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Research Paper

Electrophysiological correlates of performance monitoring under social observation in patients with social anxiety disorder and healthy controls

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ABSTRACT

Previous research suggests that electrophysiological correlates of performance monitoring, in particular the error-related negativity (ERN), vary according to psychopathology and context factors. The present study examined the effect of social context on behavioral and electrophysiological correlates of performance monitoring in healthy adult subjects and in patients with social anxiety disorder (SAD). Participants performed two runs of a Go/NoGo flanker task in different social conditions: in the observation condition, they were observed by a confederate while performing the task, whereas there was no observation in the control condition. Behavioral data showed that accuracy and response times were not modulated by social observation and also did not systematically differ between groups. Post-error slowing was more pronounced in patients, independent of observation condition. ERN amplitudes were generally increased under social observation as compared to the control condition regardless of group (patients, controls). No effects of social context or group were found for PE, NoGo-N2, and NoGo-P3. Exploratory analysis revealed a late sustained parietal negativity to errors in patients as compared to controls. Taken together, the present findings emphasize the importance of social context for the processes underlying performance monitoring. However, the notion of altered error monitoring reflected in an altered ERN in SAD is not supported by our data.

1. Introduction

Monitoring of ongoing behavior is a central function of the human central nervous system. It allows for constant adaptation to current demands and outcome optimization. Performance monitoring processes are of particular importance in social interactions, which are often complex and require flexible adaptation of one's own behavior.

Among the electrophysiological correlates of performance monitoring, the error-related negativity (ERN, Gehring, Goss, Coles, Meyer, & Donchin, 1993) or error negativity (Ne, Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991) has been studied most extensively. The ERN is a negative deflection in the event-related potential (ERP) occurring approximately 50–100 ms after commission of a performance error. The ERN is most prominent at frontocentral electrodes and most likely generated in anterior midcingulate cortex (aMCC, Debener et al., 2005). A comparable albeit smaller negative deflection in the response-locked ERP is also observed following correct responses and has been appropriately termed "correct-related negativity" (CRN) (Ford, 1999). Its neural generator has been localized in MCC (Debener et al., 2005; Ullsperger,Fischer, Nigbur, & Endrass, 2015). While ERN and CRN reflect fast efference copy-based error or conflict detection (Gehring et al., 1993; van Veen & Carter, 2002a; Botvinick, Braver, Barch, Carter, & Cohen, 2004), the error positivity (Pe), a relative parietal positivity occurring approximately 200–500 ms post-response, has been linked to more cognitive aspects of error processing and specifically error awareness (Falkenstein et al., 1991; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Olvet & Hajcak, 2008; Nieuwenhuis, Ridderinkhof, BLOM, BAND, & K.O.K.A., 2001; Endrass, Reuter, & Kathmann, 2007).

A number of studies have demonstrated that electrophysiological correlates of performance monitoring, most notably the ERN, vary depending to factors such as personality (Luu, Collins, & Tucker, 2000; Pailing & Segalowitz, 2004) or motivational significance of an error (Hajcak, Moser, Yeung, Simons, 2005; Endrass et al., 2010; Potts, 2011). With regard to social context, increased ERN amplitudes were

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found when performance was evaluated by a confederate (Hajcak et al., 2005) or when participants believed they were competing with one another (van Meel and van Heijningen, 2010), suggesting the significance of errors was increased in social situations. In a recent study, Masaki, Maruo, Meyer, and Hajcak, (2017) found that the ERN was relatively larger when athletes who reported high levels of performance-related anxiety were evaluated during a Stroop task compared to a non-social control condition. While the functional role of the ERN remains a matter of ongoing debate, these empirical findings support the general notion that the ERN is affected by motivational factors and the subjective value of errors (Hajcak et al., 2005; Proudfit, Inzlicht, & Mennin, 2005). In addition, ERN may reflect the individual salience of errors (Riesel , Weinberg , Endrass , Kathmann , & Hajcak, 2013). Still, it remains unclear to what extent ERN is modulated by state or trait-like characteristics (Moser, Moran, Schroder, Donnellan, & Yeung, 2013; Proudfit et al., 2013).

In light of these findings, the interplay of personality variables and social context may be of particular interest in psychopathological conditions such as social anxiety disorder (SAD, DSM-V, American Psychiatric Association). SAD patients fear and tend to avoid being the focus of attention, assuming that their behavior may be judged as inappropriate or embarrassing by others. In particular, being under scrutiny in performance situations such as presentations or interviews can lead to significant distress and feelings of anxiety. Cognitive models claim that SAD symptomatology is associated with negative information processing biases in social situations that include general negative beliefs about oneself and/or the consequences of one's own performance as well as exaggerated standards for one's own behavior (Clark & Wells, 1995; Rapee & Heimberg, 1997). In their model, Clark and Wells (1995) emphasize the role of internal monitoring processes for SAD. In potentially threatening social situations, SAD patients shift their attention to detailed monitoring and observation of themselves and their performance. This information is then used to predict other people's thoughts and judgment. With regard to such elevated attention to both one's own overt behavior as well as internal processes in SAD, several studies have examined alterations in the performance monitoring system in SAD.

Increased ERN amplitudes in SAD patients relative to controls have been reported by Endrass, Riesel, Kathmann, and Buhlmann, (2014). Kujawa et al. (2016) found that increased ERN in youth and young adults with SAD persisted even after treatment. In a study on the relationship between behavioral inhibition (BI) and performance monitoring processes, Lahat et al. (2014) reported that children high in BI, compared to those low in BI, displayed increased ERN amplitudes, which also predicted risk for later social phobia symptoms. In a recent study, the authors reported that in the same sample early-life BI temperament predicted later hypersensitivity toward errors, but this relationship depended on social context (Buzzell et al., 2017).

Barker, Troller-Renfree, Pine, and Fox, (2015) extended these findings by comparing high (HSA) with low socially anxious subjects (LSA). Importantly, in this study, social context was manipulated in that subjects performed the task either alone or while being observed by a confederate. Results showed an interaction between social anxiety and social context. The ERN was enhanced under observation only in HSA, and the magnitude of this effect correlated with individual differences in social anxiety. These findings thus suggest hyperactive performance monitoring in social anxiety as a function of social context. However, data on other ERP measures of performance monitoring in SAD, that is, CRN and Pe, are rather inconclusive (Barker et al., 2015; Endrass et al., 2014; Riesel, Goldhahn, & Kathmann, 2017). Moreover, these results cannot inform about potential modulation of performance monitoring by social context in SAD, given that Barker et al. (2015) tested extreme groups but did not confirm psychiatric diagnoses. It has been suggested that social anxiety is represented on a continuum ranging from subclinical behavior (e.g., shyness) to clinical manifestation (SAD) based on common underlying dysfunctional mechanisms (Stein, Torgrud, & Walker, 2000).

In view of this, the aim of the present study was to examine the effect of social context on ERN, CRN and Pe in a sample of healthy subjects and in patients with confirmed SAD diagnosis. Participants performed a variant of the Eriksen Flanker Task (Eriksen & Eriksen, 1974), a well-established task of interference control that reliably elicits an ERN. The present task variant was inspired by a previous study (Beste et al., 2013) in that it incorporated NoGo trials in order to allow investigation of response inhibition-related monitoring processes. To this end, NoGo-N2 and NoGo-P3 were analyzed to elucidate the impact of social context on response inhibition in patients with SAD. Due to similar scalp topographies and temporal courses of stimulus-locked N2 and response-locked ERN, it has been proposed that these two components may reflect activity of the same underlying performance monitoring system (Ferdinand, Mecklinger, & Kray, 2008; Folstein & van Petten, 2008; van Veen & Carter, 2002b).

Consistent with this notion, some recent studies suggest that the N2 may be subject to alterations in anxiety disorders that are comparable to those found in the ERN. As an example, Cavanagh, Meyer, and Hajcak, (2017) found both ERN and N2 to be increased in generalized anxiety disorder (GAD), and Riesel, Klawohn, Kathmann, and Endrass, (2017) reported enhanced N2 amplitudes in obsessive compulsive disorder. Furthermore, increased N2 has been linked to social reticence and other social anxiety symptoms in children (Lamm et al., 2014; Thai, Taber-Thomas, & Pérez-Edgar, 2016). To our knowledge, however, the present study is the first to analyze both ERN and N2 in a sample of adults with a diagnosis of SAD.

Similar to the procedure employed by Barker et al. (2015), subjects performed the task in two distinct conditions. In the *observation condition*, a confederate observed participants both directly and by means of a camera. In the *control condition*, they performed the task without being observed. It was hypothesized that ERP measures of performance monitoring should be increased in SAD, an effect expected to be augmented by social observation. Specifically, ERN (but not CRN) and Pe were expected to be increased in SAD particularly under observation. Investigation of NoGo-N2 and NoGo-P3 was more explorative and we thus did not have specific hypothesis with regard to modulation of these components by social anxiety and social context. On the behavioral level, we expected greater performance accuracy accompanied by slower reaction times under observation, due to more cautious response behavior or increased effects on vigilance and/or arousal under observation (Zajonc, 1965; Bond & Titus, 1983). We expected these effects to be more pronounced in SAD.

2. Materials and methods

2.1. Participants

22 patients with social anxiety disorder (mean age: 24.73 ± 2.57 years; 13 females) and 22 healthy adult volunteers (mean age: 23.00 ± 4.94 years; 15 females) were recruited at the Institute of Medical Psychology and Systems Neuroscience of the University Hospital of Muenster. Groups were matched according to age, gender, intelligence and years of education (see Table 1). All subjects had normal or corrected-to-normal vision. Exclusion criteria for all participants were psychotic, substance-related or neurological disorders, in particular a history of seizures or head injury with loss of consciousness, and severe uncontrollable medical conditions potentially influencing neurocognitive function.

The current diagnostic status of patients was assessed by an experienced clinical psychologist using the German translation of the Structured Clinical Interview for DSM-IV (SKID-I, Wittchen, 1997). None of the participants fulfilled the criteria for a diagnosis of a current episode of major depression, psychotic disorder, obsessive compulsive disorder, general anxiety disorder, eating disorder or substance abuse according to the SCID. Five participants (all in the SAD group) reported

Table 1
Sociodemographic and clinical characteristics of patients and healthy control subjects. SD = standard deviation. *Discomfort rated on a 9-point Likert scale.

Variable	Controls (n = 22)	Patients (n = 22)	Difference	
Demographics	Mean (SD)	Mean (SD)		
Age	23.00(± 4.94)	24.73 (± 2.57)	$t(42) = -1.60$	$p = 0.153$
Gender (male:female)	7:15	9:13	$\chi^2 = 0.393$	$p = 0.531$
Years of education	12.50(± 0.60)	12.68 (± 1.04)	$t(42) = -0.170$	$p = 0.481$
IQ (MWT-B)	111.05 (± 11.7)	115.14 (± 10.82)	$t(42) = -1.206$	$p = 0.235$
Clinical characteristics				
BDI	3.82(± 4.63)	12.68 (± 9.17)	$t(42) = -4.047$	$p < 0.001$
LSAS	20.5(± 15.90)	68.10 (± 20.09)	$t(42) = -8.714$	$p < 0.001$
SPS	8.00(± 7.89)	29.91 (± 12.15)	$t(42) = -7.091$	$p < 0.001$
SIAS	11.09(± 10.09)	43.00 (± 12.62)	$t(42) = -9.262$	$p < 0.001$
Discomfort during observation*	4.09 (± 2.65)	6.23 (± 1.82)	$t(42) = -3.112$	$p = 0.003$

previous episodes of major depressive disorder. 3 of the patients received psychopharmacological treatment (all with citalopram) and 2 were in cognitive behavioral treatment. All participants completed the Beck-Depression Inventory (BDI-II, Beck, Steer, & Brown, 2006), the Social Phobia Scale (SPS) and the Social Interaction Anxiety Scale (SIAS) (Mattick & Clarke, 1998) and the self-report version of the Liebowitz Social Anxiety Scale (LSAS, Liebowitz, 1987). All participants gave written informed consent to the study. The study adhered to the guidelines of ethical standards in the Declaration of Helsinki and was approved by the Ethics Committee of the German Psychological Society (Deutsche Gesellschaft für Psychologie, DGPs). Subjects received either monetary reimbursement or course credits for participation. An overview of sociodemographic data is given in Table 1.

2.2. Procedure

Participants were informed that the experiment involved a computerized reaction time task and that during either the first or second run of the task they would be observed by an experienced psychologist both directly and by means of a camera. After informed consent was obtained, and after demographic information was collected, the electrodes were attached and the experimental task was started. Participants were seated in a dimly-lit room at a viewing distance of approximately 80 cm from a computer screen. A webcam was placed on top of the computer screen and oriented towards the subject. After each run, participants were asked to complete a short questionnaire to determine their arousal level and feelings of unpleasantness related to errors and correct responses in the previous run on 9-point Likert scales. The experimental task lasted 20 min. Including EEG preparations, the entire test session lasted approximately 120 min.

2.3. Experimental task

The experimental task was an adaptation of a modified speeded flanker task (Eriksen & Eriksen, 1974). This task variant was inspired by a previous study, which incorporated NoGo trials on which participants were asked to withhold responses (Beste et al., 2013). Vertically arranged arrowheads were presented above or below a central target stimulus (arrowhead or circle). Target arrowheads pointed either to the same (compatible) or opposite (incompatible) direction as the target. Subjects were instructed to indicate the direction of the target arrowheads by responding with their left or right index finger. If they failed to respond within 600 ms after target onset, an auditory signal was presented (1000 Hz, 60 dB SPL). If the central target was a circle, subjects were instructed to withhold any overt response (NoGo condition). Flankers appeared 200 ms before target onset to increase task difficulty and error likelihood. Target stimuli were then displayed for 300 ms and switched off simultaneously with the flankers. The mean response–stimulus interval was 1100 ms and jittered between 900 and 1300 ms. Stimulus presentation was controlled by Presentation software (Neurobehavioral Systems Inc, Berkeley, CA, USA).

Importantly, participants performed two runs of the task in different social conditions (observation/control). The order of conditions was counterbalanced across participants. An illustration of the experimental setup in the observation condition as well as well as examples of the different trial types in the combined NoGo-Flanker task are given in

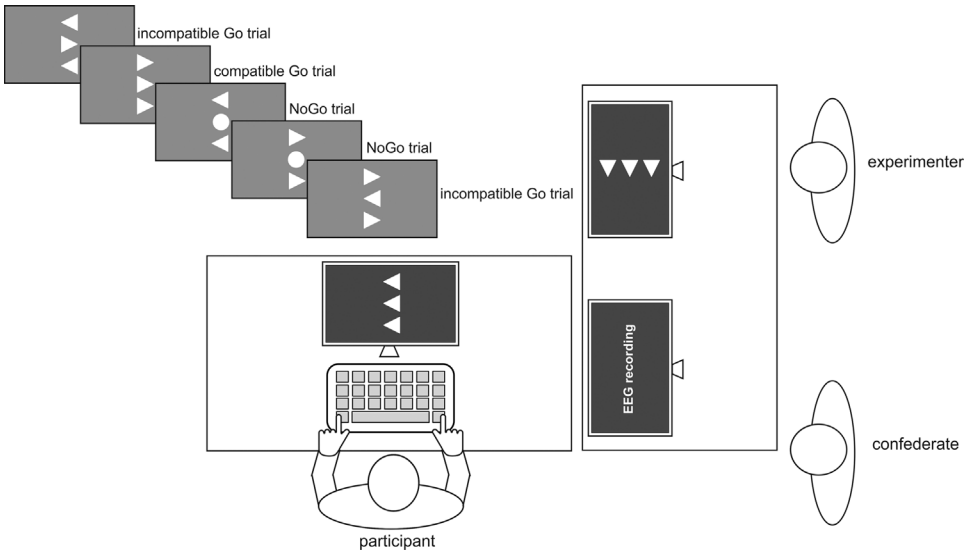

Fig. 1. Illustration of the experimental set-up in the observation condition as well as examples of the different trial types in the combined NoGo-Flanker task.

Fig. 1. Each run involved a total of 288 compatible, 96 incompatible and 96 NoGo trials, amounting to a total of 480 trials, which were presented in randomized order. In the observation condition, subjects were informed that a trained psychologist would observe them during the run. At the beginning of the observation condition, a confederate dressed in a white lab coat entered the room, the experimenter turned on the webcam, and patients were shown a live video feed of themselves while seated in front of the computer screen. They were informed that the confederate would observe them constantly during the run (both directly and by means of the camera livestream), but would focus on their performance in the task and on erroneous responses in particular. Every time the participant made an error, the confederate would take a note on a chart. The experimenter then started the task and gave general task instructions. After completion of the run, the confederate briefly thanked the participant and left the room. Participants were then asked to determine how uncomfortable they felt being observed on a Likert scale from 1 (not uncomfortable at all) to 9 (extremely uncomfortable). In the non-observation condition, the camera was (or remained) turned off and the experimenter was seated behind a curtain to avert eye contact, but remained in the room to ensure undisturbed execution of the procedure. The experimenter informed subjects that they would not be observed during this run.

2.4. EEG data acquisition, preprocessing and analysis

2.4.1. Psychophysiological recordings

EEG was recorded from 64 scalp sites using a BioSemi AD-box (BioSemi B.V., Amsterdam, Netherlands) and ActiView software at a sampling rate of 512 Hz. The BioSemi system uses a CMS/DRL feedback loop with two additional electrodes instead of ground and reference (see http://www.biosemi.com/faq/cms&drl.htm). Active Ag-AgCl electrodes were fitted to an elastic cap according to the 10–20 system (FP1, FT9, AF3, F1, F3, F5, F7, FT7, TP9, FC3, FC1, C1, C3, C5, T7, TP7, PO9, CP3, CP1, P1, P3, O9, P7, P9, PO7, PO3, O1, Iz, Oz, POz, Pz, CPz, FPz, FP2, FT10, AF4, AFz, Fz, F2, F4, F6, F8, FT8, TP10, FC4, FC2, FCz, Cz, C2, C4, C6, T8, TP8, PO10, CP4, CP2, P2, P4, O10, P8, P10, PO8, PO4, and O2). Horizontal (HEOG) and vertical eye movement (VEOG) was tracked with additional electrodes, which were attached to the left and right canthi and above and below the right eye, respectively.

EEG-data were analyzed off-line using BrainVision Analyzer 2 software (Brain Products, Munich, Germany) and MATLAB (Mathworks, Natick, Massachusetts, USA). Initially, 0.1 Hz high-pass and 30 Hz low-pass filters were applied to the raw data. Ocular correction was performed based on HEOG and VEOG channels according to the Gratton & Coles algorithm implemented in BrainVision Analyzer 2 software. ERP segments were created ranging from 200 ms before to 800 ms after the response. Baseline correction was performed based on the average signal in the 200 ms directly preceding the response. Segments containing maximum amplitudes exceeding absolute values of 100 μV or a voltage step of 50 μV were excluded by means of automatic artefact detection. Trials were pooled and averaged according to condition (observation/control) and response type (correct, error, NoGo). For NoGo trials, only correct trials, i.e. trials without button press response, were included. Analyzed ERP components included ERN, Pe, NoGo-N2 and NoGo-P3. ERN amplitudes were scored as peak-to-peak difference between the maximum negative peak occurring 0–100 ms after the erroneous response in the individual average ERP waveform and the preceding maximum positive peak occurring in a time window 80 ms just before the response at electrode FCz (Gentsch, Ullsperger, & Ullsperger, 2009). An analogous procedure was used to determine CRN on correct trials. In accordance with Olvet and Hajcak (2009) a minimum of 6 error trials per condition was needed for inclusion in the analysis. Due to an insufficient number of NoGo errors in some subjects, only errors committed in Go trials (but not false alarms in NoGo trials) were included.

Pe, NoGo-N2 and NoGo-P3 were defined following the procedures applied in previous studies (e.g., Falkenstein et al., 2000; Gehring et al, 1993; Beste et al., 2013). The Pe was defined as mean amplitude in the difference signals (error − correct) in the time window 300–500 ms post-response at electrode Pz, the NoGo-N2 as maximum negative peak from 200 to 350 ms after target onset at electrode FCz, and the NoGo-P3 as maximum positive peak occurring from 300 to 600 ms after stimulus onset at FCz.

2.5. Statistical analysis

All behavioral and EEG data were analyzed using SPSS (IBM SPSS Statistics 22, IBM Corp., Armonk, New York, USA). Accuracy ratings were analyzed for errors, misses and false alarms by means of separate repeated-measures analyses of variance (ANOVAs) with condition (observation, control) as within-subjects factor and group (SAD, HC) as between-subjects factor. Response times (RT) for Go trials were analyzed by means of a repeated-measures analysis of variance (ANOVA) with response type (error, correct) and condition (observation, control) as within-subjects factor and group (SAD, HC) as between-subjects factor. Post-error slowing was defined as the difference between reaction time on correct trials following an error minus the reaction time on correct trials following another correct response. Post-error slowing and reaction time on false alarms were calculated using 2 × 2 ANOVAS with condition as within-subjects factor and group as between-subjects factor, respectively. Data obtained from the subjective ratings questionnaire were analyzed by means of separate repeated-measures ANOVAs with condition (observation, control) as within-subjects factor and group (SAD, HC) as between-subjects factor.

ERN and Pe were investigated using separate 2 × 2 × 2 repeated-measures ANOVAS with condition and response type as within-subjects factors and group as a between-subjects factor. NoGo-N2 and NoGo-P3 amplitudes in observation and control condition were analyzed by means of 2 × 2 ANOVAs with condition as within-subjects factor and group as between-subjects factor. Significance was set to $p < 0.05$ (one-tailed). Greenhouse-Geisser correction was applied if the assumption of sphericity was violated. Post-hoc *t*-tests were performed to resolve interactions, results are reported one-sided.

3. Results

3.1. Demographic and clinical data

Table 1 shows demographic and clinical data for SAD patients and HC. Groups did not differ regarding age ($p = 0.153$), gender ($p = 0.531$), educational level ($p = 0.481$), and general intelligence ($p = 0.235$). SAD patients reported a higher number of depressive symptoms (BDI, $t(42) = -4.047$, $p < 0.001$) and scored significantly higher on various measures of social anxiety such as LSAS ($t(42) = -8.714$, $p < 0.001$), SPS ($t(42) = -7.091$, $p < 0.001$) and SIAS ($t(42) = -9.262$, $p < 0.001$). Patients also reported more intense feelings of discomfort during the observation condition of the experimental task ($t(42) = -3.112$, $p = 0.003$).

3.2. Behavioral data

Accuracy (mean percentages of correct responses, errors, and false alarms) and mean reaction times according to group (SAD/HC) and condition (observation/control) are provided in Table 2. Accuracy was similar across groups and conditions. For errors and false alarms, repeated-measures ANOVAs showed no significant main or interaction effects (all $ps > 0.470$). For misses, a trend towards significance was found for the group by condition interaction ($F_{1,42} = 3.0$, $p = 0.091$). All other effects failed to reach significance (all $ps > 0.454$).

For reaction times on Go trials, a significant main effect of response type emerged, with participants responding faster on error trials than on correct trials ($F_{1,42} = 479.332$, $p < 0.001$, $\eta_p^2 = 0.929$). Analysis

Table 2
Accuracies, reaction times and ERP measures for patients and controls in the observation and control conditions. SD = standard deviation. PE-slowing = post-error-slowing.

Variable	Controls (n = 22)		Patients (n = 22)	
	Observation	Control	Observation	Control
Behavior	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Correct (%)	88.86 (± 5.72)	88.80 (± 5.92)	87.48 (± 5.29)	87.09 (± 5.56)
Go Error (%)	7.19 (± 3.73)	7.82 (± 4.14)	8.26 (± 3.84)	8.29 (± 3.63)
Miss (%)	3.95 (± 3.40)	3.38 (± 2.73)	4.26 (± 3.19)	4.61 (± 3.35)
NoGo False alarm (%)	11.68 (± 11.26)	11.81 (± 10.93)	12.79 (± 10.06)	13.51 (± 9.27)
Correct (ms)	300.21 (± 22.23)	301.39 (± 19.01)	297.71 (± 16.42)	301.26 (± 16.40)
Go Error (ms)	265.56 (± 33.46)	264.26 (± 28.70)	255.15 (± 24.02)	262.39 (± 20.78)
PE-slowing (ms)	−2.67(± 14.96)	−0.08 (± 24.82)	18.93(± 18.71)	10.71(± 20.65)
NoGo False alarm (ms)	222.92 (± 61.48)	247.57 (± 29.20)	226.61 (± 20.58)	226.27 (± 19.16)
ERP				
ERN (μV)	−12.47 (± 4.19)	−11.13 (± 5.89)	−12.76 (± 6.69)	−10.69 (± 6.55)
CRN (μV)	−5.01 (± 2.88)	−5.07 (± 2.78)	−3.96 (± 1.83)	−4.83 (± 2.12)
Pe error (μV)	0.02 (± 2.32)	0.51 (± 1.27)	0.25 (± 1.26)	0.03 (± 1.55)
Pe correct (μV)	−0.74 (± 1.43)	−0.53 (± 1.12)	−0.57 (± 1.06)	−0.68 (± 0.86)
NoGo-N2 (μV)	−8.18 (± 4.61)	−8.51 (± 4.71)	−7.26 (± 4.82)	−7.76 (± 5.66)
NoGo P3 (μV)	6.71 (± 3.61)	6.31 (± 4.07)	6.75 (± 4.49)	7.19 (± 4.46)

of post-error slowing demonstrated a main effect of group ($F_{1,42}$ = 7.934, p = 0.007, η_p^2 = 0.159), indicating that SAD patients but not HC responded more slowly after an erroneous response, irrespective of condition.

False alarm reaction times showed a significant main effect of condition ($F_{1,41}$ = 4.081, p = 0.050, η_p^2 = 0.091) that was further qualified by a significant condition by group interaction ($F_{1,41}$ = 4.148, p = 0.048, η_p^2 = 0.092). Post-hoc *t*-tests revealed that on false alarm trials, HC responded faster in the observation condition as compared to the control condition (t (21) = −2.215, p = 0.038), while no difference was found for SAD patients (p = 0.982).

3.3. Subjective ratings

Table 3 gives an overview of subjective ratings of unpleasantness and arousal level for errors and correct responses for patients and controls in the observation and control condition. Significant main effects of group emerged for unpleasantness of errors ($F_{1,42}$ = 13.995, p = 0.001, η_p^2 = 0.250) and correct responses ($F_{1,42}$ = 14.380, p < 0.001, η_p^2 = 0.255), and for arousal related to errors ($F_{1,42}$ = 14.465, p < 0.001, η_p^2 = 0.256) and correct responses ($F_{1,42}$ = 9.770, p = 0.003, η_p^2 = 0.193), with SAD patients scoring higher on each of these measures relative to HC. Beyond this, a main effect of condition indicated higher arousal during errors in the observation condition, irrespective of group ($F_{1,42}$ = 17.443, p < 0.001, η_p^2 = 0.293). No further main or interaction effects were found (all ps > 0.227)

Table 3
Subjective ratings of unpleasantness and arousal level for errors and correct responses for patients and controls in the observation and control conditions. All measures rated on a 9-point Likert scale. SD = standard deviation.

Variable	Controls (n = 22)		Patients (n = 22)	
	Observation	Control	Observation	Control
Rating	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Unpleasantness of errors	5.18 (2.24)	4.86 (2.25)	7.00 (1.71)	6.91 (1.34)
Unpleasantness of correct responses	.95 (.21)	.95 (.21)	1.91 (1.60)	1.59 (1.26)
Arousal level on error trials	5.50 (2.15)	4.91 (2.16)	7.5 (.96)	6.68 (1.36)
Arousal level on correct trials	3.50 (1.92)	3.59 (2.20)	5.32 (1.49)	4.72 (1.58)

3.4. ERP data

Fig. 2 shows response-locked grand-average waveforms for correct responses and errors at electrode FCz and scalp topographies for the time point of post-response peak negativity (reflecting ERN and CRN) according to condition (observation/control) and group (SAD/HC). Table 2 provides mean amplitudes for all ERP measures according to group and condition. Statistical analysis of ERN/CRN data revealed a main effect of response type. Across both groups and both conditions, ERN amplitudes (i.e., neural responses to errors) were more negative compared to CRN amplitudes (i.e., neural responses to correct responses; $F_{1,42}$ = 80.738, p < 0.001, η_p^2 = 0.658). In addition, this effect was qualified by a significant response type by condition interaction ($F_{1,42}$ = 5.306, p = 0.026, η_p^2 = 0.112). Separate post-hoc *t*-tests for ERN and CRN showed that ERN amplitudes were increased in the observation as compared to the control condition (t (43) = −1.856, p = 0.035). CRN amplitudes, however, tended to be reduced in the observation condition, but this effect did not reach statistical significance (p = 0.058). Importantly, we found no group differences for ERN/CRN amplitudes ($F_{1,42}$ = 0.164, p = 0.687) and no further interaction effects (all ps > 0.222). When entering BDI scores as a covariate in the ANOVA in an attempt to statistically correct for group differences in depressiveness, the condition by response type interaction was no longer significant ($F_{1,41}$ = 0.918, p = 0.341). To validate the results of the peak-to-peak- analysis, ERN and CRN were also calculated as mean amplitudes in the time window 0–100 ms post-response. The repeated-measures ANOVA yielded similar results to the respective peak-to-peak analysis. Most importantly, a significant response type by condition interaction emerged ($F_{1,42}$ = 5.163, p = 0.028, η_p^2 = 0.109).

Analysis of relationships between ERN/CRN and clinical measures revealed no significant correlations. For the relationship between ERN in the observation condition and BDI scores, a trend towards significance emerged (r = −0.29, p = 0.06). For exploratory purposes, correlations between BDI, LSAS, SPS and SIAS scores with the "social effect ERN" (social ERN minus non-social ERN) were calculated both across all subjects and separately for SAD and HC. However, none of the correlations reached statistical significance (all ps > 0.135).

Fig. 3 presents response-locked grand-average waveforms for errors and correct responses according to condition at electrode Pz for SAD patients and HC. Analysis yielded a significant main effect of response type, with Pe amplitudes increased (i.e., more positive) for errors as compared to correct responses ($F_{1,42}$ = 23.773, p < 0.001,

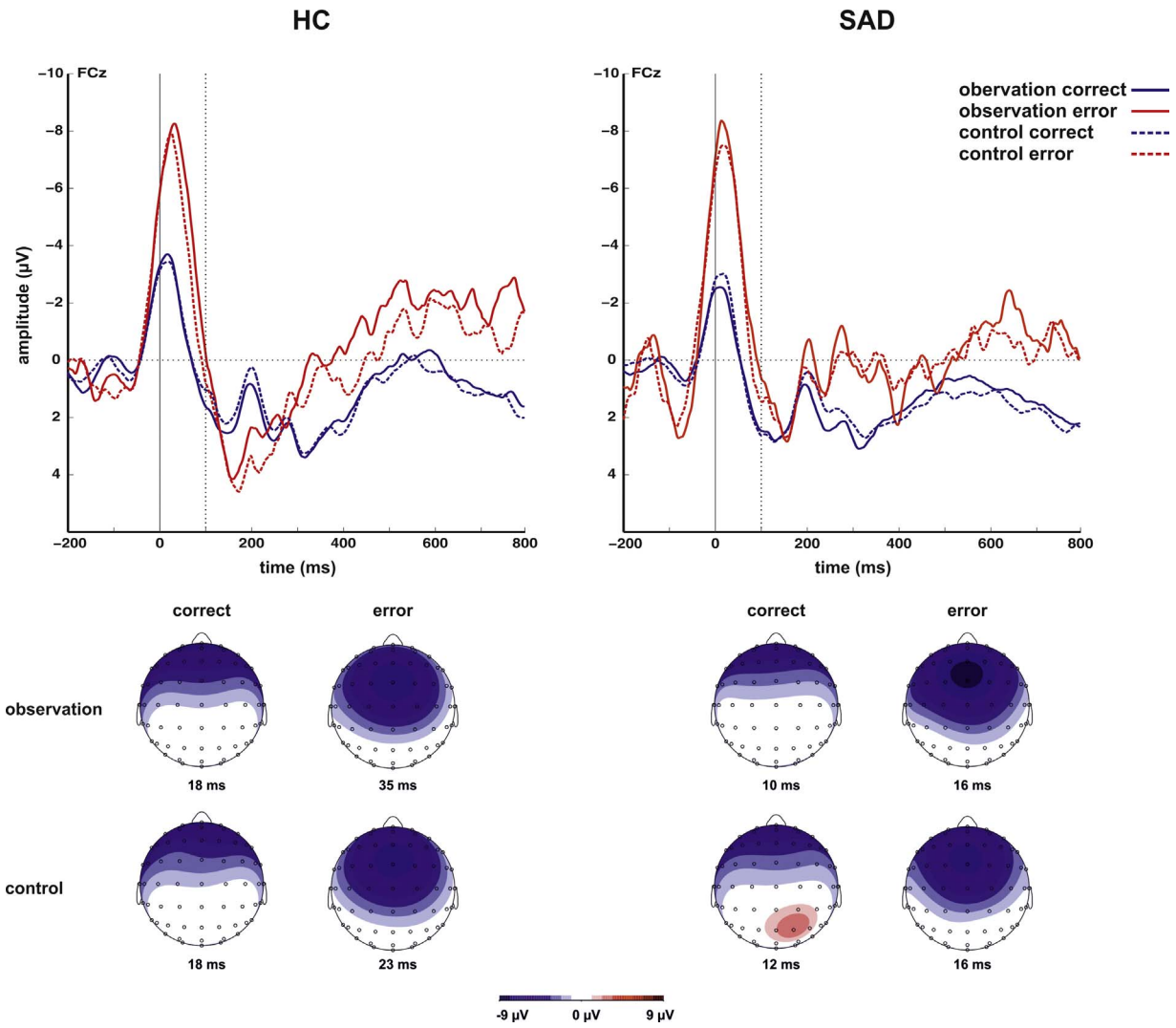

Fig. 2. Response-locked original waveforms (top) and corresponding scalp topographies (bottom) at electrode FCz for correct (CRN) and error (ERN) responses in the observation and control condition for healthy control subjects (left) and SAD patients (right).

η_p^2 = 0.361). No other significant main or interaction effects emerged (all p > 0.118). However, visual inspection of the grand-average waveforms indicated potential group differences in neural responses to errors versus correct responses at Pz in a later time window approximately from 600 to 800 ms post-response (see Fig. 3). It is conceivable that processing in such late time windows may link to performance of the subsequent trial, and specifically to response execution on subsequent trial and thereby possibly post-error slowing. In an additional exploratory analysis, we analyzed the average amplitude at Pz in the time window between 600 ms to 800 ms post-response by means of repeated-measures ANOVA with response type (error, correct) and condition (observation, control) as within-subjects factors and group (SAD, HC) as between-subjects factor. The ANOVA yielded a significant response type by group interaction ($F_{1,42}$ = 4.437, p = 0.041, η_p^2 = 0.096). Separate post-hoc paired-sample t-tests showed that post-error amplitudes compared to post-correct amplitudes were significantly increased in patients (t (21) = −2.131, p = 0.045), but not in HC (p = 0.376). The magnitude of this effect was not correlated to post-error slowing (p = 0.308). No other main or interaction effects emerged (all p > 0.344).

Fig. 3 shows target-locked grand-average waveforms on NoGo trials for SAD patients and HC in the observation and the control condition. Repeated-measures ANOVAs yielded no significant main or interaction effects for either NoGo-N2 (all p > 0.316) or NoGo-P3 (all p > 0.374).(Fig. 4)

4. Discussion

The aim of the present study was to examine performance monitoring processes in healthy subjects and SAD patients as a function of the social context. Specifically, we explored whether electrophysiological measures of performance monitoring such as the ERN were enhanced under social observation, if social observation was associated with more cautious responding on the behavioral level (e.g. increased reaction times and decreased error rates), and if these effects were further modulated by psychopathology.

Results showed that social context did indeed affect neural processes underlying performance monitoring. Social observation was associated with increased negativity in response to errors (ERN) but not to correct responses (CRN). Pe, NoGo-N2 and NoGo-P3 were unaffected by social observation. With regard to the lack of modulation in NoGo-N2 and NoGo-P3, one can speculate that only overt behavior, that is any behavior that can actually be registered and evaluated by an outside observer, is increased by social observation.

With respect to the ERN, however, our study is in line with previous empirical findings and supports the notion that the ERN is modulated by contextual factors (e.g. Hajcak et al., 2005; Fishman & Ng, 2013; Jackson, Nelson, & Proudfit, 2015). However, contrary to our

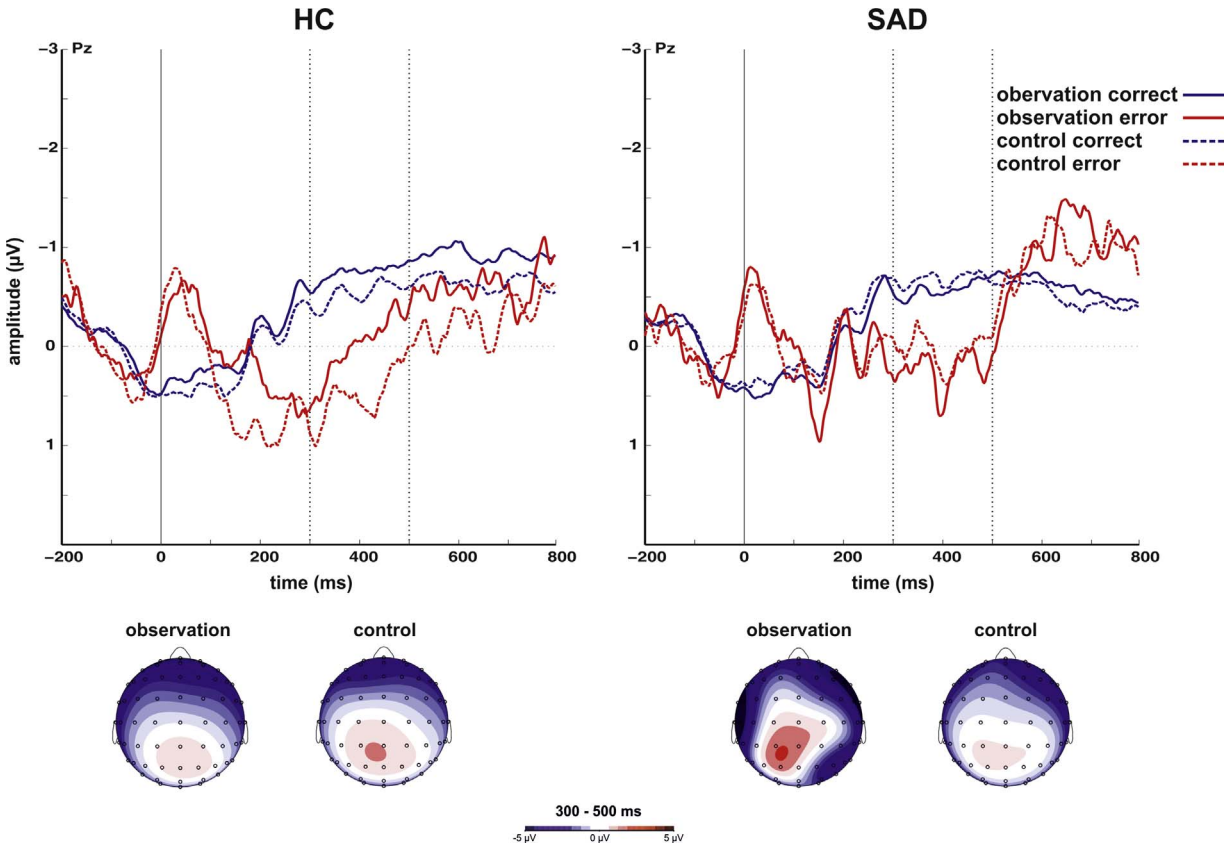

Fig. 3. Top panel shows response-locked original waveforms at electrode Pz for correct responses and errors according to condition (observation/control) and group (control subjects (left), SAD patients (right)). Bottom panel shows scalp distributions of the Pe as derived from the difference signal (error – correct) according condition (observation/control) and group (control subjects (left), SAD patients (right)).

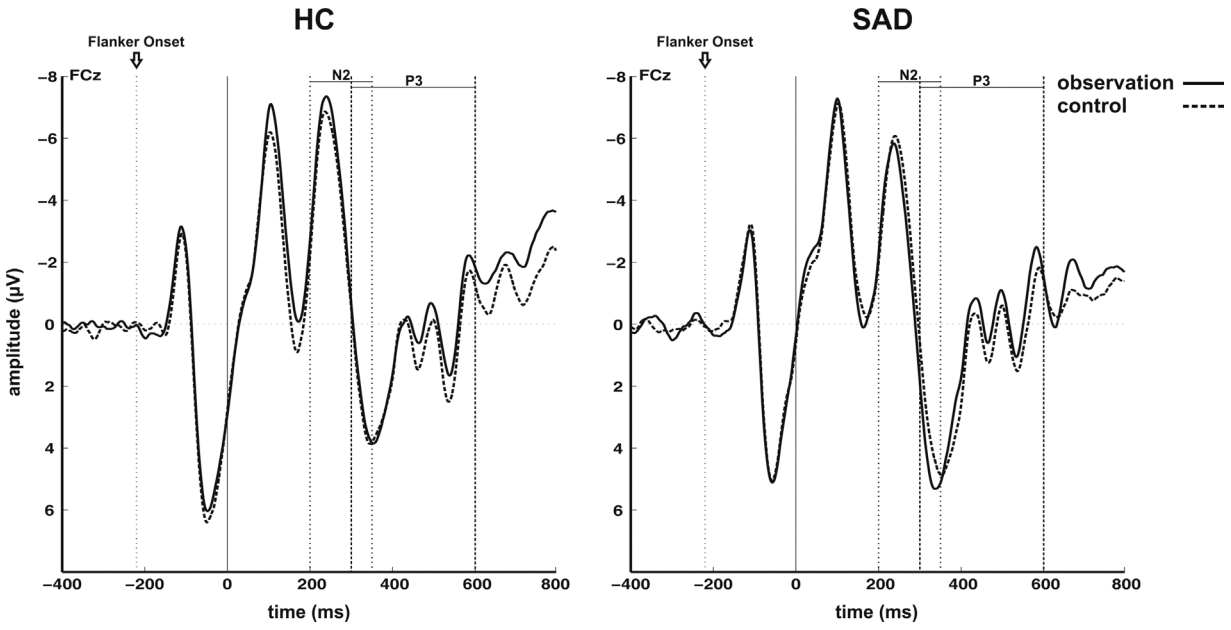

Fig. 4. shows target-locked grand-average waveforms on NoGo trials for SAD patients and HC in the observation and the control condition.

hypotheses, we found no general increase of the ERN (or Pe, NoGo-N2 and NoGo P3) amplitudes in SAD patients and no specific interaction between SAD diagnosis and social context.

The absence of any ERN-related group effect is somewhat surprising given that several studies have found increased ERN in relation to anxiety disorders (see Moser et al., 2013), and social anxiety in particular (Barker et al., 2015; Endrass et al., 2014). However, it should be noted, that at least one other study has failed to find ERN alterations in SAD patients (Schmid, Kleiman, & Amodio, 2015), and in a study by Masaki et al. (2017), individuals high in sports anxiety even tended to exhibit smaller ERN amplitudes than individuals low in sports anxiety. Furthermore, the results obtained by Barker et al. (2015) and Endrass et al.

(2014) differ in important aspects. First, Endrass et al. (2014) examined patients with a confirmed diagnosis of SAD, whereas Barker et al. (2015) reported results obtained from extreme groups (extremely high and low socially anxious individuals) for whom they did not provide information on comorbid symptomatology, medication status etc. It is conceivable that such sample differences can explain the discrepancy between the two studies to some degree. Second, Endrass et al. (2014) did not manipulate social context, but found the ERN to be generally increased in SAD. Barker et al. (2015) actively manipulated social context applying a procedure very similar to the one employed in the present study. They found no ERN alteration in high socially anxious subjects per se, but an interaction effect suggesting that the ERN was increased in HSA only under social observation. The low socially anxious group demonstrated no difference between conditions at all. In the present study, social context modulated ERN amplitude in SAD patients, too, but this effect was not exclusive to the patient group.

In fact, this result is consistent with growing evidence that ERN and related ERP components are increased in normally functioning subjects under social context (e.g. Hajcak et al., 2005; Schindler,Wegrzyn, Steppacher, & Kissler, 2015; Schindler and Kissler, 2016; Peterburs et al., 2017). Research has shown that fear in or of social situations is among the most common anxiety symptoms in the general population (Stein et al., 2000; Furmark, 2002). Social anxiety can therefore be understood as a continuous variable, ranging from absent to normal to seriously debilitating conditions (Brown & Barlow, 2009). In this context, it should be noted that both groups reported higher arousal on error trials in the observation condition, matching the finding of increased ERN amplitude in the observation condition to some degree. Along these lines, it is reasonable to assume that there is no clear threshold for neurophysiological processing of social situations. In general, the ERN has been conceptualized as a neural marker signaling suboptimal performance and the need for cognitive control (Botvinick,Braver, Barch, Carter, & Cohen2001; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). More specifically, it is also often considered a part of the brain's defensive reaction to threat. In their compensatory error-monitoring hypothesis (CEMH), Moser et al. (2013) proposed that the increased ERN found in relation to anxiety symptoms may result from an increase in effort to compensate for distracting effects of anxious apprehension such as worry. Proudfit et al. (2013) assumed that this effect should be attributed to a rather stable disposition in anxious individuals to respond more strongly to uncertain threat. While both hypotheses can explain the present results to some degree, the notion that worrisome social cognitions may have been present in both SAD and HC in the social observation condition seems quite plausible. CEMH might thus provide an adequate explanation to account for the increase in ERN amplitude across groups.

From a methodological perspective, the specific implementation of social context in the present study differs from Barker et al. (2015) in that observation was not exclusively focused on errors. Participants were informed that their performance would be observed during the run, but the instruction suggested that the confederate would also monitor the subjects' general behavior, implying that autonomous responses such as eye blinks, trembling, blushing etc. were also of interest for the observer. This was a deliberate decision, as these autonomous reactions are typical anxiety symptoms that SAD patients tend to focus on in real-life situations and we assumed that including them would increase the ecological validity of the observation run as a typical anxiety-provoking situation for patients. There is evidence, however, that the ERN may be sensitive to task-related, but not to task-unrelated anxiety (Moser et al., 2005). For future studies, it might be interesting to systematically explore the differential impact of explicit performance evaluation compared to effects of a more general observation.

In addition, Barker et al. (2015) appointed a confederate from a peer group to implement social context, while the confederate in the present study was presented as a scientific expert dressed in a white lab coat. It is possible that in the study conducted by Barker et al. (2015), HSA perceived the presence of a peer as a form of social threat, while LSA were unaffected by that particular manipulation. In the present study, however, a sense of hierarchical order was established (Wiemers, Schoofs, & Wolf, 2013), which may have resulted in a more intense sense of scrutiny (and thereby increased ERN) in both SAD and HC. This explanation would also be in line with the results obtained by Hajcak et al. (2005) who reported increased ERN amplitude during evaluation by a research assistant in a sample of healthy subjects.

With regard to the present behavioral findings, SAD and HC showed some notable differences. While accuracy and reaction times were similar between groups, post-error slowing was evident in SAD patients but not in HC. This is in line with our original hypotheses and can be interpreted as increased motivation to avoid errors. Taking into account the subjective ratings, this assumption is supported by the finding that SAD patients rated errors as more arousing and more unpleasant than HC. While there was no interaction effect of condition and group, SAD patients generally rated errors as quite aversive events, with a mean rating of 6.91 (SD = 1.34) on a 9-point Likert scale in the control and mean rating of 7.00 (SD = 1.71) in the observation condition. Given subjects' tendencies to not use the most extreme ratings when responding on Likert scales, the lack of a significant difference between conditions could potentially be attributed to a ceiling effect. Interestingly, correct responses were also associated with an increased sense of unpleasantness and arousal in SAD. This is in line with previous findings suggesting that not only negative, but also positive information can lead to increased feelings of unease in SAD (Weeks, Heimberg, Rodebaugh,& Norton, 2008; Peterburs, Sandrock, Miltner, & Straube, 2008). Finally, patients reported more intense feelings of discomfort during observation, which is also consistent with our hypotheses and emphasizes that the experimental manipulation itself was effective.

Behavioral differences between SAD patients and HC mark an interesting contrast to the electrophysiological findings. Previous research has shown that behavioral and electrophysiological measures of performance monitoring can diverge to some degree (e.g. Fischer, Endrass, Reuter, Kubisch, & Ullsperger, 2015; Peterburs et al., 2012). With regard to the present study (and taking into account CEMH), one could speculate that the basic neural response to observation was similar across groups, but SAD patients and HC may have employed different cognitive strategies to adjust to the given situation and to control their behavior. In future studies, it might therefore be helpful to obtain data regarding such strategies to allow for a better understanding of the processes mediating effects of personality traits (e.g. Pailing and Segalowitz, 2004) and contextual factors on ERP measures such as ERN.

Another possibility relates to the potentially diminishing effect of depressive symptoms on ERN magnitude (Weinberg et al., 2016). In the present study, ERN amplitude in the social observation condition tended to be negatively correlated with depressive symptoms, indicating that higher depression scores were associated with more negative ERN amplitudes, although this effect did not reach statistical significance. When entering BDI scores as a covariate in an attempt to statistically correct for group differences in depressiveness, the condition by response type interaction was no longer significant, possibly suggesting that the trending correlation was mainly driven by the patients' scores. Importantly, BDI scores and SAD diagnosis were somewhat confounded in our study (as is the case in virtually every study in the field), as both were significantly higher in the SAD group. It is therefore difficult to disentangle their particular contributions in a clear-cut fashion. Weinberg et al., (2016) propose that depressive and anxiety symptoms exert opposing effects on the ERN, so that comorbid depression can suppress the enhancing effect of anxiety symptoms. However, given the negative correlation, this pattern does not seem to hold for the present sample. Interestingly, Endrass et al. (2014) also report larger ERN amplitudes in relation to more depressive symptoms in an SAD sample. It has to be noted that while SAD patients in the present study reported significantly more depressive symptoms than

HC, none fulfilled the criteria for a current episode of major depressive disorder. Moreover, the ERN in the control condition was unrelated to depressive symptoms, arguing against a profound impact of depressive symptoms on ERN magnitude in patients. However, it is conceivable that depressiveness enhances sensitivity to the observation. In general, more research is needed to disentangle the relationship between ERN, (social) contextual factors, depressiveness and social anxiety. The overall pattern of results remains inconclusive (e.g., Endrass et al., 2014; Barker et al., 2015; Schmid et al., 2015; Masaki et al., 2017). Our data therefore suggest that one has to be cautious assuming that increased ERN amplitude is a stable and reliable biomarker for anxiety disorders (Riesel, Goldhahn et al., 2017), particularly for patients with less extreme symptoms.

While the expected group differences in early ERP components did not emerge, patients as compared to controls showed increased parietal negativity after errors in a late time window 600 ms–800 ms post-response. Recent studies have shown that errors can trigger adjustments in late ERP components that may explain post-error effects such as post-error slowing (Chang et al., 2014; Perri et al., 2016). Along these lines, it is conceivable that the patients' increased late neural response to errors may link to performance of the following trial. It should be noted, however, that correlational analysis did not support a link between the late negativity and post-error slowing. It also has to be noted that analysis of the late parietal negativity was exploratory and its interpretation is rather speculative. Restrictions in our design, particularly the relatively short inter-trial interval, precluded further investigation of the neural basis of post-error effects.

Several further limitations of the present study need to be considered. In general, null results are a challenge for studies in the field. Given the lack of significant group effects for the ERN in the present study, the possibility of a type 2 error has to be taken into account. Visual inspection of the grand-average waveforms suggests that the main finding of the present work, the interaction effect between response type and condition, may have been driven by the SAD group and that inter-individual variability and a lack of statistical power prohibited a significant group effect. In this context, it has to be noted that the sample size in the present study was relatively small. While certainly comparable to other studies in the field (Endrass et al., 2014; Barker et al., 2015), a larger sample size would have allowed for better stratification of the two groups and hence a more thorough investigation of the relationship between social context and social anxiety level. Another limitation concerns the operationalization of the social context manipulation. In the control condition, subjects were not in a completely private setting as the experimenter was still present in the same room (although eye contact was prevented), potentially blurring differences between the two conditions. We would expect observation effects as found in the present study to be augmented with even more rigorous manipulations of social context.

Finally, some of the patients in the sample were on antidepressive medication. Even though this concerned only a few subjects and although previous work has suggested that SSRIs do not affect ERN magnitude directly (e.g. de Bruijn, Sabbe, Hulstijn, Ruigt, & Verkes,2006; Fischer et al., 2015), we cannot rule out that medication may have influenced the results to some degree.

In conclusion, the present study investigated modulation of electrophysiological measures of performance monitoring in relation to social context and social anxiety. Results showed that social observation during performance of a modified flanker task was associated with increased ERN amplitude. Importantly, this effect was not exclusive to the SAD group. Pe, NoGo-2, and NoGo-P3 were unaffected by observation condition and social anxiety. While overall behavioral performance was largely comparable between groups and across conditions, SAD patients displayed increased post-error-slowing across conditions. In general, the present findings emphasize the importance of social context for the processes underlying performance monitoring. However, the notion of generally error monitoring reflected in an altered ERN in SAD is not supported by our data.

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