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# The effects of fear on performance monitoring and attentional allocation

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**Abstract**

Evidence from event-related potential (ERP) studies indicates abnormal error processing and attentional allocation in "trait"-anxious individuals. However, few studies have been conducted that evaluate relevant ERP components during the induction of an anxious state (i.e., fear). In the present study, ERPs were measured in 16 undergraduates during control and fear induction conditions to examine the effects of fear on error processing and attentional allocation. Despite comparable performance in both experimental conditions, the ERP data indicated reductions in attentional allocation and error salience during fear induction. Fear did not appear to directly alter early error processing, as indicated by the error-related negativity, however. The implication of these results for understanding how trait and state anxiety may affect error processing and attentional allocation are discussed.

**Descriptors:** Anxiety, Error-related negativity, Ne, Attention, P300, Error positivity

Research involving event-related brain potentials (ERPs) has identified a negative deflection in the response-locked ERP when subjects make mistakes in speeded reaction time tasks. This error-related negativity (ERN or Ne) is observed at fronto-central recording sites, begins around the time of an erroneous response, and peaks approximately 50 ms later (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Falkenstein, Hoorman, Christ, & Hohnsbein, 2000; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The ERN is thought to reflect generic response monitoring processes because it has been observed across different response modalities and different error types (Bernstein, Scheffers, & Coles, 1995; Dehaene, Posner, & Tucker, 1994; Falkenstein et al., 1991, 2000; Holroyd, Dien, & Coles, 1998; Luu, Flaisch, & Tucker, 2000; Miltner, Braun, & Coles, 1997; Van't Ent & Apkarian, 1999).

Studies that employ source-localization procedures suggest that the ERN is generated by a single source in the medial frontal cortex, most likely the anterior cingulate cortex (ACC; Dehaene et al., 1994; Gehring et al., 1993; Holroyd et al., 1998; Miltner et al., 1997). Other studies that use magnetoencephalography (MEG) and functional magnetic resonance imaging (fMRI) further support the notion that the ACC is active during error processing (Garavan, Ross, Murphy, Roche, & Stein, 2002; Kiehl, Liddle, & Hopfinger, 2000; Miltner et al., 2003).

A number of studies have reported an enhancement of the ERN in affectively distressed subjects. Gehring, Himle, and Nissenson (2000) first reported an increased ERN in patients with obsessive-compulsive disorder (OCD). Similar results were later reported by Johannes et al. (2001) and Hajcak and Simons (2002). Consistent with the notion that this enhanced error-related brain activity is generated in the ACC, studies using fMRI have also found error-related ACC hyperactivity in patients with OCD (Ursu, Stenger, Shear, Jones, & Carter, 2003). This hyperactive action monitoring does not seem to be specific to OCD, as an enhanced ERN has also been observed in other groups that experience high levels of affective distress. For instance, increased error-related brain activity has been found in worried college students (Hajcak, McDonald, & Simons, 2003) and in college students who report high levels of negative affect on the Positive and Negative Affect Schedule (NA; Hajcak, McDonald, & Simons, 2004; Luu, Collins, & Tucker, 2000). Results such as these suggest that enhanced error-related brain activity is not specific to OCD and may be due to ACC hyperactivity that characterizes a variety of "trait" anxious subjects.

Consistent with this proposed relationship between affective distress and the ACC, evidence of increased ACC activity during *states* of affective distress has also been reported. For instance, studies conducted with healthy individuals have demonstrated increases in ACC activity during anxiety induction (Benkelfat et al., 1995; Chua, Krams, Toni, Passingham, & Dolan, 1999; Kimbrell et al., 1999; Servan-Schreiber, Perlstein, Cohen, & Mintun, 1998), anticipatory anxiety (Javanmard et al., 1999), and during fear-potentiated startle (Pissiota et al., 2003). Similarly, increases in ACC activity during symptom provocation have been reported in patients with panic disorder (Boshuisen, Ter Horst, Panns, Reinders, & den Boer, 2002; Bystritsky et al., 2001), OCD (Breiter et al., 1996; Rauch et al., 1994), posttraumatic stress disorder (PTSD; Rauch et al., 1996), and simple phobias (Rauch et al., 1995).

Taken together, these results suggest that ACC activity is enhanced in subjects with chronic anxiety and also during transient increases in fear and anxiety, and provide initial support for the

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notion that negative affect might similarly increase ACC activation during response monitoring. This possibility is consistent with a recent proposal regarding the ERN by Luu and Tucker (2004), who argue that the ERN may be an affective signal that indexes the distress associated with detecting the deviation between an action and an intended goal (i.e., error commission; see also Luu et al., 2000). Based on this interpretation of the ERN (and important to the present study), they proposed that individuals characterized by higher levels of affective distress generate larger ERNs because their typical negative affective state biases the monitoring system in favor of larger deviation signals; specifically, that the "emotional state sets the magnitude of the response to the discrepancy [between actual and intended action]" (Luu & Tucker, 2004, p. 18). Despite the possibility that a negative emotional state might result in a larger ERN, this causal relationship has not yet been established experimentally, and this is the primary hypothesis tested in the current study.

A second ERP component related to error processing may also be sensitive to affective distress. This is the Pe, or error positivity; it follows the ERN, peaks at more posterior regions approximately 200–400 ms after response execution, is also thought to have a source in the ACC (Hermann, Römmler, Ehlis, Heidrich, & Fallgatter, 2004; van Veen & Carter, 2002) and has been implicated in processes that follow error detection, possibly related to error awareness or error salience (Falkenstein, Hohnsbein, & Hoormann, 1995; Falkenstein et al., 2000; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001). For instance, Nieuwenhuis et al. found that although the ERN was unchanged by lack of error awareness in an antisaccade task, the Pe was significantly reduced. In a study of a modified word Stroop task in high and low NA college students, Hajcak, McDonald, and Simons (2004) noted a significantly decreased Pe in high-NA participants compared to low-NA participants, possibly indicating less awareness of errors in affectively distressed individuals. Although other studies of "trait" anxious individuals did not formally evaluate the Pe, visual inspection of the raw waveforms reveals a mixed picture; figures from Gehring et al. (2000), Hajcak and Simons (2002), and Hajcak et al. (2003) suggest Pe reductions in obsessive-compulsive subjects but not in subjects with chronic worries.

In addition to indexing error processing, ERPs can be used to shed light on more basic cognitive processes associated with stimulus evaluation itself. For instance, the P300 is a stimulus-locked ERP component that peaks at posterior sites approximately 300 ms following the onset of a stimulus; the P300 is a well-studied index of attentional allocation and orienting (Donchin, 1981; Donchin & Coles, 1988). Importantly, the P300 also appears to be modulated by affective distress. Fear-relevant stimuli, for instance, seem to elicit larger P300s in anxious patients (Attias, Bleich, Furman, & Zinger, 1996; Pauli et al., 1997). On the other hand, task-relevant (but fear-irrelevant) stimuli presented during simple vigilance tasks (e.g., target tones in an auditory discrimination task) are associated with reduced P300s in OCD patients (Beech, Ciesielski, & Gordon, 1983; Ciesielski, Beech, & Gordon, 1981; Towey et al., 1994) and P300 reductions have also been related to higher levels of state anxiety in PTSD patients (Metzger, Orr, Lasko, & Pitman, 1997). Such results suggest that negative affective states during vigilance may drain attentional resources from the primary task in both trait and state anxious subjects. Consistent with this possibility, studies by Lang and his colleagues have reported reduced P300 amplitudes to probe stimuli presented while brief high-arousal emotion states are induced by picture viewing (Cuthbert, Schupp, Bradley, McManis, & Lang, 1998; Schupp, Cuthbert, Bradley, Birbaumer, & Lang, 1997).

Despite evidence that fear and anxiety are related to abnormal ERPs that index error processing and attentional allocation to task-relevant stimuli in simple vigilance tasks, there have been no studies evaluating each of these ERPs during the induction of a specific fear. If affective distress plays a causal role in altering error processing and attentional resource deployment, then fear induction should have a corresponding effect on error-related brain potentials (ERN and Pe) and the P300, respectively. In particular, we predicted that inducing a state of fear would increase the ERN and reduce the amplitude of the Pe and the P300. To test these hypotheses, we measured ERPs in spider-fearful subjects while they performed a choice reaction time task both in the presence of a spider and during a control condition. The decision to use spider-fearful participants in this study was based on reports that suggest inducing a fearful state in these subjects is relatively easy and results in changes in ACC activity (see Rauch et al., 1995). Stimulus- and response-locked ERPs were evaluated to assess the impact of fear, or increases in "state" levels of anxiety, on attentional and error-related processes, respectively.

## Method

### *Participants*

Participants were recruited through the University of Delaware Psychology Department subject pool. Nine hundred and seventy-three undergraduate students completed the Spider Questionnaire (SPQ; Klorman, Hastings, Weerts, Melamed, & Lang, 1974) and the Positive and Negative Affect Schedule (PANAS; Watson, Clark, and Tellegen, 1988) at a preliminary testing session as partial fulfillment of course requirements. The SPQ is a self-report measure of spider phobia comprised of 31 questions that are rated as either true or false. The PANAS is a 20-item self-report measure that measures two mood dimensions: positive affect (PA; 10 items) and negative affect (NA; 10 items). PANAS items are rated on a Likert-type scale ranging from 1 (*very slightly or not at all*) to 5 (*extremely*). Following the preliminary testing session, participants were rank-ordered on the basis of their SPQ score. Twenty students (6 male, 14 female) from the top of the SPQ distribution were recruited to participate in the current experiment ($M = 24.9$; $SD = 3.39$). Data for 1 participant were discarded due to a technical malfunction. In addition, 1 participant was too fearful to participate in the fear-induction condition of the experiment and was not included in the final study sample. The study sample comprised 18 spider fearful (5 male, 13 female) undergraduates.

### *Task*

A modified version of the Eriksen Flanker task (Eriksen & Eriksen, 1974) was administered on a Pentium I class computer, using Presentation software (Neurobehavioral Systems, Inc.) to control the presentation and timing of all stimuli, the determination of response accuracy, and the measurement of reaction times.

During the task, subjects were shown sets of five arrowheads (<<<<<, <<><<, >>>>>, or >><>>). In this way, there were two congruent conditions (<<<<< and >>>>>) and two incongruent conditions (<<><< and >><>>). The stimuli were presented randomly such that 50% of trials were congruent. Sets of arrowheads were presented

in the center of the computer screen for 200 ms in white font against a black background at random intervals between 1700 and 2300 ms. At a viewing distance of roughly 65 cm, each set of arrowheads occupied 1.3° of visual angle vertically and 9.2° horizontally. A fixation mark (+) was presented just prior to the onset of each stimulus. Subjects were instructed to press the left or right mouse button in accordance with the direction of the center arrowhead.

***Task Procedures***

After participants received a general description of the experiment, EEG/EOG electrodes were attached and participants were given detailed task instructions. Each participant was seated approximately 0.5 m directly in front of the computer monitor and given two blocks of 48 practice trials. In the first practice block, participants were simply instructed to respond to the direction of the center arrowhead by clicking the left or the right mouse button. The instructions for the second practice block were modified such that participants were asked to focus on being fast and accurate while responding to the direction of the center arrowhead. Following the practice blocks, participants received 12 blocks of 48 trials (576 trials) in each of two experimental conditions (1152 total trials).

***Fear-Induction Procedures***

Participants performed the flanker task both with and without the presence of a Chilean rose-haired tarantula. Each condition lasted approximately 20 min and a 5-min break was taken between conditions—the order of conditions was counterbalanced between subjects such that half the subjects performed the flanker task in the control condition first.

During both experimental conditions, participants performed the flanker task in a dimly lit room and were seated approximately 0.5 m from a male lab assistant. To get a general measure of the subjects' emotional state at the beginning, middle, and end of each experimental condition, we utilized the Subjective Units of Discomfort Scale (SUDS; Wolpe, 1958) and asked subjects to rate from 0 to 100 how afraid, anxious or distressed they felt with 0 indicating no fear/anxiety/distress and 100 indicating the most fear/anxiety/distress they had ever experienced. The SUDS is a widely used measure of state anxiety that has been shown to be sensitive to exposure to feared stimuli (e.g., Johnstone & Page, 2004), treatment effects (e.g., Antony, McCabe, Leeuw, Sano, & Swinson, 2001) and degree of emotional engagement in exposures (e.g, Jaycox, Foa, & Morral, 1998). In the provocation condition (i.e., during fear induction), participants were asked to perform the flanker task with the spider removed from a glass aquarium and in the hands of the lab assistant. Two participants could not tolerate the spider outside of its glass aquarium and performed the flanker task with the spider in the aquarium on the floor next to the lab assistant. In the provocation condition, the lab assistant encouraged the spider to move from hand to hand, whereas in the control condition, the lab assistant passed a small ball between his hands mimicking the hand movements made in the provocation condition.

***Psychophysiological Recording, Data Reduction, and Analyses***

The electroencephalogram (EEG) was recorded from the frontal (Fz), central (Cz), and parietal (Pz) recording sites using a Neurosoft Quick-Cap. In addition, tin disc electrodes were placed on the left and right mastoids (A1 and A2, respectively). During the recording, all activity was referenced to Cz. The electrooculogram (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 EEG/EOG electrode impedances were below 10 KΩ and the data from all channels were recorded by a Grass Model 7D polygraph with Grass Model 7P1F preamplifiers (bandpass = 0.05–35 Hz).

All bioelectric signals were digitized on a laboratory microcomputer using VPM software (Cook, 1999). The EEG was sampled at 200 Hz. Data collection began with the onset of the imperative stimulus and continued for 1500 ms. Off-line, the EEG for each trial was corrected for vertical EOG artifacts using the method developed by Gratton, Coles, and Donchin (1983; Miller, Gratton, & Yee, 1988) and then re-referenced to the average activity of the mastoid electrodes. Trials were rejected and not counted in the subsequent analysis if there was excessive physiological artifact or if the reaction time fell outside of a 200–800-ms window. Finally, the EEG for each trial was time-locked to either reaction time or stimulus onset and averaged across trials to yield error- and correct-trial ERPs for each electrode site.

To quantify the response-locked ERPs, each data point after response onset was subtracted from a baseline equal to the average activity in the 100-ms window prior to the response. The ERN was then defined as the average activity in a window from 0 to 100 ms postresponse. The Pe was defined as the average activity in the 200–400-ms postresponse window (Nieuwenhuis et al., 2001). Because data collection began with stimulus onset, the stimulus-locked ERP was identified by deviating each data point after stimulus onset from the stimulus-onset point, and then quantifying the P300 as the most positive peak in a window from 300 to 500 ms poststimulus.

Performance measures consisted of accuracy and response times during the flankers task and post-error slowing. Because the number of trials rejected due to EEG/EOG artifact varied between experimental conditions, the number of errors and percentage correct are not redundant behavioral measures of accuracy, and both are reported. Numerous studies have shown that reaction time is increased on trials that follow errors and this is often thought to reflect a compensatory behavior to prevent further errors (Rabbitt, 1981). Some investigators have suggested that the ERN and Pe may relate to this compensatory post-error behavior (Gehring et al., 1993; Nieuwenhuis et al., 2001). In the current study, an analysis of post-error slowing was conducted comparing RTs on correct trials that followed correct trials and on correct trials that followed error trials for the two experimental conditions.

Repeated-measures analyses of variance (ANOVAs) were performed on behavioral and ERP measures with Greenhouse–Geisser corrected $p$ values applied where appropriate. The ERN and Pe were evaluated on error trials and the P300 was evaluated on correct trials.

## Results

Two of the original 18 participants were not included in the behavioral and ERP analyses because they failed to become anxious in the provocation condition (SUDS score was equal to that in the control condition). Therefore, all analyses described below are based on data from the 16 participants who responded to the manipulation. The mean PANAS score for these subjects was 21.2 ($SD = 8.5$) for NA and 32.8 ($SD = 7.2$) for PA and these scores did not differ significantly from the population NA

**Table 1.** *Mean (Standard Deviation) SUDS Ratings*

| | Control condition | Provocation condition |
|---|---|---|
| SUDS rating 1 | 20.31 (21.56) | 79.81 (9.55) |
| SUDS rating 2 | 18.44 (19.38) | 77.81 (12.24) |
| SUDS rating 3 | 16.50 (15.94) | 74.50 (16.11) |
| Overall | 18.42 (17.80) | 77.38 (11.22) |

($\mu = 18.9, \sigma = 6.65$; $z = 1.36, p > .15$) and PA ($\mu = 32.9, \sigma = 7.14$; $z = 0.05, p > .90$) scores.

### *Behavioral Measures*

Table 1 presents SUDS data for both experimental conditions. Participants reported high SUDS scores (i.e., more fear/anxiety/distress) in the provocation condition than they did in the control condition. We conducted a 2 (Experimental Condition) × 3 (Time Point) ANOVA on SUDS scores that yielded a main effect for Experimental Condition, $F(1,15) = 221.29, p < .001$, no main effect for Time Point, $F(2,30) = 1.83, p > .15$, and no interaction of Experimental Condition and Time Point, $F(2,30) < 1$. In addition, behavioral observations supported these SUDS results. Participants exhibited a number of physiologic and behavioral signs of acute distress during task breaks and instruction periods in the provocation condition that included trembling and tearing.[1] None of these signs were present during the control condition.

Accuracy and RT data for the control and provocation conditions are presented in Table 2. Although participants reported higher SUDS during the provocation condition, their performance on each performance index did not suffer as a result. Overall, participants did not make more errors in the provocation condition than in the control condition, $F(1,15) < 1$. Likewise, participants did not differ in terms of overall proportion correct, $F(1,15) < 1$. In terms of RT, a 2 (Experimental Condition) × 2 (Trial Type) ANOVA indicated that participants were faster on error trials, $F(1,15) = 195.42, p < .001$, but not faster in the provocation condition, $F(1,15) < 1$. The interaction between Experimental Condition and Trial Type did not reach significance, $F(1,15) < 1$.

A 2 (Experimental Condition) × 2 (Trial Type) ANOVA comparing RTs on correct trials that followed correct trials and on correct trials that followed error trials for the two experimental conditions indicated that subjects were slower following errors, $F(1,15) = 7.03, p < .05$; however, no overall effect of Experimental Condition, $F(1,15) < 1$, and no significant interaction between Experimental Condition and Trial Type, $F(1,15) < 1$, were found. Thus, although participants evinced post-error slowing, this effect was not larger following errors in the provocation condition. In addition, there was no difference between participants' accuracy following errors in the control condition and their accuracy following errors in the provocation condition, $F(1,15) < 1$. Finally, the number of artifact-based rejected trials did not differ between the control ($M = 9.00$, $SD = 19.18$) and provocation ($M = 4.56$, $SD = 9.50$) conditions, $F(1,15) < 1$, nor did the number of rejected trials based on reaction times falling outside the acceptable 200–800-ms window ($M = 5.00$, $SD = 8.83$ and $M = 11.69$, $SD = 24.56$ for the control and provocation conditions, repectively), $F(1,15) = 1.09, p > .30$.

[1]These physiologic signs of anxiety were assessed via behavioral observations at testing and are presented to provide a sense of the strong impact the experimental manipulation had on participants.

### *ERPs*

The response-locked average ERP waveforms at Fz, Cz, and Pz for all errors in the control and provocation conditions are presented in Figure 1. The figure illustrates that the response-locked waveforms on error trials are characterized by a sharp negative deflection that reached its maximum approximately 50 ms postresponse at the frontal location (Fz). A 2 (Experimental Condition) × 3 (Electrode Site) ANOVA confirmed that the ERN was frontally maximal, $F(2,30) = 22.98, p < .001$. Thus, our results are consistent with other reports of ERN morphology and topography. Contrary to our prediction that the ERN would be larger in the provocation condition, the ANOVA revealed no main effect for Experimental Condition, $F(1,15) = 1.15, p > .25$, and no interaction between Experimental Condition and Electrode Site, $F(2,30) < 1$. Confirming the impressions illustrated by Figure 1, a 2 (Experimental Condition) × 3 (Electrode Site) ANOVA indicated that the Pe had a centro-parietal scalp distribution, $F(2,30) = 30.65, p < .001$. Consistent with our prediction, the Pe was smaller in the provocation condition, $F(1,15) = 7.29, p < .05$. Finally, there was no significant interaction between Experimental Condition and Electrode Site, $F(2,30) < 1$.

The stimulus-locked average ERP waveforms at Fz, Cz, and Pz for all correct trials in the control and provocation conditions are presented in Figure 2. Consistent with previous research, a 2 (Experimental Condition) × 3 (Electrode Site) ANOVA indicated that the P300 had a centro-parietal scalp distribution, $F(2,30) = 16.45, p < .001$. In addition, our hypothesis that the P300 would be smaller in the provocation condition was confirmed, $F(1,15) = 5.51, p < .05$.[2] Finally, there were no significant interactions between Experimental Condition and Electrode Site, $F(2,30) = 1.58, p > .20$.

## Discussion

During the fear-induction procedure, subjects reported significantly increased levels of affective distress. Consistent with our hypotheses, we found that the magnitude of both the P300 and Pe were attenuated during the fear-induction condition. However, contrary to our original hypothesis, fear induction did not enhance the ERN, and we did not find any impact of the fear induction on measures of performance. Taken together, these results suggest that the fear induction reduced attentional allocation to the imperative stimuli and reduced the salience of flanker-task mistakes but did not appear to alter early error-processing.

Studies demonstrate that the P300 is elicited by stimuli given attentional priority through task instructions and is reduced when these same stimuli are unattended (see Donchin & Coles, 1988, for a review). In this way, the reduced P300 during the fear-induction condition suggests that fear interferes with attentional

[2]The ERN was also analyzed using a peak-to-peak measure. Specifically, the most positive peak in the 0–100-ms preresponse window was subtracted from the most negative peak in the 0–100-ms postresponse window. The results from this analysis yielded results similar to that of the analysis using the area measure of the ERN in that there was no main effect for Experimental Condition, $F(1,15) < 1$, or an interaction between Experimental Condition and Electrode Site, $F(2,30) = 1.57, p > .20$.

**Table 2.** *Mean (Standard Deviation) RT and Accuracy Measures*

| | Control condition | Provocation condition |
|---|---|---|
| Number of errors | 38.50 (30.47) | 37.63 (25.84) |
| Accuracy (% correct) | 93.23 (5.39) | 93.18 (4.70) |
| Error RT (ms) | 334.35 (56.85) | 342.35 (64.28) |
| Correct RT (ms) | 402.21 (50.28) | 400.76 (44.78) |
| RT following errors (ms) | 413.81 (52.87) | 413.80 (42.50) |
| RT following corrects (ms) | 401.10 (49.76) | 399.82 (45.83) |
| Accuracy following errors (% correct) | 93.86 (6.50) | 94.43 (6.50) |

allocation to task relevant stimuli. This would suggest that the fear-relevant stimulus was competing for attentional resources with the task relevant stimuli and partially drained resources that would have otherwise been allocated to the foreground flankers task. These data are consistent with the idea that motivationally relevant stimuli attract attentional resources and thus decrease the processing of other information (e.g., Schupp et al., 1997). This P300 finding is also consistent with previous reports relating higher levels of state anxiety to reduced P300 amplitudes in PTSD patients (Metzger et al., 1997) as well as results suggesting that OCD patients are characterized by smaller P300 amplitudes than normal controls (Beech et al., 1983; Ciesielski et al., 1981; Towey et al., 1994). The present data, however, extend the results of the previous studies by suggesting that affective distress may play a causal role in reducing attentional allocation to task-relevant stimuli, with the implication that in anxious subjects it is the experience of affective distress that relates to the observed attentional deficits.

In addition to the reduced P300, we also found an attenuated Pe during the fear-induction condition. The Pe is thought to reflect late error processing related to the awareness of errors or error salience (Falkenstein et al., 1995, 2000; Nieuwenhuis et al., 2001). Thus, the present data suggest that fear also reduces later indices of error processing such that errors were perhaps less salient and that participants may have been less aware of their mistakes during the fear induction. Similar Pe findings have been found in college students high in trait negative affect (Hajcak, McDonald, & Simons, 2004). Because we did not include specific behavioral (e.g., self report) proxies of error awareness, our conclusions regarding the possibility that the Pe may also reflect differences in error awareness between conditions are preliminary. However, results from a previous study from our laboratory (Hajcak et al., 2003) showing that Pe was related to an increased skin conductance response and greater heart-rate deceleration following an error would suggest that the attenuated Pe in the current study might be understood in terms of diminished orienting to the internal detection of errors during fear induction. Further evaluations of error awareness, anxiety, and the Pe are necessary to better understand their interrelationships.

Based on the morphological, topographical, and temporal characteristics of the P300 and Pe, some researchers have suggested that the Pe and P300 may be manifestations of the same underlying functional system (Davies, Segalowitz, Dywan, & Pailing, 2001; Falkenstein et al., 1991, 2000; Nieuwenhuis et al., 2001). Specifically, both components seem to reflect orienting responses to motivationally significant events. Whereas the P300 appears to reflect such a response to external stimuli, the Pe may reflect a similar orienting response to the internal detection of an error. The fact that both the Pe and P300 have been associated with autonomic indices of orienting (Hajcak et al., 2003; Lagopoulos et al., 1998; Miles, 1992; Simons, Graham, Miles, & Balaban, 1999) and both were reduced in the fear-induction condition provides some additional evidence that these two components are similar.

Unlike the Pe and P300, the ERN was unaffected by the induction of fear. Thus, contrary to results that indicate an enhanced ERN in trait-anxious subjects, the results from the current study indicate that increasing *state* levels of anxiety does not result in an enhanced ERN. These results do not provide support for Luu and Tucker's (2004) tender that the emotional state of negative affect biases the response monitoring system toward producing a larger error signal (ERN). In terms of previous research demonstrating an increased ERN in trait-anxious subjects (Gehring et al., 2000; Hajcak et al., 2003; Hajcak, McDondald, & Simons, 2004; Hajcak & Simons, 2002; Johannes et al., 2001; Luu, Collins, & Tucker, 2000), the present results suggest that the affective state of the subjects, per se, may not relate to an enhanced ERN. Rather, Hajcak, Moser, Yeung, and Simons (2005) suggest that chronically anxious subjects such as those with OCD or Generalized Anxiety Disorder may have enhanced ERNs due to their perfectionistic tendency to overvalue errors. Hajcak et al. (2005) found that the ERN was sensitive to the value of errors, such that more significant errors were characterized by a larger ERN. Considering that affective distress, in both clinical and nonclinical samples, has been associated with increased self-reported concern about making mistakes (Antony, Purdon, Huta, & Swinson, 1998; Enns & Cox, 1999; Flett, Hewitt, Endler, & Tassone, 1995; Frost & Steketee, 1997; Frost et al., 1995), it may be the overvaluation of errors that explains an enhanced ERN in chronically distressed individuals.

Thus, while we concur with Luu and Tucker that contemporary computational theories of the ERN (e.g., Yeung, Botvinick, & Cohen, 2004) must take motivational factors into consideration, the results of the present study do not support the view that a negative affective state "biases" the system toward producing a larger ERN. Instead, we take the view that motivational factors (i.e., overvaluation of errors) moderate the output of the error-detection system such that motivation-related differences may explain the enhanced ERN in "trait" anxious subjects.

It is worth noting, however, that our manipulation involved an induction of a specific fear, and that we assessed the subjects' state only via the SUDS, a fairly global measure of affective distress. Because we do not, for purposes of this study, attempt to differentiate fear from an acute state of anxiety, we did not include assessment measures that attempt to distinguish among fear, anxiety, and other specific negative affective states (e.g., disgust). Along these lines then, it is still possible that other forms of acute affective distress and negative affect may relate to an enhanced ERN in their own right. Furthermore, because we induced fear in a sample of spider-fearful undergraduates, it is

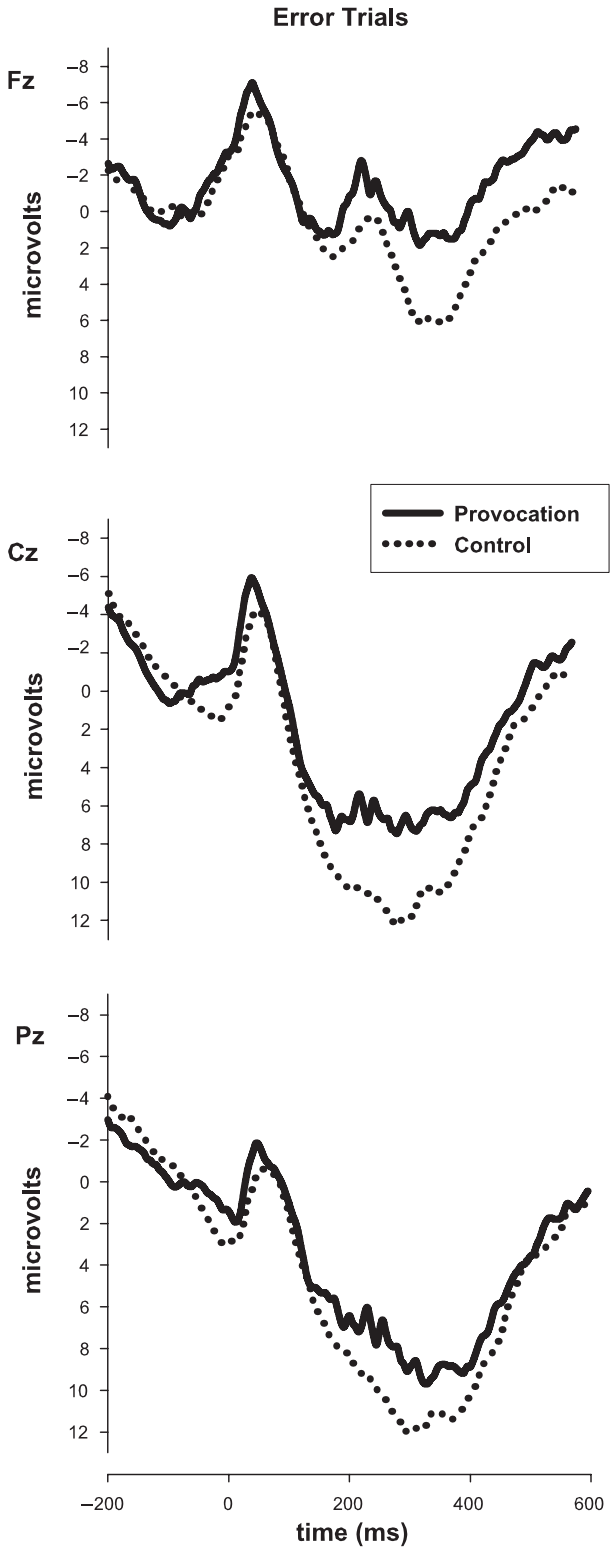


**Figure 1.** Response-locked ERPs at Fz, Cz, and Pz for errors in the control and provocation conditions.

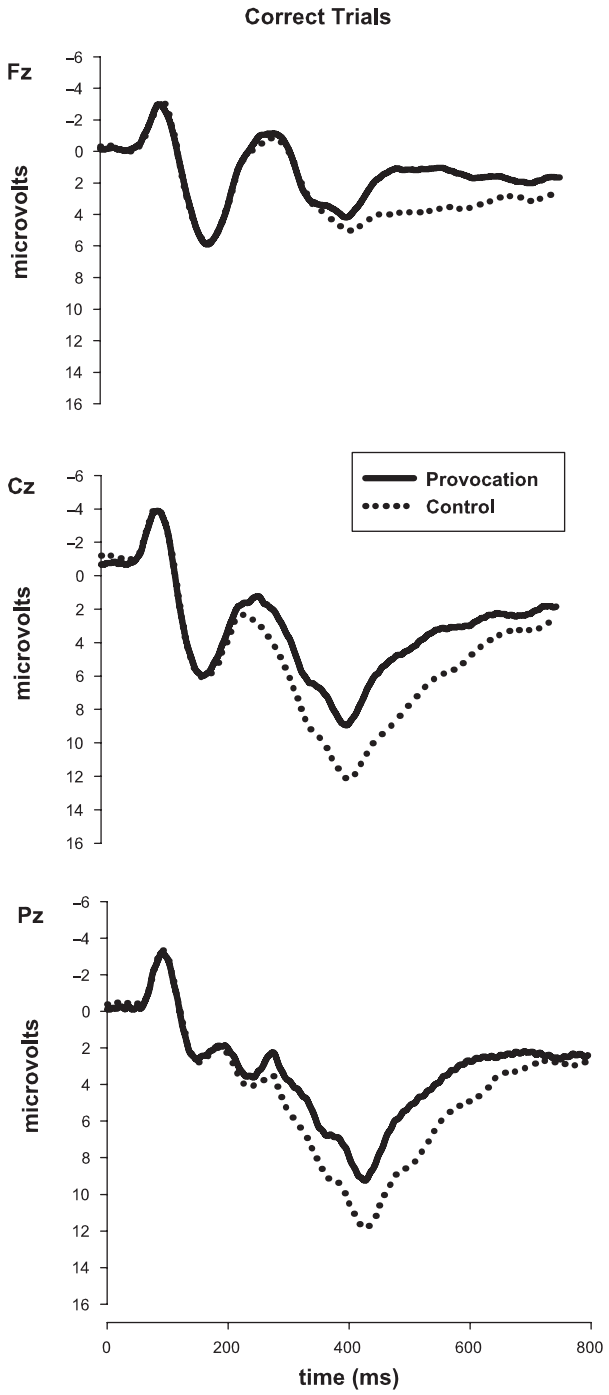


**Figure 2.** Stimulus-locked average ERP waveforms at Fz, Cz, and Pz for corrects in the control and provocation conditions.

possible that these findings might not generalize to the induction of fear in subjects not already fearful.

It is interesting to note that although participants were very distressed during the fear induction, their state of emotional distress did not appear to interfere with their performance on the flankers task. These results are consistent with our previous studies of subjects with high trait anxiety or negative affect (Hajcak & Simons, 2002; Hajcak et al., 2003; Hajcak, McDonald, & Simoms, 2004). Although the behavioral results are inconsistent with the ERP indices of attentional allocation and perhaps counterintuitive, the flankers task is actually quite easy and the reduction of attentional resources suggested by the reduced Pe and P300 in the fear condition may simply suggest excess capacity at the outset and the possibility that performance would have suffered during fear induction if subjects had been given a more difficult task. Nonetheless, the comparable number of

errors, reaction time, post-error reaction time slowing, and post-error accuracy in the two experimental conditions are important data insofar as they rule out behavioral differences as explanations of ERP differences (cf. Hajcak, Vidal, & Simons, 2004; Yeung, 2004; Yeung et al., 2004).

In sum, the P300 and Pe results indicate that the induction of a high-fear state disrupts attentional allocation to task relevant (but fear irrelevant) stimuli and the response to internal error detection, respectively. Thus, the effects of fear were observed both on ERPs related to error processing and to attentional allocation. The fact that both the P300 and the Pe were reduced during fear induction further suggests that these two ERP components are similar. In the context of the P300 data, the unchanged ERN and reduced Pe indicate that early, but not later, error processing is unaffected by attentional differences. The present study further supports the notion that the ERN results from a relatively automatic process and is consistent with Nieuwenhuis et al. (2001), who found that the ERN was unaffected by differences in awareness that altered the Pe. Similarly, the unchanged ERN and reduced Pe during fear induction further substantiates the notion that two independent error monitoring processes exist: an early error-detection process reflected in the ERN and a later error-awareness/error-salience process reflected in the Pe (Nieuwenhuis et al., 2001). Overall, the results of the present study suggest that the experience of affective distress itself may not explain an enhanced ERN in anxious subjects, and emphasizes the need to better understand the relationships between motivation, affect, and performance monitoring.

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