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Error-related psychophysiology and negative affect☆

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Abstract

The error-related negativity (ERN/Ne) and error positivity (Pe) have been associated with error detection and response monitoring. More recently, heart rate (HR) and skin conductance (SC) have also been shown to be sensitive to the internal detection of errors. An enhanced ERN has consistently been observed in anxious subjects and there is some suggestion that the ERN is related to general negative affective experience (NA). The ERN has been source localized to the anterior cingulate cortex—a structure implicated in the regulation of affective, response selection, and autonomic resources. Thus, the findings that autonomic measures and affective distress are related to response monitoring are consistent with anterior cingulate cortex function. In the present experiment, we sought to evaluate more comprehensively the relationship between self-reported negative affect and error-related physiology in a between-groups design. Results indicate that high NA was associated with significantly greater ERN and error-related SCR, and smaller Pe. These results are discussed in terms of anterior cingulate cortex function, psychopathology, and response monitoring.

Keywords: NA, Negative Affect; ERN; ERP; Response monitoring; HR; SCR

1. Introduction

The error-related negativity (ERN or Ne) is a response-locked event-related brain potential (ERP) observed at fronto-central (Fz, FCz, Cz) recording sites that begins around the time of an erroneous response, and peaks 50–100 ms later (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Gehring, Coles, Meyer, & Donchin, 1990; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The ERN has been observed across a variety of stimulus and response modalities, and appears to reflect the activity of a generic response-monitoring system (Bernstein, Scheffers, & Coles, 1995; Falkenstein et al., 1991; Holroyd, Dien, & Coles, 1998; Van 't Ent & Apkarian, 1999). In terms of the source of the ERN, studies utilizing source localization have consistently found that error-related brain activity can be best described by a neural generator in the anterior cingulate cortex (Dehaene, Posner, & Tucker, 1994; Holroyd et al., 1998).

Numerous studies have found that the ERN is also sensitive to motivational and contextual factors. For instance, increased focus on accuracy over speed has been found to increase the magnitude of the ERN; similarly, the ERN appears larger when subjects are more certain that they have made a mistake—suggesting that the response-monitoring system is sensitive to motivational factors during performance (Falkenstein et al., 2000; Gehring et al., 1993).

Individual differences in anxiety have also been found to influence the ERN. Specifically, Gehring, Himle, and Nisenson (2000) found that patients with obsessive-compulsive disorder (OCD) have significantly larger ERNs than age-matched control subjects (for similar results

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in patients with OCD, see Johannes et al., 2001). In studies from our own laboratory, we have likewise reported enhanced error-related brain activity in obsessive-compulsive and worried undergraduates when compared to both control subjects and subjects with specific phobias (Hajcak & Simons, 2002; Hajcak, McDonald, & Simons, 2003b).

These results, linking anxiety to enhanced response monitoring, are consistent with both anxious behavior and the pathophysiology of anxiety. Specifically, OCD and pathological worry have been shown to relate to increased doubts about actions and concerns over mistakes (Frost & Steketee, 1997; Hajcak & Huppert, 2004; Stober & Joormann, 2001). Furthermore, the anterior cingulate cortex has been shown to be hyperactive in many anxiety disorders and this has led some researchers to postulate that anterior cingulate cortex dysfunction may be related to the experience of symptoms common to all anxiety disorders (Kimbrell et al., 1999; Malizia, 1999).

In addition to individual differences in anxiety, an increased ERN-like component to negative feedback has also been found in subjects diagnosed with clinical depression (Tucker, Luu, Frishkoff, Quiring, & Poulsen, 2003). In considering the substantial symptom overlap and diagnostic comorbidity between anxiety and depression, Clark and Watson (1991) proposed that both disorders are characterized by high levels of affective distress, or negative affect (NA). In terms of the differentiation between anxiety and depression, the tripartite model suggests that only depression is characterized by low levels of positive affect (PA), whereas anxiety is uniquely characterized by physiological hyperarousal (Clark & Watson, 1991; for empirical support, see Brown, Chorpita, & Barlow, 1998). Within this framework, it is possible that an enhanced ERN is not a function of either anxiety or depression specifically, but relate to the underlying high NA characteristic of both syndromes.

Some support for this possibility was reported by Luu, Collins, and Tucker (2000), who found significant correlations between NA and ERN amplitude in college students. That is, ERNs were enhanced in college students who were high on self-reported NA. High-NA students in the Luu et al. study, however, had larger ERNs only in the first testing quartile; in fact, the relationship between NA and ERN was in the opposite direction for the remaining three quartiles. Luu et al. interpret this result in terms of task disengagement over time in the high-NA students despite their initial higher than normal concerns with performance. Because the task-disengagement explanation was based solely on an increased reduction in post-error slowing across time in the high-NA group and was not accompanied by any change in performance (e.g., errors or RT), the relationship between ERN and negative affect must be regarded as theoretically plausible, but not well substantiated at this point.

Far less studied than the ERN, the error positivity (Pe) is an ERP component that also appears to be related to response monitoring processes. The Pe has a slightly more posterior scalp distribution, and follows the ERN—peaking approximately 200–400 ms after subjects make a mistake (Falkenstein et al., 2000; Hohnsbein, Falkenstein, & Hoormann, 1989; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001). Falkenstein et al. (2000) suggested that the Pe may index processing that occurs after error detection, such as error recognition or error salience. This possibility was supported by a Nieuwenhuis et al. (2001) study, which reported a relationship between Pe and the subjective awareness of making a mistake. Nieuwenhuis et al. found that when subjects were unaware of their mistakes the ERN was unaffected, but the Pe was substantially reduced. In the several studies that have related ERN to differences in affective variables such as anxiety, there have been no formal evaluations of the anxiety variable and its impact on the Pe. Visual inspection of the relevant data suggests either no differences in Pe (Hajcak et al., 2003b; Luu et al., 2000) or smaller Pe in high-anxious subjects (Gehring et al., 2000; Hajcak & Simons, 2002).

In addition to the ERN-Pe ERP complex, a number of recent studies have reported that autonomic nervous system (ANS) activity is also sensitive to response monitoring. For instance, Somsen, Van der Molen, Jennings, and van Beek (2000) found that cardiac deceleration was related to negative feedback in a Wisconsin card sorting task; similar data relating HR deceleration to negative feedback during response monitoring has also been reported by Crone et al. (2003). Finally, data from our own lab indicates that both HR and SCR are also sensitive to endogenous error detection (Hajcak, McDonald, & Simons, 2003a). Like the Pe, these ANS responses to errors have not been evaluated with respect to individual differences in anxiety or NA.

The present study was conducted to more comprehensively evaluate the relationship between self-reported affective experiences and error-related psychophysiology in a between-groups design. The primary aim was to compare the ERN, Pe and ANS responses from subjects that were either high or low in self-reported NA. A secondary aim was to compare the same variables in subgroups of high-NA subjects with different levels of positive affect (PA). All subjects were selected on the basis of the NA and PA subscales of the Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988). To this end, we measured ERPs and ANS activity while subjects performed a speeded reaction time task in which they had to respond to Stroop stimuli. In particular, subjects saw color words (e.g., ''red'') presented in either a congruent (red) or incongruent (blue) color;

subjects were instructed to respond based on whether or not the color and name of the stimuli matched as quickly and accurately as possible.

2. Method

2.1. Subjects

Undergraduate students in an introductory psychology class completed the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988). 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). All items are rated on a Likert-type scale ranging from 1 (very slightly or not at all) to 5 (extremely). Although the PANAS can be administered in terms of various time frames, subjects responded to PANAS items on the basis of how they felt in general in the present study. The PANAS has been shown to have excellent psychometric properties, especially in terms of the general time frame instructions (see Watson et al., 1988).

Sixty undergraduates (30 male, 30 female) were selected on the bases of their PANAS scores. Twenty subjects who scored low in NA ($M = 5.90$, $SD = 2.46$, PA: $M = 35.75$, $SD = 2.15$) comprised the low-NA group and forty subjects who scored high on NA comprised the high-NA group ($M = 21.1$, $SD = 3.27$, PA: $M = 25.37$, $SD = 6.15$). In order to examine the possible moderating role of PA on subjects with high negative affect, the high-NA group was further divided based on PA scores. Thus, 20 subjects were selected who scored high in NA ($M = 20.38$, $SD = 3.10$) and low in PA ($M = 19.27$, $SD = 3.10$) and another 20 subjects were selected who scored high in NA ($M = 21.68$, $SD = 3.53$), but also had moderate to high scores in PA ($M = 30.31$, $SD = 2.15$). High-NA/Low-PA subjects, according to the tripartite model, display a depression profile, while High-NA/High-PA subjects are thought to evince an anxiety profile.

All subjects received course credit for their participation and the experimenter was blind to group membership until data reduction was complete.

2.2. Task

The Stroop-like 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'', or ''blue'') presented either in red, green, or blue font against a black background. Each word occupied approximately 3 degrees of visual angle, and all words were positioned in the center of the screen. A fixation mark (+) was presented just prior to the onset of each stimulus. As opposed to more traditional Stroop tasks in which subjects are required to respond to one stimulus dimension (e.g., color) while ignoring the other (e.g., color name), subjects in the current experiment were instructed to respond to whether or not the color and name of the stimulus matched. Specifically, subjects were instructed to press one mouse button when the color and name of the stimulus were the same, and press the other mouse button when the color and name of the stimulus were different. In this way, the task contained matching conditions (when color and name of the word were the same) and mismatching conditions (when color and name of the word were different). The stimuli were presented randomly such that 50% of trials were matching stimuli.

2.3. Procedure

After a brief description of the experiment, EEG/EOG, SCR and HR sensors 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 2 blocks of 48 practice trials. In one condition, the subjects were told to press the left mouse button when the name of the color matched the color of the font, and the right mouse button when the name of the color and color of the font did not match. In the other condition, the correspondence between mouse button and stimulus congruence was reversed. These conditions were counter-balanced across subjects. The subjects were told to place equal emphasis on speed and accuracy in their responses. Following practice, the subjects received 12 blocks of 48 trials (576 total trials) with each block initiated by the subject. Stimuli were presented for 200 ms at random intervals between 5300 and 5700 ms. After subjects completed this task, they were also asked to complete the 21-item version of the Depression, Anxiety, and Stress Scales (DASS-21; Lovibond & Lovibond, 1995) to verify differences in depression between the two high-NA groups.

2.4. Psychophysiological recording, data reduction and analysis

The electroencephalogram (EEG) was recorded using a Neurosoft Quik-Cap. Recordings were taken from 3 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 EEG/EOG electrode impedances were below 10K ohms and the data from all channels were recorded by a Grass Model 7D polygraph with Grass Model 7P1F preamplifiers (bandpass = 0.05–35 Hz).

Heart-rate was obtained by attaching a Grass Photoelectric Transducer Model PPS to the subject's left ear lobe. The photocell output was fed into a Grass Model 7P1 Low Level DC Preamplifier and Model 7D Driver Amplifier (Bandpass = 1.6–3.0 Hz) and then into a series of Coulbourn logic modules that did threshold detection and shaping prior to the online computation of interbeat intervals.

Skin conductance responses were recorded using a Coulbourn Model S21–22 constant voltage (0.5 V) skin conductance coupler. Med Associates Standard (0.5 cm^2) Ag/AgCl electrodes were placed on the thenar and hypothenar eminence of the palm with Johnson & Johnson KY Jelly used as an electrolyte.

All bioelectric signals were digitized on a laboratory microcomputer using VPM software (Cook, 1999). The EEG was sampled at 200 Hz. Data collection began 1500 ms prior to stimulus presentation and continued for 5000 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 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 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 200 ms window prior to the response. The ERN was then defined as the most negative peak occurring in a window from 0 to 100 ms post-response. The Pe was defined as the average activity in the 200–400 ms window following response execution (Nieuwenhuis et al., 2001).

Interbeat intervals obtained from the photocell were converted to heart-rate in beats per minute per real-time epoch (250 ms). When epochs contained portions of two beats, each rate was weighted according to the fraction of the epoch that it occupied (Graham, 1978). Heart-rate waveforms were then generated by deviating quarter-second averages during a 3.0 s post-response epoch from the quarter-second average immediately preceding stimulus onset. Twelve (3 s) quarter-second averages, along with the onset point, constituted the heart-rate data that were then submitted to statistical analysis.

Skin conductance was sampled at 50 cps. Although the short poststimulus recording epoch did not allow for the development of a discrete SCR in most instances, the epoch was quantified by visually identifying activity that began with an onset latency greater than 0.5 s post response and measuring the difference, in uSiemens, between the identified SC onset point and the maximum SC value present in the 3.0 s post-response window.

Because uniformly fast reaction times can give rise to stimulus-related activity in the response-locked ERN, and because correct trials vastly outnumber incorrect trials, the ERN and the two autonomic measures were evaluated for errors and a sub-set of correct trials that were matched to error trials on the basis of reaction times. In addition to equating the number and speed (RT) of error and correct trials, this matching procedure allowed a comparison of post-error slowing with potential RT-slowing after equally fast correct trials.

The ERN, ANS and performance measures were statistically evaluated using SPSS (Version 10.1) General Linear Model software with the Greenhouse-Geisser correction applied to the p values of multiple df repeated measures comparisons.

3. Results

3.1. Personality measures

A number of analyses were conducted to confirm that subject assignment resulted in distinct groups and subgroups. In terms of the PANAS scores, there was a highly significant difference in NA between the forty high-NA and 20 low-NA subjects ($t(58) = 17.87$, $p < .001$). The two groups also differed on PA, with the high-NA group scoring lower on PA than the low-NA group ($t(58) = 7.29$, $p < .001$). These two groups also differed from each other on both the depression ($t(58) = 4.71$, $p < .001$) and anxiety ($t(58) = 2.11$, $p < .05$) subscales of the DASS-21, suggesting that the high-NA subjects were more psychologically distressed than the low-NA subjects.

Similar comparisons were made between the two subgroups of the high-NA subjects. The two groups were equivalent on the NA measure ($p > .20$), they differed significantly, as intended, on PA ($t(38) = 13.31$, $p < .001$), but did not differ on either the depression ($p > .15$) or the anxiety ($p > .95$) subscale of the DASS-21. Thus, contrary to predictions derived from the tripartite model, PA scores did not moderate the relationship between high-NA and self-reports of either depression or anxiety. Likewise, the two subgroups of high-NA subjects did not differ on any performance measure or on any of the physiological measures obtained as part of this investigation. Consequently, details of these various comparisons will not be further described and the remainder of the presentation will involve the more important comparison of subjects characterized by either high- or low negative affect.

Eight subjects from the high-NA group and seven subjects from the low-NA group were not included in the data analysis due to near-perfect task performance (subjects who made fewer than 15 commission errors). An additional 12 subjects (eight high-NA and four low-NA) were eliminated from the analysis of the skin conductance data because they produced no visible SCR on any error or matched-RT correct trial.

3.2. Performance measures

Performance measures as a function of high and low NA are presented in Table 1. In terms of reaction time (RT), subjects tended to be faster on error trials than on correct trials ($F(1,43) = 90.64$, $p < .001$); however, there were no RT differences between the high- and low-NA groups ($F(1,43) < 1$), and no interaction between group and trial type in terms of RT ($F(1,43) = 2.37$, $p > .10$). Additionally, there were no differences between the high- and low-NA groups in terms of number of errors ($F(1,43) = 1.69$, $p > .20$).

Many studies have noted significant RT slowing on trials that follow errors, and this post-error slowing is thought to represent a compensatory mechanism to increase performance on post-error trials (Gehring & Fencsik, 2001; Rabbit, 1981). However, in previous studies we have found that there is also significant RT slowing following correct trials that are RT-matched to error trials, suggesting that at least some post-error slowing is due to regression toward the mean (Hajcak et al., 2003b). Thus, in the present study, we computed post-error RT slowing and compared it to RT slowing after equally fast correct trials. This analysis indicated that slowing was significantly greater after error trials ($F(1,43) = 4.78$, $p = .034$), but again, post-error slowing did not differ between groups ($F(1,43) < 1$). Likewise, in terms of post-error accuracy, there was no significant between-groups difference ($F(1,43) = 1.25$, $p > .25$). Thus, consistent with our previous studies on anxious subjects, there was no evidence of performance differences between the high- and low-NA subject groups.

Table 1
Mean RT in ms (and *SD*) and accuracy as percent correct (and *SD*) for high and low NA subjects

	High NA	Low NA
Errors	18.81 (11.99)	25 (19.49)
Error RT	574 (70)	546 (81)
Correct RT	640 (46)	637 (73)
RT following errors	665 (61)	661 (92)
RT following RT-matched correct trials	635 (70)	648 (113)
Accuracy following errors	96.6 (4.6)	94.6 (7.1)
Accuracy following RT-matched correct trials	95.6 (5.9)	93.5 (7.1)

3.3. ERP measures

The response-locked average ERP waveforms at Fz, Cz, and Pz for all errors and RT-matched correct trials for high-NA and low-NA subjects are presented in Fig. 1.

As anticipated, a negative deflection associated with error trials began shortly after response execution and peaked approximately 55 ms later. A 2 (Group) × 2 (Trial Type Type) × 3 (Electrode Site) analysis of variance (ANOVA) confirmed that the ERN was significantly greater on error trials than on correct-response trials ($F(1,43) = 13.72$, $p < .001$). The interaction of Trial Type and Site was also significant ($F(2,86) = 4.64$, $p < .05$) with the difference in the ERN magnitude between correct and incorrect trials depicted in Fig. 1 smallest at Fz and increasing at the Cz and Pz recording sites. Thus, though somewhat idiosyncratic, our results are generally consistent with previously reported ERN morphology and topography.

More importantly, there was a significant main effect for group ($F(1,43) = 6.57$, $p = .014$) indicating that high-NA subjects had enhanced negative deflections in the time window of the ERN relative to low-NA subjects. There was no significant interaction between Group and Trial Type ($F(1,43) < 1$), indicating enhanced activity on both error and correct trials. In addition, there were no interactions between Group and Location ($F(2,86) = 3.38$, $p > .05$), nor among Group, Trial Type, and Location ($F(2,86) = 1.81$, $p < .20$).

Pe had a fronto-central scalp distribution ($F(2,86) = 65.61$, $p < .001$) and was also significantly associated with errors ($F(1,43) = 77.27$, $p < .001$). Like the ERN, there was a significant main effect for Group ($F(1,43) = 13.54$, $p < .001$), confirming that post-response positivity in the 200–400 ms window was smaller in the high-NA group. Again, there was no significant interaction between Group and Trial Type ($F(1,43) < 1$), indicating that the smaller Pe in the high NA group was associated with both error and correct trials. Again, there were no significant interactions between Group and Location ($F(2,86) < 1$), nor among Group, Trial Type, and Location ($F(2,86) < 1$).

3.4. ANS measures

Fig. 2 illustrates both the skin-conductance (bottom) and heart rate data (top) associated with error and correct trials. Analysis of variance on the magnitude of the SCR on error trials and RT-matched correct trials revealed a highly significant effect of Trial Type ($F(1,31) = 25.40$, $p < .001$) and a marginally significant Group × Trial Type interaction ($F(1,31) = 3.22$, $p < .10$). Because there was virtually no SCR on correct trials in either group, we sought to examine the effect of NA in more detail by computing between-group t-tests on error trials. Consistent

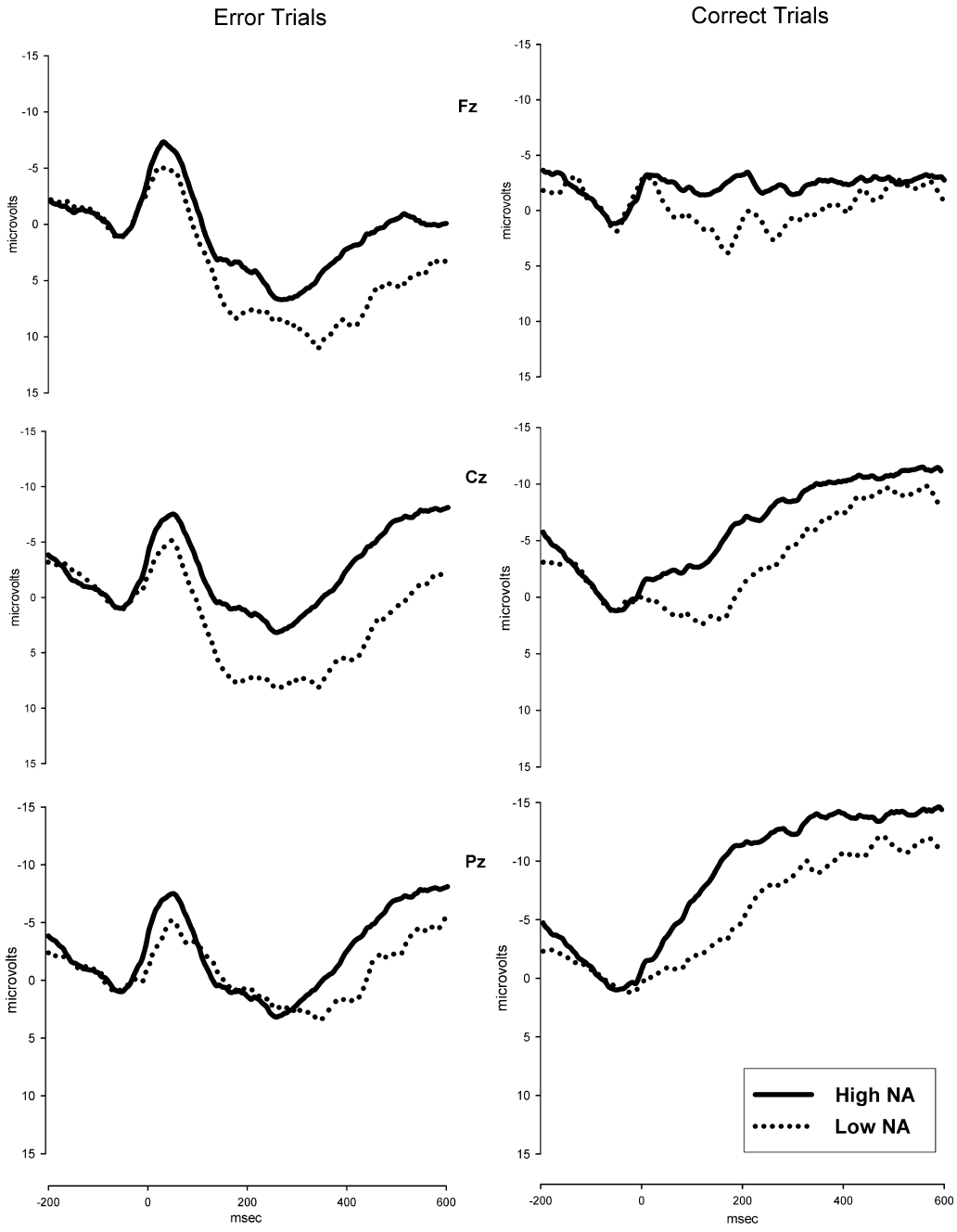

Fig. 1. Response-locked ERPs at Fz, Cz, and Pz for errors (left) and RT-matched correct (right) trials for high- and low-NA subjects.

with the impression gleaned from Fig. 2, high-NA subjects produced larger SCRs in response to errors than low-NA subjects ($t(31) = 2.24$, $p < .01$).

To evaluate heart-rate slowing, average HR for both error and RT-matched correct trials was subjected to a 2 (Trial Type) × 11 (Time) repeated measures ANOVA with orthogonal polynomial contrasts used to evaluate the Time factor. As illustrated in Fig. 2, the trend analysis revealed a significant main effect for Trial Type ($F(1,43) = 32.27$, $p < .001$) with error trials prompting more deceleration than correct trials. This difference between error and correct trials was reflected by significant linear ($F(1,43) = 41.53$, $p < .001$), quadratic ($F(1,43) = 7.51$, $p = .009$), and cubic ($F(1,43) = 16.37$, $p < .001$) Trial Type × Time interactions. Despite the impression from Fig. 2 that, like SCR, high-NA subjects responded to errors with more HR deceleration than low-NA subject, this was not confirmed statistically (Group × Trial Type × Time $F_{quad}(1,43) = 1.84$, $p < .20$).

3.5. ERN, performance, and time

A subset of subjects (eight high- and 20 low-NA) with sufficiently high error rates were employed to examine

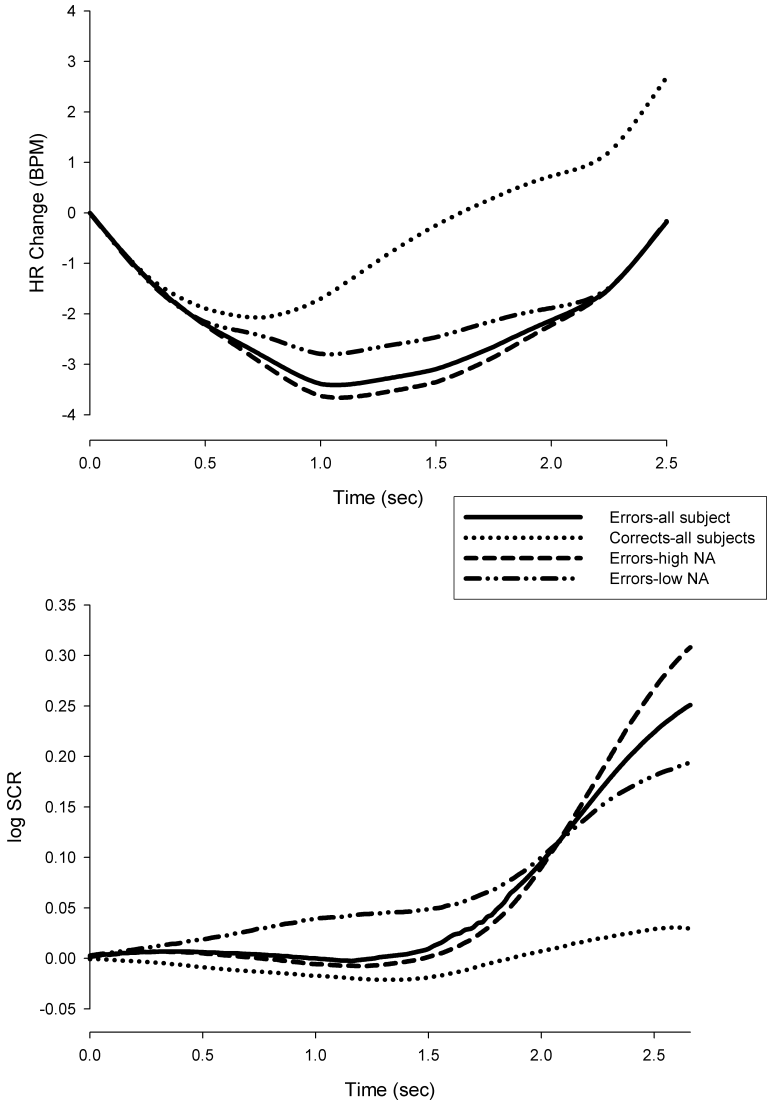

Fig. 2. Averaged and heart-rate (top) and skin-conductance response (bottom) waveforms for error trials and RT-matched correct trials for all subjects, and error trials for high- and low-NA subjects.

the ERN and Performance measures across halves of the experimental session. ERN, Pe, error-rate and post-error RT slowing were analyzed as a function of Group and Session Half. On none of the measures were there significant effects of Half or significant Group × Half interactions.

4. Discussion

We found that college students scoring high on self-reported negative affect were characterized by enhanced ERN and CRN, larger error-related SCR, and reduced post-ERN/CRN positive activity. These results are consistent with Luu et al. (2000), who report evidence for a relationship between ERN and NA in college students. Also consistent with Luu et al., we found that within the context of high NA, PA did not have a moderating role on response monitoring psychophysiology. Because only NA is characteristic of both anxiety and depression, the current study fits well with a growing body of studies relating anxiety and depression to enhanced error-related brain activity (Gehring et al., 2000; Hajcak & Simons, 2002; Hajcak et al., 2003b; Johannes et al., 2001; Luu et al., 2000; Tucker et al., 2003).

We have consistently found that both the ERN and correct-response negativity (CRN) are larger in affectively distressed groups (obsessive-compulsive, worried, and now high NA). The CRN appears as a small ERN on correct trials, and has the same temporal characteristics and scalp topography as the ERN (see Fig. 1; also Vidal, Hasbroucq, Grapperon, & Bonnet, 2000; Vidal, Burl, Bonnet, Grapperon, & Hasbroucq, 2003). Although a complete discussion of the CRN is beyond the scope of the present study, it has been suggested that *both* the CRN and ERN reflect the engagement of the

response monitoring system, and signal a need for increased response control (Ridderinkhof, Nieuwenhuis, & Bashore, 2003; Ridderinkhof, Nieuwenhuis, Hajcak, van den Wildenberg, & Burle, 2004). In terms of this conceptualization, NA appears related to increased engagement of the response monitoring system, evident on both correct and error trials.

The ERN has consistently been source-localized to the anterior cingulate (Bush, Luu, & Posner, 2000). Insofar as the ERN is an index of anterior cingulate function, the present study suggests that NA is associated with anterior cingulate cortex hyperactivity. The anterior cingulate has also been described as an important structure of the central autonomic network (CAN; Benarroch, 1993, 1997). Thayer and Lane (2000) propose that the CAN regulates attentional, affective, autonomic, and response selection resources. Thus, the finding that high NA subjects had greater error-related SCRs is also consistent with anterior cingulate cortex function and hyperactivity.

In addition to an increased ERN/CRN and error-related SCR, high-NA subjects also had reduced post-response positive activity in the 200–400 ms window. Thus, the high-NA subjects had both smaller Pe, and reduced positive activity on correct trials in the same time window. The Pe appears to index subsequent response monitoring processes such as error awareness (Nieuwenhuis et al., 2001); similarly, it has been suggested that the Pe is related to error salience (Falkenstein et al., 2000). In these terms, the present study suggests that subjects high in NA may find their errors less salient or be less aware of their errors than low-NA subjects; furthermore, the present data indicate that a similar difference also exists on correct trials, perhaps reflecting reduced processing following correct responses. Although we did not collect self-report data, this possibility is consistent with Luu et al.'s finding that high-NA students report less awareness of having made mistakes. Thus, differences in ERN and Pe in high-NA subjects may reflect two dysfunctional processes: enhanced response monitoring and decreased error expectancy or awareness, respectively.

Consistent with our previous studies on anxious subjects, we found that the high- and low-NA groups did not differ with respect to any performance measure. Specifically, the high-NA and low-NA groups had comparable number of errors, reaction time, and post-error reaction time slowing. Yeung (2004) proposes that between-group ERN differences could be explained in terms of differences in basic information processing (e.g., differences in accuracy or RT). However, the present study presents between-group ERN differences in high-NA subjects despite a lack of performance differences.

The construct of NA has significant implications for psychopathology research. In previous studies, NA has been related to health complaints, perceived stress, and the experience of unpleasant events (see Clark & Watson, 1991, for a review). To this list, the present study adds response monitoring abnormalities. It is interesting to note that NA has also been related to perfectionistic concern over mistakes (Frost, Heimberg, Holt, Mattia, & Neubauer, 1993). The present study, then, may provide evidence for response monitoring abnormalities that underlie maladaptive perfectionistic concerns.

In sum, we have suggested that high NA and increased response monitoring can both be understood in terms of underlying anterior cingulate cortex hyperactivity. Specifically, we proposed that the increased ERN and CRN might be understood in terms of the over-engagement of the response monitoring system in the high-NA group on both error and correct trials. Additionally, it is possible that the smaller Pe in the high-NA group is related to reduced error awareness. Finally, the finding that errors were associated with increased arousal in the high-NA group also makes sense in terms of the anterior cingulate cortex's role in regulating affective and autonomic resources. Taken together, these data demonstrate that the effects of NA may be observable in both early (ERN, Pe) and later (SCR) indices of response monitoring. It should be noted, however, that causal inferences relating NA and abnormal psychophysiological indices of response monitoring are premature at the present time. Future studies should address this issue, perhaps by manipulating affective state and measuring subsequent changes in psychophysiological measures related to response monitoring. Finally, future studies may wish to explore response monitoring abnormalities in the context of other concepts related to psychopathology research, such as perfectionism, symptom profiles, or even treatment outcome research.

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