopharmacological compounds may affect ERP correlates of action monitoring. While administration of a benzodiazepine to healthy volunteers caused attenuation of ERN amplitudes along with a slowing of responses, an SSRI neither affected ERN amplitudes nor behavioral or other ERP measures (De Bruijn et al., 2004; De Bruijn, Sabbé, Hulstijn, Ruitert, & Verkes, 2006). Therefore, we additionally analyzed the subset of AN patients who were not taking a benzodiazepine. This analysis demonstrated that the ERN amplitudes were also reduced for the benzodiazepine-free subset of patients. As a result, it seems unlikely that the current ERP results were affected by psychotropic drugs.

Another limitation of the present study might be the absence of data that enable us to provide the exact amount of error corrections, as we only recorded the first response during the experiment. While the question whether error correction affects the ERN is still a matter of debate (see e.g. Fiehler, Ullsperger, & Von Cramon, 2005; Rodriguez-Fornells, Kurzbuch, & Münte, 2002), we believe that the influence of error-correction related processes was limited or non-existent in the current study. Correcting was not an option in the present experiment. Participants were instructed that a corrective response would serve no purpose as it would always be disregarded. Moreover, this was always confirmed by the trial-to-trial feedback indicating
whether the given response was correct, incorrect, or too late. As a result, participants almost never corrected their erroneous responses, thus severely limiting any possible effects on the current measures of interest.

Further research seems warranted to elucidate the possible role of altered action monitoring in the development of AN. Direct comparison of patients with different types of eating disorders thought to vary in perfectionism and impulsivity, could shed further light on the role of action-monitoring processes in the psychopathology of eating disorders. Neuroimaging studies in AN patients during the speeded-choice reaction task used in the present study could confirm the hypothesized ACC hypofunctioning with increased functioning in prefrontal and parietal cortical regions of the attentional network of the brain.

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