Anxiety and error-related brain activity☆

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Abstract

Error-related negativity (ERN/Ne) is a component of the event-related brain potential (ERP) associated with monitoring action and detecting errors. It is a sharp negative deflection that generally occurs from 50 to 150 ms following response execution and has been associated with anterior cingulate cortex (ACC) activity. An enhanced ERN has been observed in patients with obsessive-compulsive disorder (OCD)—reflecting abnormal ACC activity hypothesized as part of the pathophysiology of OCD. We recently reported that the ERN is also enhanced in a group of college students with OC characteristics. The present study extended these findings by measuring the ERN in college undergraduates who scored high on either the Penn State Worry Questionnaire (PSWQ) or a combined version of the Snake (SNAQ) and Spider (SPQ) Questionnaires. Results indicate that, like OC subjects, subjects who score high on a measure of general anxiety and worry have enhanced error-related brain activity relative to both phobic and non-anxious control subjects. The enhanced ERN was found to generalize beyond OCD within the anxiety spectrum disorders but also shows some specificity within these disorders.
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Keywords: ERN; ERP; Anxiety; Error processing; Executive function

1. Introduction

The error-related negativity (ERN) or error negativity (Ne) is a frontocentrally maximal response-locked event-related brain potential (ERP) observed as a sharp negative deflection approximately 50–150 ms after subjects make an error (Falkenstein et al., 1991, 2000; Gehring et al., 1990, 1993). Because it has been observed across various stimulus and response modalities, as well as for different types of errors, the ERN is generally discussed as a neural index of generic response-monitoring processes (Bernstein et al., 1995; Dehaene

et al., 1994; Falkenstein et al., 1991, 2000; Holroyd et al., 1998; Luu et al., 2000a; Miltner et al., 1997; Van't Ent and Apkarian, 1999).

Gehring et al. (2000) found that the ERN was significantly enhanced in a group of patients with obsessive-compulsive disorder (OCD) and ERN magnitude was significantly correlated with symptom severity. Johannes et al. (2001) also found an enhanced ERN in OCD patients relative to controls. In a systematic replication of the Gehring et al. (2000) study, Hajcak and Simons (2002) found that error-related brain activity was significantly greater in high-OC college students than in low-OC students. Unlike Gehring et al. (2000), Hajcak and Simons (2002) found increased error-related brain activity in the high-OC group for both error and correct trials. These results were interpreted in terms of hyperactive action monitoring systems—consistent with OCD symptomology (see Gehring et al., 2000 for a similar explanation).

The enhanced ERN in OCD and OC college students is consistent with a number of neurobiological findings regarding both response monitoring and the pathophysiology of OCD. ERP studies that utilize whole-head recording systems and source localization software have indicated that the ERN is generated by a single source in the medial frontal cortex (Dehaene et al., 1994; Holroyd et al., 1998), most likely the anterior cingulate cortex (ACC) as Gehring et al. (1993) originally suggested. Although a unifying theory of ACC function is currently lacking, Allman et al. (2001) describe the ACC as a "specialized area of the neocortex devoted to the regulation of emotional and cognitive behavior" (p. 109).

The ACC appears to be composed of two distinct regions: a ventral 'affective' region and a dorsal 'cognitive' region (Bush et al., 2000; Allman et al., 2001). These regions appear to have distinct function, cytoarchitechture, and anatomical connectivity (Bush et al., 2000). In terms of their functional roles, the cognitive region of the ACC has typically been implicated in 'counting' Stroop tasks, whereas recent studies have found that the ventral region is active during an 'emotional' Stroop task. These results suggest that affective information activates the ventral region, whereas increased cognitive load activates the dorsal region.

In terms of the ACC and ERN, fMRI studies have shown that the ERN may actually be generated in the affective subdivision of the ACC (Kiehl et al. 2000). The localization of the ERN within the affective portion of the ACC, together with studies indicating that the ERN is larger in groups with emotional disorders, has led some researchers to propose that the ERN is an affective signal related to response monitoring (Luu et al., 2000a,b).

In a study that relates these neurobiological links between the ERN and ACC to OCD, Ursu et al. (2001) used fMRI methodology to measure error-related brain activity in patients with OCD and found that error-related brain activity was significantly enhanced in OCD and also correlated with symptom severity. Like ERP studies that use source localization, Ursu et al. (2001) found that the neural generator of the enhanced error-related brain activity was located in the ACC.

The Ursu et al. results implicating enhanced ACC activity on error trials are consistent with a number of other studies on the pathophysiology of OCD. The ACC is part of a frontostriatal system that also encompasses orbitofrontal cortex (OFC), and areas of the basal ganglia (BG); numerous imaging studies have found that it is precisely this frontostriatal system that appears to be dysfunctional in OCD (Baxter et al., 1987; Breiter et al., 1996; Schwartz et al., 1996; Baer et al., 1995). Rosenberg and Keshavan (1998) found that OCD patients had significantly larger ACC volumes than controls and that ACC volume was

significantly correlated with the severity of obsessions. Swedo et al. (1989) used positron emission tomography (PET) and found that increased metabolism in the ACC was one of the few differences between childhood-onset OCD patients and matched controls. Similarly, Breiter et al. (1996) used fMRI and found that the ACC was one of the brain regions that became active during OCD symptom provocation. In terms of the distinction between the cognitive and affective regions of the ACC, a Rauch et al. (1995) study indicates that it is the ventral 'affective' region of the ACC that is hyperactive in OCD (for consistent results, see Busatto et al., 2000; Rosenberg and Keshavan, 1998).

At the same time, several imaging studies found that the ACC may also be dysfunctional in other anxiety disorders. For example, Bystritsky et al. (2001) found that patients with panic disorder (PD) had significantly increased activation in the ACC during guided imagery that was anxiety provoking. Likewise, Shin et al. (2001) found that veterans with PTSD had abnormal ACC activity on an emotional Stroop task, and interestingly, the brain area that appeared to be dysfunctional was in the rostral 'affective' region of the ACC. In addition, Rauch et al. (1995, 1996) report that the ACC was active in patients with post-traumatic stress disorder (PTSD) and simple phobia (Rauch et al., 1995, 1996) when symptoms of each were provoked. This involvement of the ACC in multiple anxiety disorders has led some researchers to postulate that ACC hyperactivity may be related to the experience of symptoms common to all anxiety disorders (Malizia, 1999; Kimbrell et al., 1999) and not specific to any one disorder such as OCD. This view is supported by data from a recent PET study in which the induction of transient anxiety in healthy adults was associated with increased blood flow in the ACC (Kimbrell et al., 1999).

Additional evidence of a relationship between the ACC and anxiety can be found in studies that demonstrate that stimulating the ACC can produce intense emotional experiences, including anxiety (Baleydier and Mauguiere, 1980). Likewise, the converse of ACC stimulation—the surgical removal of the ACC—is a potential treatment for intractable OCD; studies of patients who have undergone cingulotomies indicate that while these patients continue to experience obsessions, these obsessions are experienced as less distressful after cingulotomy (Baer et al., 1995; Baleydier and Mauguiere, 1980).

Considering the role of the ACC in the pathophysiology of multiple anxiety disorders and the role of the ACC in ERN generation, an enhanced ERN may not be specific to OCD within the anxiety spectrum disorders. Rather, the enhanced ERN found in OCD and OC college students may be more an index of the general pathophysiology of anxiety rather than indicative of a specific action monitoring deficit in OCD.

The present study was conducted to determine if the enhanced ERN found in OCD and high OC students could also be found in other groups of anxious students. We sought to include subjects whose anxiety was either 'trait'- or 'state'-like. For trait-anxious subjects we chose to identify individuals analogous to clinical patients with generalized anxiety disorder (GAD). According to the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV), the main criterion for GAD is excessive or unrealistic worry (American Psychiatric Association, 1994). This group of subjects was identified by high scores on the Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990). For state-anxious subjects we chose students who reported simple phobias based on high scores on a combined version of the Snakes (SNAQ) and Spiders (SPQ) Questionnaire (Klorman et al., 1974). Non-anxious controls were defined as subjects who scored below the mean on both instruments. We

hypothesized that an enhanced ERN would be more related to trait anxiety and therefore would be enhanced only in the high-worry group in the absence of direct symptom provocation.

To test this hypothesis, we measured ERPs while subjects performed a speeded reaction time task. Specifically, we used the Gehring et al. (2000) modified Stroop in which subjects were shown color words such as 'red', presented in either a congruent color (red) or an incongruent color (green). The subjects' task was to respond to the color of the word stimuli with a button press as quickly and accurately as possible.

2. Method

2.1. Subjects

Undergraduate students in an introductory psychology class completed the Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990) and a combined version of the Snake (SNAQ) and Spider (SPQ) Anxiety Questionnaires (Klorman et al., 1974). The PSWQ is a sixteen item self-report measure that assesses dysfunctional attitudes about worry on a five-point Likert scale. The PSWQ has been shown to have excellent psychometric properties in both clinical and non-clinical populations (Brown et al., 1992; Gillis et al., 1995; Stober, 1998; van Rijsoort et al., 1999). The modified version of the SPQ/SNAQ was composed of 25 true or false questions adapted to target fears about either snakes and/or spiders.

Subjects were rank-ordered on the bases of their scores on the PSWQ and modified SPQ/SNAQ. Twenty-four subjects (10 male, 14 female) from the top the PSWQ distribution comprised the worry group (PSWQ = 67.5,[1] modified SPQ/SNAQ = 11.6), 20 subjects (9 male, 11 female) from the top of the modified SPQ/SNAQ distribution comprised the phobic group (PSWQ = 46.4; modified SPQ/SNAQ = 13.8), and 23 subjects (12 male, 11 female) who scored in the bottom of both distributions comprised the control group (PSWQ = 30.2; modified SPQ/SNAQ = 2.2)[2]. All subjects received course credit for their participation and the experimenter was blind to group membership until data reduction was complete.

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[1] Although this study attempted to examine error-related brain activity in a clinical-analog group, no formal clinical assessment was attempted. Accordingly, it is unknown whether subjects in the high-worry group would have met formal diagnostic criteria for generalized anxiety disorder (GAD). Nonetheless, the average PSWQ score for subjects in the high-worry group bears a striking resemblance to scores reported for 50 patients with clinically diagnosed GAD (M = 67.5 in the present study versus 68.1 in Brown et al., 1992).

[2] The overall between-group difference in PSWQ scores was statistically significant ($F(2,64) = 182.78$, $P < 0.001$) as were all the pairwise comparisons of the group means based on post hoc Newman–Keuls tests. There was also an overall mean difference on the specific phobia measure ($F(2,64) = 42.35$, $P < 0.001$), although in this case, post-hoc comparisons revealed that the non-anxious control group scored lower than either the Worry or Phobia group while the latter two groups did not differ significantly. Again, this is consistent with clinical presentation. Specific phobias are not necessarily worriers, but worriers tend to report multiple fears and anxieties. In terms of the entire initial sample ($n = 393$), we found a small ($r = 0.147$) but significant ($P < 0.01$) correlation between the PSWQ and the modified SPQ/SNAQ.

2.2. Task

The Stroop task was administered on a Pentium I class computer, using Presentation software (Neurobehavioral Systems) to control the presentation and timing of all stimuli, the determination of response accuracy, and the measurement of reaction times.

Throughout the task, subjects were shown three color words ('red', 'green', and 'blue') presented either in red or green font on a computer monitor using a black background. Each word occupied approximately 3 degrees of visual angle. A fixation mark (+) was presented below the stimuli, prior to each word. The subjects were instructed to press the right or left mouse button in response to the color of the words.

2.3. Procedure

After a brief description of the experiment, EEG/EOG sensor electrodes were attached and the subject was given detailed task instructions. Each subject was seated 0.5 m directly in front of the computer monitor and given two blocks of 24 practice trials. In one condition, the subjects were told to press the left button on the mouse when the color word was written in red, and the right mouse button when the word was written in green. In the other condition, counter-balanced across subjects, the correspondence between mouse button and word color was reversed. The subjects were told to place equal emphasis on speed and accuracy in their responses. Following two practice blocks, the subjects received 24 blocks of 48 trials (1152 total trials) with each block initiated by the subject. Word stimuli were presented for 200 ms at random intervals between 2000 and 2400 ms.

2.4. Psychophysiological recording, data reduction and analysis

The electroencephalogram (EEG) was recorded using an ECI electrocap. Recordings were taken from three locations along the midline: frontal (Fz), central (Cz), and parietal (Pz). In addition, Med-Associates miniature Ag–AgCl electrodes were placed on the left and right mastoids (A1 and A2, respectively). During the recording, all activity was referenced to Cz. The electro-oculogram (EOG) generated from blinks and vertical eye-movements was also recorded using Med-Associates miniature electrodes placed approximately 1 cm above and below the subject's right eye. The right earlobe served as a ground site. All electrode impedances were below 10 KΩ.

Fz, Pz, A1, A2, and EOG were recorded by a Grass Model 7D polygraph with Grass Model 7P1F preamplifiers (bandpass = 0.05–35 Hz). The EEG was digitized on a laboratory microcomputer at 200 samples per second, using VPM software (Cook, 1998). Data collection began at stimulus presentation and continued for 1500 ms.

Off-line, the EEG for each trial was corrected for vertical EOG and artifacts using the method developed by Gratton et al. (1983) (Miller et al., 1988) and then re-referenced to the average activity of the mastoid electrodes. Trials were rejected and not counted in subsequent analysis if there was excessive physiological artifact (out of range A/D conversion or 'flat' analog signal exceeding 25 ms in duration), or if the reaction time fell outside of a 200–800

Table 1
Reaction time and accuracy means

	Reaction Time (ms)		Accuracy	
	Error	Correct	No. of errors	% Correct
Worry	362	400	79.91	92.83
Phobic	384	424	59.95	94.56
Non-anxious Control	390	425	57.75	94.68

ms window.[3] Finally, the EEG for each trial was time-locked to its respective reaction time and averaged across trials to yield error- and correct-trial ERPs for each electrode site.

To quantify the ERN, each data point after response onset was subtracted from a baseline equal to the average activity in a 100 ms window prior to the response. The ERN was then defined as the most negative peak occurring in a window from 0 to 150 ms post-response. Because there has been some the suggestion that uniformly fast reaction times can give rise to stimulus related activity in the response-locked ERN, the ERN was evaluated for two sets of ERPs. The first set of ERPs involved the comparison of errors to *all* correct trials; the second set of ERPs involved the comparison of errors to a sub-set of *reaction-time matched* correct trials. The ERN and performance measures were statistically evaluated using SPSS (Version 10.0) General Linear Model software.

3. Results

3.1. Performance measures

Accuracy and RT data are presented in Table 1. Subjects tended to have faster RTs for errors than for correct responses ($F(1,64) = 107.99$, $P < 0.001$); however, there was no between-group effect ($F(2,64) = 1.65$, $P > 0.20$) and no interaction between trial and group ($F(2,64) < 1$). Because the number of rejected trials varied between subjects, the number of errors and percentage correct are not redundant statistics, and both are reported. Although worried subjects tended to make more mistakes, this apparent accuracy difference did not reach statistical significance for either the percent correct ($F(2,64)=2.39$, $P > 0.10$) or number of errors ($F(2,64) = 2.71$, $P > 0.05$) performance measure.

Several recent ERN studies on speeded reaction-time tasks have noted a slow-down following commission errors (Falkenstein et al., 2000; Luu et al., 2000a; Nieuwenhuis et al., 2001). To examine this effect and to rule out the possibility that the slow-down effect was nonspecific—i.e. the result of fast responses and simply a matter of regression toward the mean—we calculated the average reaction time for trials that followed an error and compared this RT to the average RT for trials that followed correct trials matched to the error trials on the basis of reaction time. These data are presented in Table 2.

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[3] Trials rejected for each all of these reasons accounted for 4.9% of the 1152 total trials and did not differ as a function of subject group ($F(2,64) = 2.30$, $P > 0.10$).

Table 2
Accuracy and reaction-time data for error trials, RT-matched correct trials and trials subsequent to each

	Worry	Phobic	Non-anxious Control
RT for error trials	362	384	390
RT for RT-matched correct	362	384	389
RT following error	420	439	447
RT following RT-matched correct	387	413	422
Accuracy following error	93%	94%	93%
Accuracy following RT-matched correct	92%	94%	94%

The analysis of these data confirmed that trials subsequent to error trials were associated with slower RTs ($F(1,64) = 123.11$, $P < 0.001$), and that this slow-down effect was significantly greater after errors than after equally rapid correct responses ($F(1,64) = 32.48$, $P < 0.001$). Thus, although some regression was noted, there remains significant slowing that is specific to error trials. As was the case for the other performance measures, however, this post-error slowing was independent of group status ($F(2,64) < 1$).

3.2. ERN—all trials

The response-locked average waveforms are presented in Fig. 1.

The figure illustrates that when subjects made errors, there was a sharp negative deflection that peaked around 55 ms post-response, primarily at the frontal recording site. A 3 (Group) $\times$ 2 (Trial Type) $\times$ 3 (Electrode Site) analysis of variance (ANOVA) with Greenhouse–Geisser corrected p-values supported the impression the ERN was predominantly frontal ($F(2,128) = 81.38$, $P < 0.001$, $e = 0.76$), that negative activity was significantly greater when subjects made errors ($F(1,64) = 69.10$, $P < 0.001$) and that trial and site interacted such that the difference in the ERN magnitude between correct and incorrect trials was largest at the Fz recording site ($F(2,128) = 62.17$, $P < 0.001$, $e = 0.75$). Thus, our results are consistent with previously reported ERN morphology and topography.

The hypothesis that ERN peak magnitude would systematically vary between groups was confirmed. ERN means are presented in Fig. 2. The ANOVA yielded a significant group effect ($F(2,64) = 4.66$, $P < 0.05$); the ERN was larger among worried subjects than it was in the phobic and non-anxious control groups. Interestingly, there was no three-way group by trial by location interaction and no interactions between group and trial type or between group and location, suggesting that ERN activity was generally enhanced in the worry group following both correct and error responses.

3.3. ERN—RT matched trials

Although, on average, each ERP is response-locked between four and five hundred milliseconds after the presentation of the stimulus, it is possible that some of the ERP activity could nonetheless be stimulus driven (Gehring et al., 1993; Scheffers et al., 1996). If RTs on error trials are faster and more uniform than correct trials, these trials might contribute stimulus-related artifact to the ERN. To avoid this potential confound, we further examined
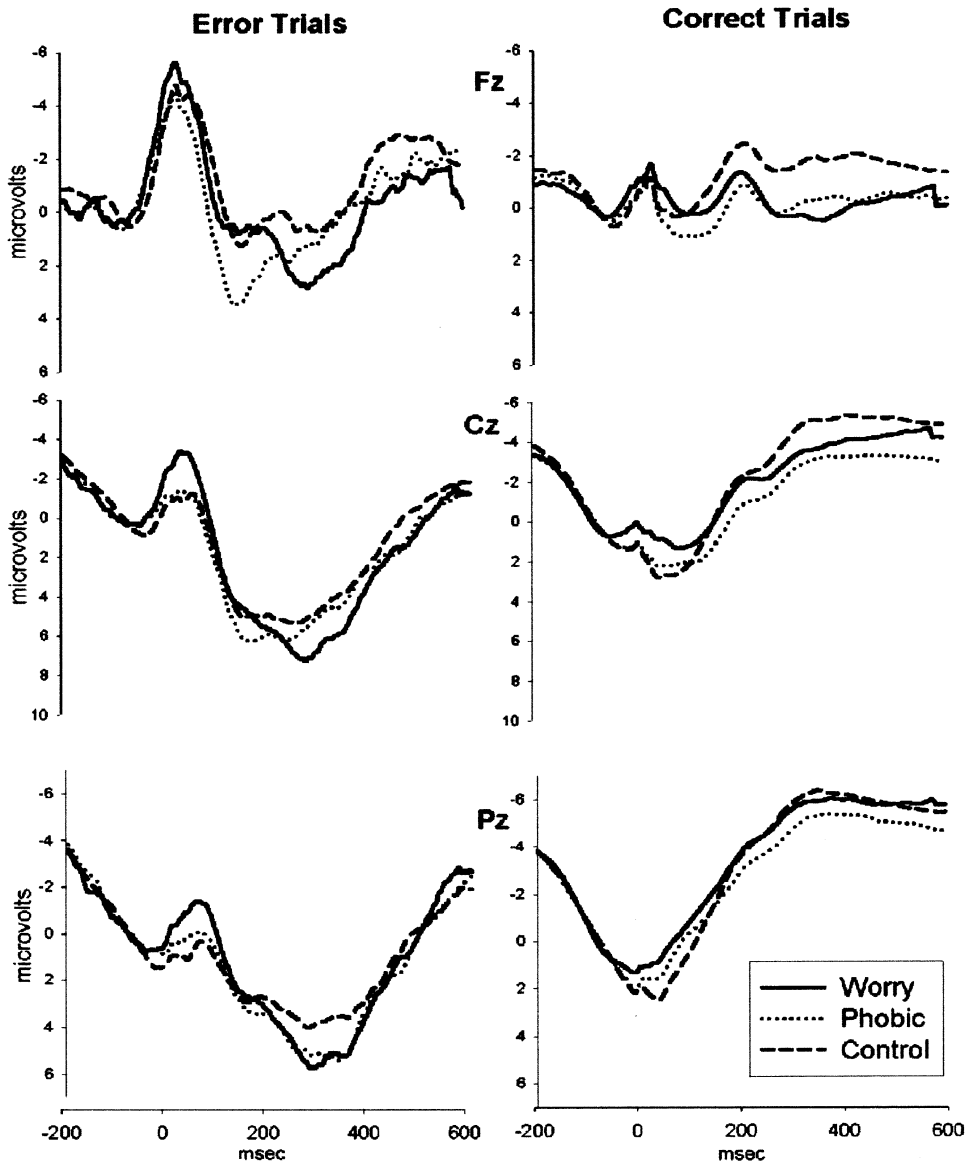
Fig. 1. ERP waveforms for each group for error trials (left) and correct trials (right) at the Fz, Cz, and Pz recording sites.

a subset of the correct trials for each subject. To do this, each error trial was matched to a correct trial on the basis of reaction time. This way, any stimulus-locked activity resulting from fast and uniform reaction times would equally effect correct and error trials and eliminate the potential confound.
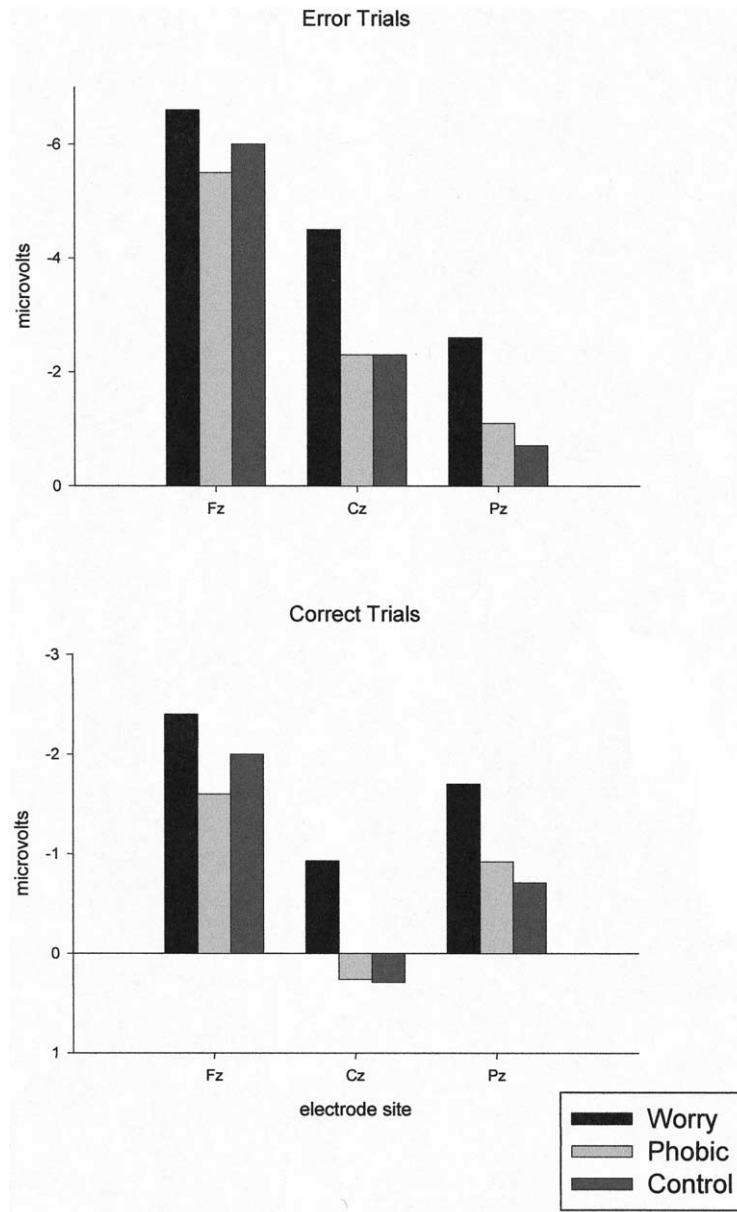
Fig. 2. ERN magnitude for error and correct trials as a function of subject group and electrode site.

After the subset of matched trials was selected on the basis of reaction time, ERP averages for matched-correct and error trials were once again created and the peak negativity in the 0–150 ms window was identified and scored. As before, the ANOVA revealed a highly significant effect for electrode site ($F(2,128) = 88.72$, $P < 0.001$, $e = 0.72$) and trial type

($F(1,64) = 42.96$, $P < 0.001$), as well as a significant interaction between electrode site and trial type ($F(2,128) = 28.44$, $P < 0.001$, $e = 0.79$). Most importantly, the effect of group membership was still significant ($F(2,64) = 5.80$, $P < 0.01$), and there were no significant interactions involving group membership. That is, the ERN was enhanced in the worried subjects, and this enhancement was present on both correct and incorrect trials.

Lastly, to determine whether the significant between group difference is, in fact, attributable to the increase in the ERN specifically, the amplitude was rescaled at the three different electrodes as suggested by McCarthy and Wood (1985). That is, the amplitude at each electrode site was divided by the standard deviation of the six (Trial × Site) ERN values and then submitted to analysis of variance. Supporting the specificity of the Group effect to the ERN, the electrode site ($F(2,128) = 89.47$, $P < 0.001$, $e = 0.72$), trial type ($F(1,64) = 60.16$, $P < 0.001$,) and trial × site ($F(2,128) = 36.66$, $P < 0.001$, $e = 0.78$) effects remained highly significant while the main effect of group membership was eliminated. Again, no interactions with the group variable were evident.

4. Discussion

Consistent with our original hypothesis, we found that subjects who scored high on the PSWQ differed from subjects who scored high on a measure of specific phobia and from non-anxious control subjects on electrophysiological measures related to response monitoring. In particular, we found that the worried group had significantly greater error-related brain activity than did either the phobic or non-anxious control groups. These results indicate that within the anxiety spectrum disorders an enhanced ERN may not be specific to OCD.

Like our previous study on OC college students, we did not find an interaction between group (worry, phobic, non-anxious control) and trial type (correct vs. error), suggesting that the enhanced error-related brain activity in the worry group is not specific to errors. Rather, the data indicate that enhanced error-related brain activity is associated with both error and correct trials. Although the attribution of *error*-related activity to the processing of *correct* trials appears incongruous, multiple researchers have noted a small ERN-like potential on correct trials. Such activity has been referred to as the correct response negativity (CRN; Coles et al., 2001; Falkenstein et al., 2000; Vidal et al., 2000) and it has been the subject of various hypotheses. Coles et al. (2001), for example, have proposed that the CRN could be the result of stimulus artifact, or partial error-processing on correct trials with sub-threshold incorrect activity or violations of implicit temporal parameters that define a correct response (for a different point of view, however, see Vidal et al., 2003). While the elucidation of information-processing mechanisms that underlie both the ERN and CRN is still ongoing, it is nonetheless true that both the ERN and CRN were enhanced in worry, relative to phobic and non-anxious control subjects—and these data provide some support for the similarity of the ERN and CRN.

In our previous study on OC college students, we interpreted the enhanced ERN/CRN as consistent with OCD characteristics. That is, patients with OCD continually doubt and check their actions, regardless of whether or not a 'mistake' has actually been made—and we felt these characteristics may be related to an enhanced ERN/CRN. Although the worry group in the present study was not specifically assessed for OC characteristics, previous data

collection in our laboratory ($n = 396$) revealed a correlation between the PSWQ and the OCI of $r = 0.40$. Though a significant relationship, it is also true that the PSWQ and the OCI are not redundant measures. In another community sample (i.e. nonpatient), van Rijsoort et al. (2001) found that worry and obsessive-compulsive characteristics were clearly differentiated, and this distinction between OC and worried symptomology is consistent with clinical research that demonstrates that OCD and GAD have rather low rates of co-morbidity (Brown et al., 1993; Abramowitz and Foa, 1998). Thus, there is reason to believe that the enhanced ERN/CRN recorded from worried subjects is not due to OC-specific characteristics. Rather, OC and worried subjects likely share one or more underlying features that relate to enhanced error-related brain activity.

One possible explanation for the enhanced ERN in OC and worried subjects has been suggested by Luu et al. (2000a), who report a relationship between ERN and negative affect (NA). Negative affect has been described as 'emotional distress' and figures prominently in Clark and Watson's (1991) tripartite model of depression and anxiety. The tripartite model suggests that both anxiety and depression are characterized by high levels of NA, whereas depression is uniquely related to low positive affect (PA). In terms of this model, Brown et al. (1998) confirmed that both GAD and OCD are characterized by high levels of negative affect (NA) and moderate to high levels of positive affect (PA). Thus, the current results could be interpreted in terms of high NA associated with anxiety. Of course, high NA is not specific to anxiety—and future studies may wish to evaluate the potential effects of high and low PA on the ERN. If an enhanced ERN is specific to anxiety as we currently believe, then an enhanced ERN should only be observed for high NA in the context of moderate to high PA.

Although the electrode montage employed in the present study was limited to three recording sites, the distinct frontal maximum of the ERN is consistent with other more focused studies using both ERP and fMRI techniques that localize the neural source of the error-related activity to medial frontal cortex, specifically the ACC (Dehaene et al., 1994; Gehring et al., 1993, 2000; Luu et al., 2000a). As discussed earlier, the affective subdivision of the ACC appears to be hyperactive in OCD, PTSD, and Panic—and may be related to the pathophysiology of anxiety disorders, more generally. The present data suggest that this hyperactivity can be observed using an ERN paradigm in young-adult college students who report excessive and uncontrollable worry.

Although we found ERN/CRN differences between the worry, phobic, and non-anxious control groups in the present study and between OC and control subjects in our previous study, we did not find corresponding differences in task performance between groups in either study. Although the worry group did make relatively more mistakes than the other two groups, this difference did not reach statistical significance in post-hoc between-group tests. Like previous studies, we found that all groups had similar reaction times and accuracy during the procedure (Gehring et al., 2000; Luu et al., 2000a; Hajcak and Simons, 2002). As in previous ERN studies, we found that trials following mistakes were characterized by slower reaction times, but again, this post-error compensatory slowing was unrelated to group status and ERN magnitude (Hajcak and Simons, 2002; Gehring and Fencsik, 2001; Gehring and Knight, 2000; Scheffers et al., 1999). Thus, despite an enhanced ERN/CRN in the worried students, there were no group differences in accuracy or RT, and no evidence that larger error-related brain activity was related to the RT slowing that occurs on trials following an error.

This dissociation of the ERN and behavioral measures is consistent with anxious pathology. Pathological anxiety reflects *unnecessary* emotional reactions that are typically not associated with measurable performance differences. For instance, subjects with high perfectionistic concerns typically make errors that are similar in magnitude and quantity to subjects with low perfectionistic concerns; however, highly perfectionistic subjects appear to differ significantly in terms of their reaction to the errors they commit (Frost et al., 1997). In short, the dissociation between performance and self-reported reaction is quite like the enhanced ERN/CRN observed in the present study.

Thus, the present study fits well into a growing body of research indicating that anxiety and anxiety disorders are associated with neural hyperactivity of the ACC. The present study adds to this literature by suggesting that within the anxiety spectrum disorders enhanced error-related brain activity may not be specific to OCD. Rather, the enhanced ERN/CRN found both in OC and Worry subjects suggests that more general personality dimensions such as negative and positive affect and their configuration may best explain neural differences related to response monitoring.

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