



Error-preceding brain activity: Robustness, temporal dynamics, and boundary conditions[☆]

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Abstract

A recent study reported a positive modulation of the response-locked event-related brain potential (ERP) on trials *preceding* errors (i.e., error – 1 trials). It was proposed that this error-preceding positivity (EPP) reflects the disengagement of the response monitoring system prior to errors. In three studies, we sought to replicate the EPP, and to delineate the conditions under which it can be observed. Study 1 replicated the finding of a positive modulation of the ERP on error-preceding compared to RT-matched correct-preceding trials. This enhanced positivity was not due to differences in stimulus-related processing, and a similar modulation was not observed on error – 2 or error + 1 trials. Studies 2 and 3 indicated that similar EPP-like phenomena could be observed across a variety of tasks, and using much longer inter-trial intervals. The onset and duration of the modulation, however, did vary between studies. These results are discussed in terms of response-locked ERPs and action monitoring.

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Studies of response monitoring in humans that measure event-related brain potentials (ERPs) during speeded reaction time tasks consistently find a sharp negative deflection at fronto-central recording sites in the response-locked ERP that begins around the time of an incorrect response and peaks approximately 80 ms later (Gehring et al., 1993; Dikman and Allen, 2000; Luu et al., 2000; Falkenstein et al., 2000; Scheffers and Coles, 2000; Nieuwenhuis et al., 2001). This component is referred to as the error negativity (Ne: Hohnsbein et al., 1989) or error-related negativity (ERN; Gehring et al., 1990). The ERN is thought to reflect the activity of a general error-processing system, active across stimulus and response modalities

(Holroyd et al., 1998; Van't Ent and Apkarian, 1999; Nieuwenhuis et al., 2001). Using whole head recording systems, the ERN has been source-localized to the anterior cingulate cortex (ACC; Dehaene et al., 1994; Holroyd et al., 1998). Data utilizing both magnetoencephalography (MEG) and functional magnetic resonance imaging (fMRI) corroborate the view that the ACC is activated when subjects make errors (Kiehl et al., 2000; Garavan et al., 2002).

A small ERN-like wave in the response-locked ERP has also been observed on correct trials (Falkenstein et al., 2000; Vidal et al., 2000; Coles et al., 2001; Vidal et al., 2003), and has been referred to as the correct-response negativity (CRN; Ford, 1999). The CRN has a scalp topography and morphology that is similar to the ERN, and it has been suggested that the two components may reflect the activity of the same response monitoring system (Vidal et al., 2000).

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Behaviorally, errors are typically followed by relatively slow trials. This post-error slowing is thought to reflect a compensatory adjustment that minimizes the risk of subsequent errors (Rabbitt, 1966; Laming, 1968). Relating the ERN to these behavioral data, Gehring et al. (1993) found that trials characterized by relatively large ERNs were followed by trials with the most post-error slowing. A recent neuroimaging study found that trial-to-trial changes in ACC activity predicted subsequent performance changes (Kerns et al., 2004). Similarly, Ridderinkhof and colleagues examined performance measures on trials that followed small and large CRN trials, and found that trials following large-CRN trials were more accurate, and characterized by less interference from irrelevant stimulus dimensions than trials that followed small-CRN trials (Ridderinkhof et al., 2004a). These authors proposed that CRN amplitude may reflect the level of engagement of the response monitoring system on a trial-by-trial basis, and may thus be used as a predictor of subsequent performance.

In support of this possibility, Ridderinkhof et al. (2003) examined ERP activity in the time window of the ERN on error-preceding and correct-preceding trials, and found that error-preceding trials were characterized by enhanced positive activity at Fz in the 0–100 ms time window following the subjects' response. This error-preceding positivity (EPP) was interpreted in terms of a neural index of "transient deficiencies in the functioning of the monitor system prior to actual execution of an error" (p. 3). In other words, the EPP may reflect the disengagement of the ACC and this disengagement may be a causal antecedent to subsequent errors.

A limitation of the Ridderinkhof et al. (2003) study was its small ($n = 7$) sample size. The goal of the current research was to replicate and extend the finding of an EPP on error-preceding trials using a much larger data set. A second goal was to rule out alternate interpretations of the response-locked EPP. To this end, we carried out analyses to rule out the possibility that the EPP reflects the differential contribution of stimulus-evoked EEG activity to the response-locked ERPs associated with error-preceding and correct-preceding trials. Additionally, we investigated whether the occurrence of the EPP is specific to trials immediately preceding errors or whether a similar modulation might be evident as early as the error – 2 trial. Likewise, we sought to determine whether the EPP is characteristic of error-surrounding trials, including trials that follow errors. A final goal of the present research was to evaluate error-preceding brain activity in a variety of experimental paradigms. To this end, we analyzed data from 75 participants that were collected using different tasks and procedures. This allowed us to test the robustness of the EPP using different experimental paradigms, with different intertrial intervals. Specifically, error-preceding brain activity was evaluated in both flankers and Stroop tasks, with intertrial intervals that ranged from 1.5 to nearly 6 s. These issues were addressed in three different studies, two of which

(Studies 2 and 3) involved the reanalysis of previously published data sets.

1. Study 1

In Study 1, participants performed an arrows version of the Eriksen flankers task, similar to the task used by Ridderinkhof et al. (2003). The primary objective of Study 1 was to replicate the finding of an EPP on error-preceding trials. We also examined whether response-locked error-preceding ERP differences might be due to differences in the stimulus-evoked ERPs on error-preceding trials (cf. Coles et al., 2001). To this end, we examined stimulus-locked ERPs on error-preceding and correct-preceding trials. Lastly, we evaluated the temporal dynamics of the EPP; we sought to determine whether the EPP was specific to error – 1 trials by investigating whether a similar modulation could be observed on error – 2 trials and trials following errors (i.e., error + 1 trials).

1.1. Method

1.1.1. Participants

Fourteen undergraduate students in an upper-level psychology course participated in the current experiment, and received extra-credit for their participation. All participants provided informed consent prior to participating.

1.1.2. Task and procedure

An arrows version of the Eriksen flankers task (Eriksen and 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, and the measurement of response accuracy and reaction times (RTs). During the task, participants were shown sets of five arrowheads (<<<<<, <<><<, >><<, >>>> or >><>>), and were instructed to press the left or right mouse button in accordance with the direction of the center arrowhead. There were two compatible stimuli (<<<<< and >>>>>) and two incompatible stimuli (<<><< and >><>>). The stimuli were presented randomly such that 50% of the trials were congruent. The stimuli were presented in white against the black background of the computer screen. Each stimulus was presented for 200 ms, and successive stimuli were separated by random interstimulus 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.

After participants received a general description of the experiment, each participant was given two blocks of 48 practice trials. Following the practice blocks, participants received 12 blocks of 48 trials (576 trials). Participants were

instructed to respond as fast as possible while preventing errors.

1.1.3. Psychophysiological recording, data reduction and analysis

The electroencephalogram (EEG) was recorded from frontal (Fz), central (Cz), and parietal (Pz) midline recording sites using a Neurosoft Quick-Cap (Compumedics USA Ltd.). In addition, tin disc electrodes were placed on the left and right mastoids. During the recording, all activity was referenced to Cz. To monitor for blinks and vertical eye movements, we recorded the electro-oculogram (EOG) using Med-Associates miniature electrodes placed approximately 1 cm above and below the participant's right eye. The right earlobe served as a ground site. All EEG/EOG electrode impedances were kept below 10 k Ω 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. Offline, the EEG for each trial was corrected for vertical EOG 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 analyses if the signal fell out of the range of the analog-to-digital converter or if the signal was flat for 25 ms or longer. Additionally, trials were not included in ERP averages if the response time fell outside of a 200–800 ms window. Each error-preceding trial was RT-matched to a correct-preceding trial with the most similar RT using a computer algorithm.¹ The same matching algorithm was used for error + 1 and error – 2 trials. Finally, the EEG for each trial was time-locked to either stimulus onset or reaction time and averaged across trials to yield stimulus- and response-locked ERPs associated with each electrode site. The EPP was quantified as the average activity in the 0–100 ms post-response window relative to the average activity in a 100-ms window prior to the response (cf. Ridderinkhof et al., 2003). Measurements of the EPP and other indices were statistically evaluated using SPSS (version 10.0) General Linear Model software with the Greenhouse-Geisser correction applied to *p*-values associated with multiple degrees of freedom repeated measures comparisons.

1.2. Results

1.2.1. Task performance

On average, participants made 36.1 errors (S.D. = 19.4), corresponding to 6.3% (S.D. = 3.4) of the trials. Consistent

¹ The algorithm began with the first error-preceding trial and found the correct-preceding trial that was closest in RT. It repeated this process for each subsequent error-preceding trial, using the remaining correct-preceding trials for matching.

with previous studies, RTs on error trials ($M = 371$ ms, S.D. = 41 ms) were significantly faster than RTs on correct trials ($M = 460$ ms, S.D. = 37 ms; $t(13) = 14.70$, $p < .001$).

1.2.2. Error – 1 trials

The RT-matching procedure was successful: RTs for error-preceding trials ($M = 419$ ms, S.D. = 34 ms) did not differ from RTs for RT-matched correct-preceding trials ($M = 420$ ms, S.D. = 34 ms; $t(13) = 1.60$, $p > .10$).² Response-locked ERPs for error-preceding and RT-matched correct-preceding trials are presented in Fig. 1. In addition, the average signal values in the 0–100 ms post-response window are presented in Fig. 2. These measurements were submitted to a 2 (trial type: error-preceding versus correct-preceding) \times 3 (electrode site: Fz, Cz, Pz) repeated measurements ANOVA.

Consistent with the report by Ridderinkhof et al. (2003), error-preceding trials were characterized by enhanced positive activity in the 0–100 ms post-response window, as indicated by a significant main effect of trial type ($F(1, 13) = 4.84$, $p < .05$). The interaction between trial type and electrode site was not significant ($F(2, 26) < 1$), consistent with the observation that the EPP modulation did not differ much among the three electrode sites.

To rule out the possibility that the observed EPP was due to differences in stimulus-related ERP components such as the P300, we examined the stimulus-locked ERPs associated with error-preceding and correct-preceding trials. As Fig. 3 shows, there were no systematic differences in these stimulus-locked ERPs that could explain the EPP in the response-locked ERPs.

1.2.3. Error + 1 trials

Because trials that follow errors are generally characterized by substantial post-error slowing, we were unable to successfully match RTs for error-following trials with RTs for correct-following trials. The RTs for error-following trials ($M = 495$ ms, S.D. = 47 ms) were significantly longer than the RTs for the correct-following trials selected by the matching algorithm ($M = 441$ ms, S.D. = 38 ms; $t(13) = 6.25$, $p < .001$). The response-locked ERPs associated with error + 1 trials and correct + 1 trials are presented in Fig. 4 (left panel). An ANOVA with factors trial type and electrode site indicated that error + 1 trials and

² RT-matched correct-preceding trials were significantly faster than the mean reaction time on correct trials ($t(13) = 3.00$, $p < .05$). This difference indicates that error-preceding trials (and therefore RT-matched correct-preceding trials) were relatively fast trials, and highlights the potential importance of RT-matching. Additionally, the number of incompatible correct-preceding trials ($M = 12.7$, S.D. = 7.2) did not differ from the number of incompatible error-preceding trials ($M = 11.4$, S.D. = 7.3; $t(13) = 1.49$, $p > .15$). Thus, ERP differences between correct-preceding and error-preceding trials cannot be accounted for by potential differences related to the number of incompatible flanker trials included in the analyses.

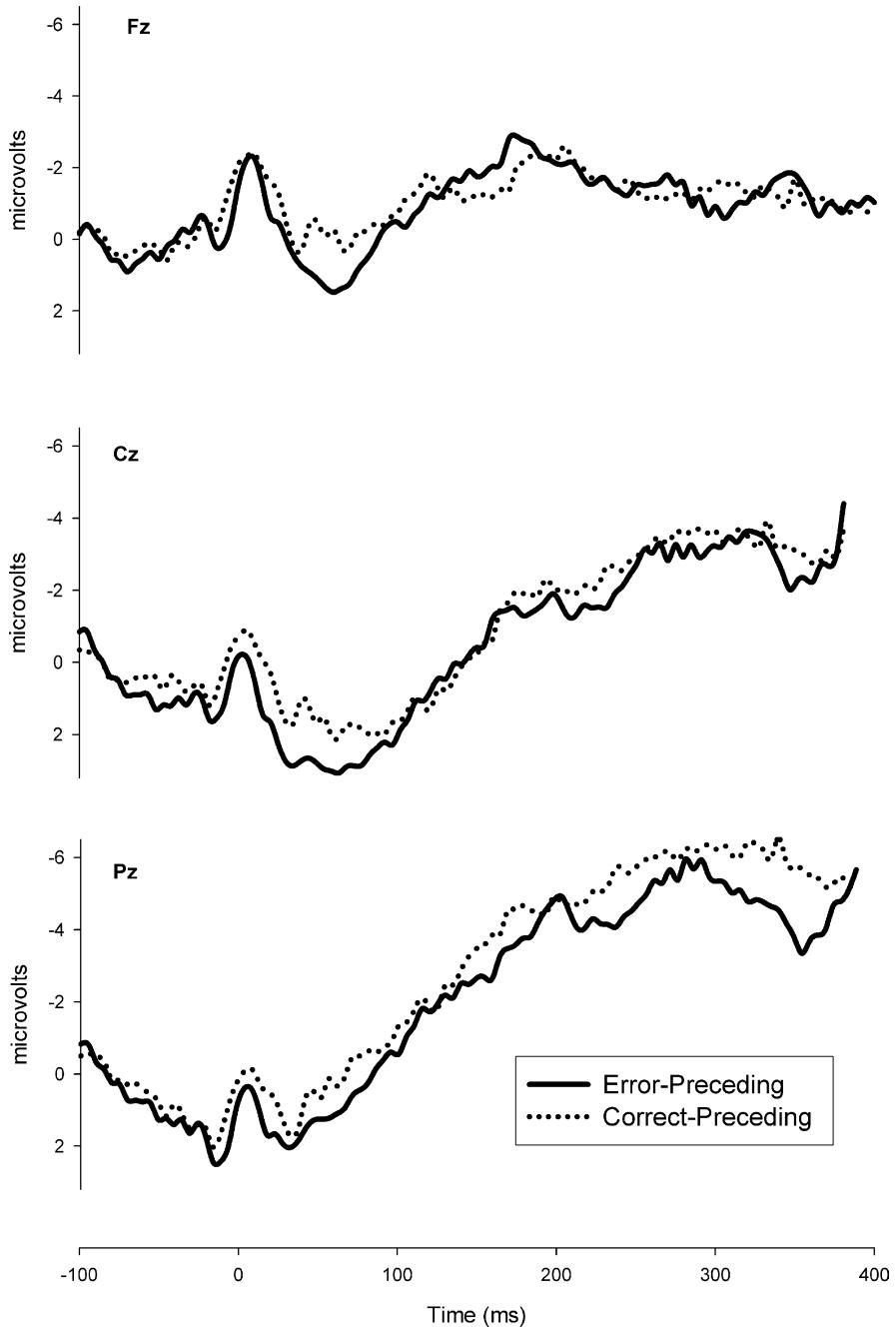


Fig. 1. Response-locked ERPs at Fz, Cz, and Pz for error-preceding trials and RT-matched correct-preceding trials in Study 1.

correct + 1 trials did not differ in the 0–100 ms post-response window ($F(1, 13) < 1$). There was no significant interaction between trial type and electrode site ($F(2, 26) < 1$).

1.2.4. Error – 2 trials

The aim of this analysis was to investigate whether the EPP was specific to trials that directly preceded errors, or if a similar modulation could be observed two trials prior to errors. The RT-matching procedure was successful: RTs

for error – 2 trials ($M = 439$ ms, S.D. = 36 ms) did not differ from RTs for RT-matched correct – 2 trials ($M = 439$ ms, S.D. = 37 ms; $t(13) = 1.07$, $p > .30$). Fig. 4 (right panel) presents response-locked ERPs for error – 2 and correct – 2 trials. Although, the waveforms of the error – 2 trials appear slightly more positive than the correct – 2 waveforms in the 0–100 ms post-response window at the central and parietal recording sites, an ANOVA with factors trial type and electrode site indicated that any difference between the two trial types was not

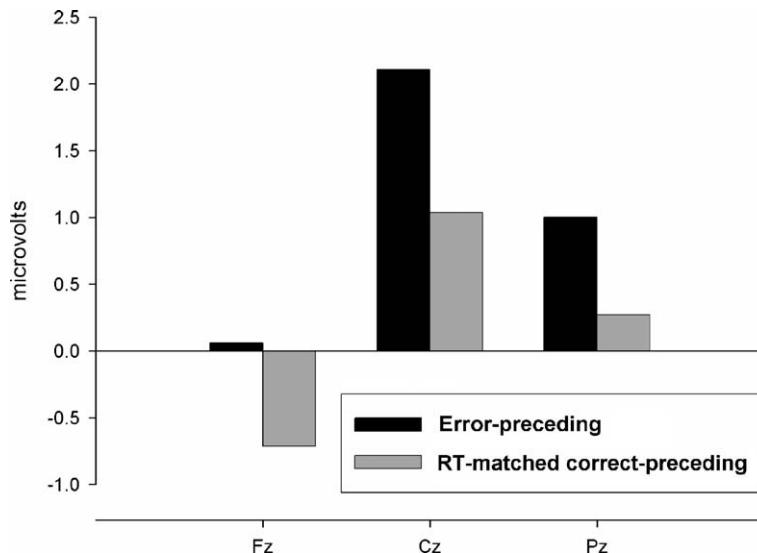


Fig. 2. Average amplitude at Fz, Cz, and Pz for error-preceding and RT-matched correct-preceding trials in the 0–100 ms post-response window in Study 1.

significant ($F(1, 13) = 1.81, p > .20$). The interaction of trial type and electrode site was also not significant ($F(2, 26) < 1$).

1.3. Discussion

A major aim of the present study was to replicate the existence of an EPP in the response-locked ERP on trials followed by errors (Ridderinkhof et al., 2003). Consistent with the initial report, error-preceding trials were characterized by enhanced positive activity in the 0–100 ms post-response window, relative to RT-matched correct-preceding trials. Like Ridderinkhof and colleagues, we observed an EPP at all midline recording sites. In further analyses, we ensured that the EPP was not the result of differences in stimulus-related ERP components. Comparison of stimulus-locked error-preceding trials and correct-preceding trials did not reveal any differences that could explain the EPP in the response-locked averages. Thus, these data provide support for the notion that the EPP reflects neural activity that is tightly locked to the response.

In addition to replicating the EPP, we examined the specificity of this modulation to error-preceding trials. First, we examined response-locked activity on trials that followed errors to rule out the possibility that error-preceding activity was actually related to error-surrounding activity. Error-following ERPs did not differ from correct-following trials, although it should be acknowledged that RT-matching was not successful for this analysis. In a second analysis, we examined error – 2 trials to determine whether the EPP was specific to trials directly prior to errors, or if similar activity was associated with more distal trials. Although there was some evidence of enhanced positive activity on error – 2 trials, this effect was not reliable. Thus, the EPP appears

uniquely associated with trials that immediately precede performance errors.

2. Study 2

The specific aim of Study 2 was to determine whether the EPP observed in the Eriksen flankers task used in Study 1 and by Ridderinkhof et al. (2003) would also characterize error-preceding trials in a different experimental context. Toward this aim, we examined error-preceding trials and compared these trials to RT-matched correct-preceding trials in 40 participants who performed a modified version of the Stroop task.

2.1. Methods

2.1.1. Participants

Data obtained from 40 control participants who had served in previous studies were used in Study 2 (Hajcak and Simons, 2002; Hajcak et al., 2003a). All participants received course credit for their participation in the original experiments.

2.1.2. Task and procedure

Participants performed a slightly modified version of the computerized Stroop task. On each trial, participants were shown one of three color words ("red", "green", and "blue") presented either in red or green font against a black background. Each combination of word and color occurred equally often. Each word occupied approximately 3° of visual angle. A fixation mark (+) was presented below the stimuli, prior to each word. The participants were instructed to press the left or right mouse button in response to the color of the words.

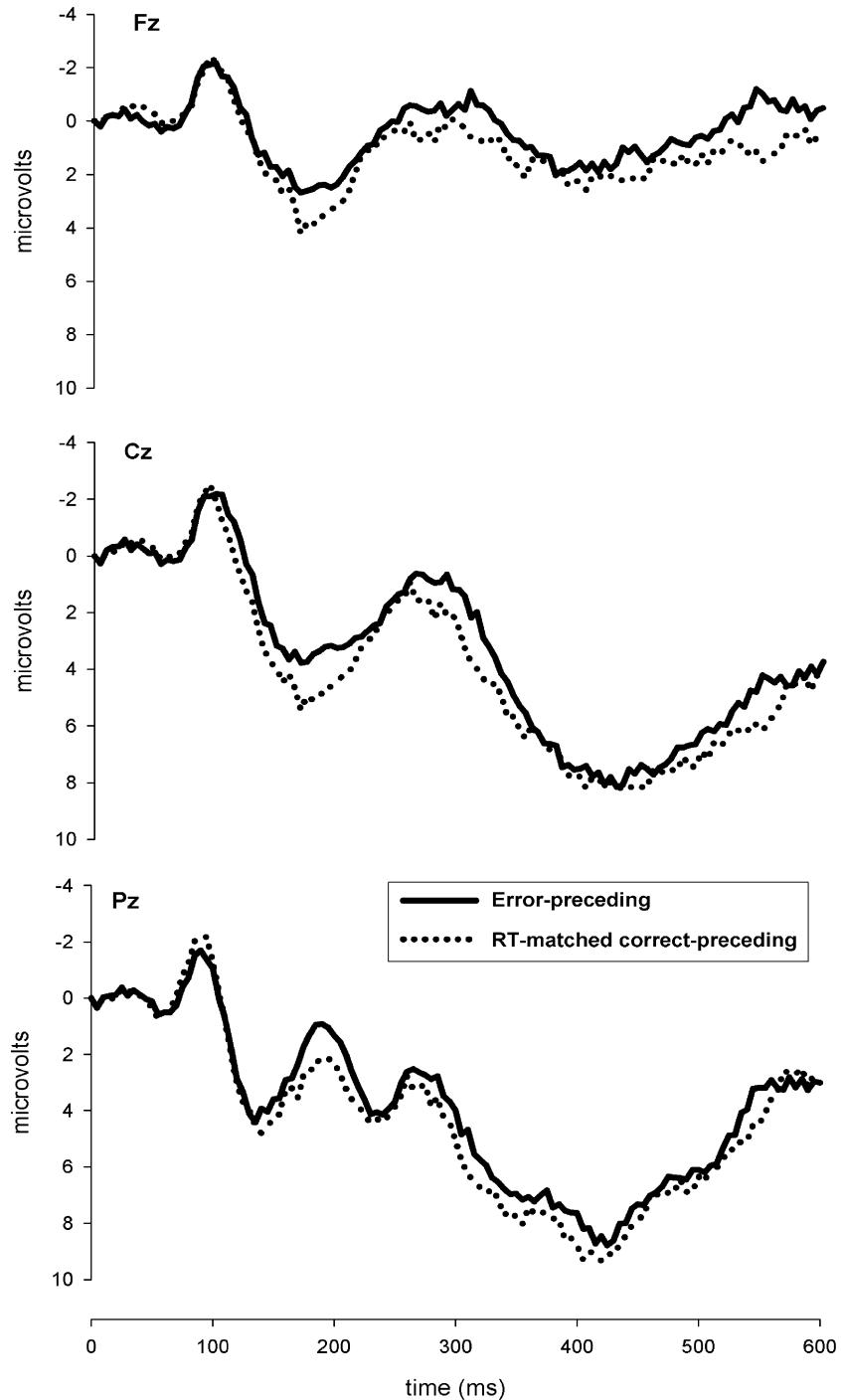


Fig. 3. Stimulus-locked ERPs at Fz, Cz, and Pz for error-preceding trials and RT-matched correct-preceding trials in Study 1.

Each participant was given two blocks of 24 practice trials. Half of the participants 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. For the other participants, the assignment of words to buttons was reversed. The participants were told to place equal emphasis on speed and accuracy of responding. Following the two practice blocks, the participants received 24 blocks of 48 trials (1152 total), with each block initiated

by the participant. Word stimuli were presented for 200 ms, separated by random interstimulus intervals between 2000 and 2400 ms.

2.1.3. Psychophysiological recording, data reduction and analysis

The recording and data analysis procedures have been described elsewhere (Hajcak and Simons, 2002; Hajcak

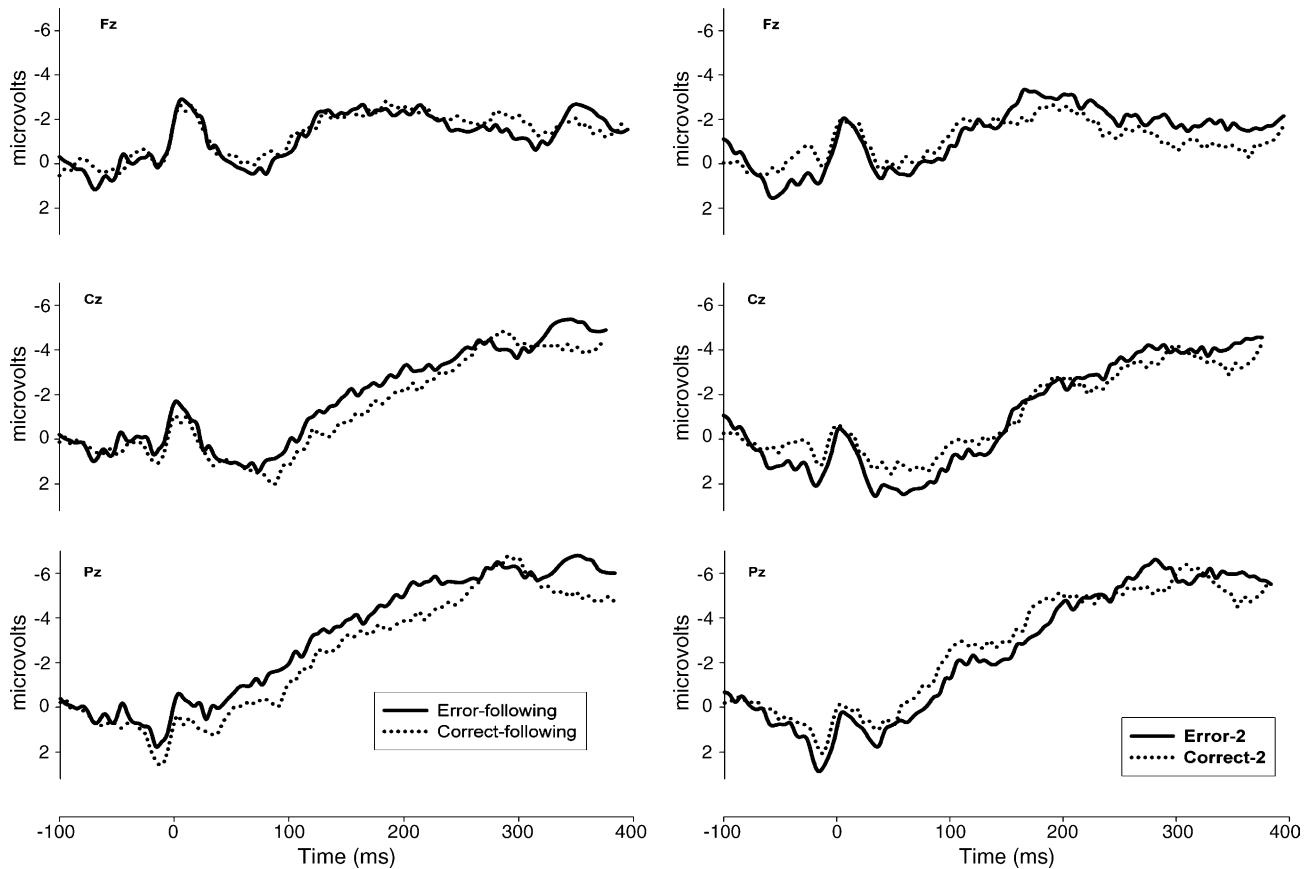


Fig. 4. Response-locked ERPs at Fz, Cz, and Pz for error + 1 trials and RT-matched correct + 1 trials (left panel), and error - 2 and RT-matched correct-2 trials (right panel) in Study 1.

et al., 2003a) and for all intents and purposes are the same as those described above for Study 1.

2.2. Results and discussion

2.2.1. Task performance

On average, participants made 81.7 errors (S.D. = 40.4), corresponding to 7.4% (S.D. = 3.7) of the trials. RTs on error trials ($M = 364$ ms, S.D. = 54 ms) were significantly faster than RTs on correct trials ($M = 406$ ms, S.D. = 52 ms; $t(39) = 10$, $p < .001$).

2.2.2. Error - 1 trials

The RT-matching procedure was successful: RTs for error-preceding trials ($M = 387$ ms, S.D. = 54 ms) did not differ from RTs for RT-matched correct-preceding trials ($M = 387$ ms, S.D. = 54 ms; $t(39) = .68$, $p > .50$).³ Response-locked ERPs for error-preceding and RT-matched correct-preceding trials are presented in Fig. 5.

³ Although RT-matched correct-preceding trials were faster than the overall mean RT on correct trials, this difference did not reach significance ($t(39) = 1.63$, $p > .10$). As in Study 1, the number of incompatible correct-preceding trials ($M = 24.2$, S.D. = 11.2) did not differ from the number of incompatible error-preceding trials ($M = 24.0$, S.D. = 12.5; $t(39) = .84$, $p > .50$).

Consistent with Study 1, error-preceding trials were characterized by enhanced positive activity in the post-response ERP. Although this effect was small (~1 microvolt), the main effect of trial type was reliable, ($F(1, 39) = 5.79$, $p < .05$). The interaction between trial type and electrode site was not significant ($F(2, 78) < 1$). Interestingly, Fig. 5 reveals that the error-preceding modulation of the ERP was more protracted than the EPP observed in Study 1. Statistical tests of successive 100-ms time windows confirmed that, in addition to the 0–100 ms window, the modulation remained significant in the 100–200 ms window ($F(1, 39) = 11.86$, $p < .001$) but did not in the 200–300 ms or 300–400 ms windows ($F(1, 39) = 2.01$, $p > .15$ and $F(1, 39) < 1$, respectively). The interaction between trial type and electrode site did not reach significance in any of these windows (all $p > .20$). Thus, although error-preceding trials in the Stroop task were associated with an EPP, the modulation appeared to be different to some extent, beginning somewhat later and continuing further in the response-locked epoch.

3. Study 3

The aim of Study 3 was to determine whether the modulation of error-preceding brain activity could still be

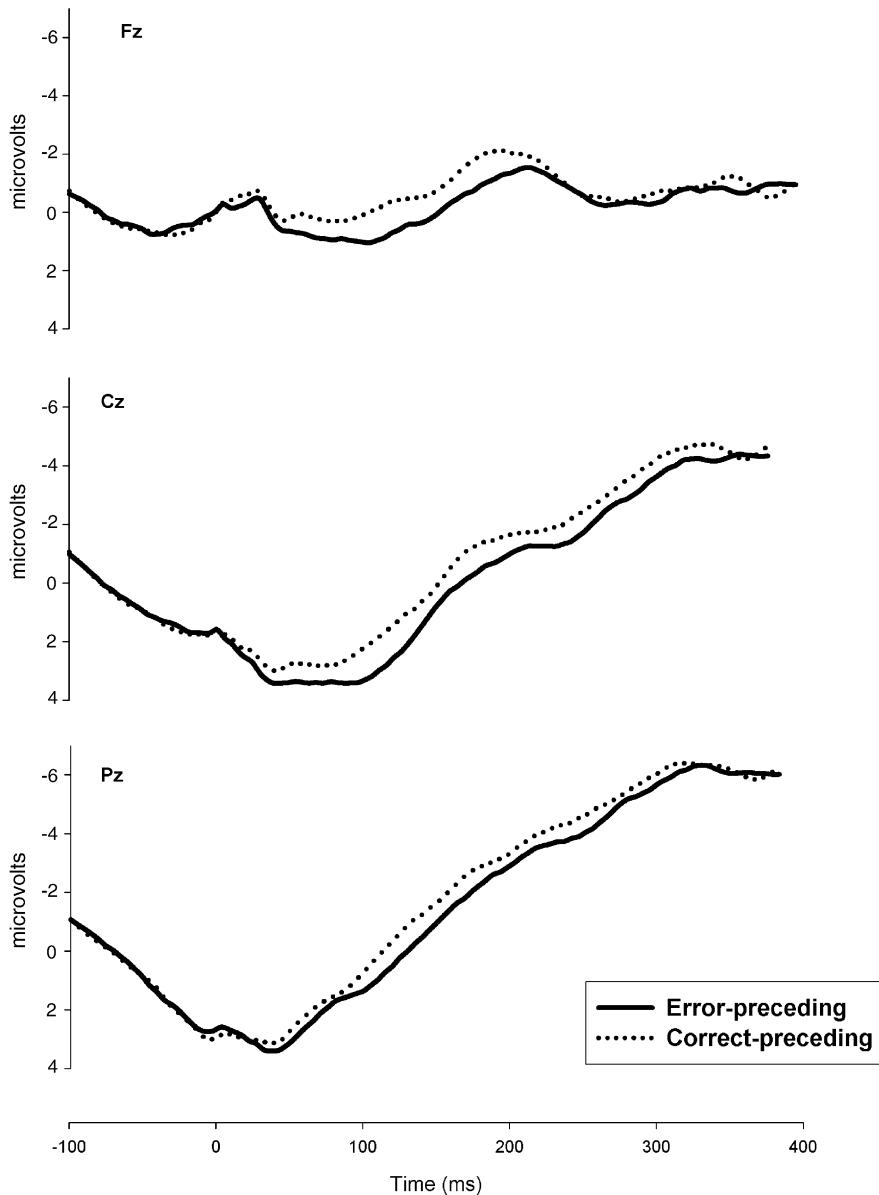


Fig. 5. Response-locked ERPs at Fz, Cz, and Pz for error-preceding trials and RT-matched correct-preceding trials in Study 2.

observed when between-trial intervals were lengthened. The possibility that EPPs might persist was raised in Study 1 by the observation of a small, but not statistically reliable modulation of the error – 2 waveforms. The question addressed in Study 3, therefore, was whether an EPP could be observed on error – 1 trials when these trials precede errors by relatively long time intervals. To address this question, we examined error-preceding trials and RT-matched correct-preceding trials in 21 participants who performed a modified Stroop task with an inter-trial interval of more than 5 s.

3.1. Methods

3.1.1. Participants

For Study 3 we reanalyzed data published previously (Hajcak et al., 2003b). Twenty-two undergraduate students

participated in the experiment in return for either monetary remuneration (\$15) or course credit. The data from two participants was discarded due to near-perfect task performance.

3.1.2. Task and procedure

On each trial, participants were shown one of three large arrows oriented either to the right, to the left or to the top of a 17" monitor screen. The arrows were positioned in the center of the screen and were presented either in red or green against a black background. A fixation mark (+) was presented just prior to the onset of each stimulus. The participants were instructed to press the left or right 'ctrl' key with the left and right hands, respectively, in response to the color of the arrows and to disregard their orientation. Thus, the orientation of the arrows could be congruent,

incongruent, or neutral with respect to the correct response hand.

After a brief description of the experiment, participants were given two blocks of 18 practice trials. Half of the participants were told to press the left ‘ctrl’ key when the arrow was red, and the right ‘ctrl’ key when the arrow was green. For the other participants the stimulus–response mapping was reversed. The participants were told to place equal emphasis on speed and accuracy. Following the two practice blocks, participants received 12 blocks of 48 trials (576 total), with each block initiated by the participant. Arrow stimuli were presented for 200 ms, separated by random intertrial intervals between 5300 and 5700 ms.

3.2. Results and discussion

3.2.1. Task performance

On average, participants made 33.1 errors (S.D. = 32.0), corresponding to 5.4% (S.D. = 5.2) of the trials. RTs on error trials ($M = 434$ ms, S.D. = 64 ms) were significantly faster than RTs on correct trials ($M = 465$ ms, S.D. = 58 ms; $t(19) = 3.78$, $p < .001$).

3.2.2. Error – 1 trials

The RT-matching procedure was successful: RTs for error-preceding trials ($M = 458$ ms, S.D. = 62 ms) did not differ from RTs for RT-matched correct-preceding trials ($M = 454$ ms, S.D. = 67 ms; $t(19) < 1$).⁴ Response-locked ERPs for error-preceding and RT-matched correct-preceding trials are presented in Fig. 6.

Consistent with Studies 1 and 2, error-preceding trials were characterized by enhanced positive activity in the post-response ERP. In this experiment, the modulation started later and as in Study 2, it was more sustained than it was in either Study 1 or in Ridderinkhof et al. (2003). As in Study 2, we tested whether the modulation was statistically significant in successive 100 ms time windows following the response. Although the error-preceding difference was not significant in the 0–100 ms post-response window ($F(1, 19) = 2.45$, $p > .10$), it was significant in the 100–200 ms window ($F(1, 19) = 10.12$, $p < .01$), the 200–300 ms window ($F(1, 19) = 6.51$, $p < .05$), and in the 300–400 ms window ($F(1, 19) = 4.41$, $p < .05$). In none of the windows did the interaction between trial type and electrode site reach significance (all $Fs < 1$). This is consistent with the notion that the error-preceding modulation was again equally large at all electrode sites. Thus, the error-preceding ERP modulations in Studies 2 and 3 were similar insofar as they were more sustained than the EPP in Study 1. Additionally, error-preceding positive activity

began somewhat later in Study 3. One possible explanation for this difference is that participants responded with the keyboard using two hands in Study 3, but with the mouse using two fingers in both Studies 1 and 2; thus, the fact that the modulation started later in Study 3 may be related either to the temporal precision of the response measure or to factors related to the response modality. These data suggest that although error-preceding positive activity is a robust phenomenon, there may be task-related differences in the timing and duration of this effect.

4. General discussion

We evaluated brain activity on error-preceding trials compared to RT-matched correct-preceding trials in three studies using different participants and tasks. The general aim was to replicate and extend the recent study of Ridderinkhof et al. (2003), who reported a modulation of the response-locked ERP on trials preceding errors. Specifically, Ridderinkhof et al. found enhanced positive activity on error-preceding trials that peaked approximately 50 ms post-response.

In Study 1, we replicated these results and found that error-preceding trials were uniquely characterized by enhanced positive activity in the 0–100 ms post-response window. Furthermore, this modulation was discrete; it closely followed the response and its duration was brief. In a series of subsequent analyses, we demonstrated that error-preceding trials did not differ in terms of stimulus-locked ERPs, and therefore the response-locked modulation on error-preceding trials cannot be explained in terms of stimulus-evoked activity. Furthermore, the results of Study 1 indicated that the EPP was specific to trials preceding errors. Trials immediately following errors were not associated with enhanced positive activity, excluding the possibility that the EPP is a phenomenon characteristic of all error-surrounding trials. Although enhanced positive activity was statistically reliable only in the error – 1 waveforms, a smaller post-response positivity was evident in the error – 2 waveforms. This raises the possibility that the ‘disengagement’ of the response-monitoring system is a gradual process that may begin even before error-preceding trials.

The aims of Studies 2 and 3 were to investigate whether the EPP would generalize to different subject samples, to tasks other than the Eriksen flankers task, and to longer intertrial intervals. In Study 2, we found an enhanced positivity on error-preceding trials utilizing a modified Stroop task. In Study 3, we found a similar modulation preceding errors in yet a different version of the Stroop task. Furthermore, Study 3 employed an interstimulus interval that exceeded five seconds—much longer than the interstimulus intervals used in previous EPP investigations. Thus, these data indicate that a positive enhancement of the ERP on error-preceding trials is a robust phenomenon that can be observed under various task conditions and that the state

⁴ As in Study 2, correct-preceding trials were not significantly faster than the overall mean RT on correct trials ($t(19) = .22$, $p > .50$). Similar to both Study 1 and 2, the number of incompatible correct-preceding trials ($M = 10.6$, S.D. = 9.0) was comparable to the number of incompatible error-preceding trials ($M = 10.7$, S.D. = 9.7; $t(19) = .19$, $p > .50$).

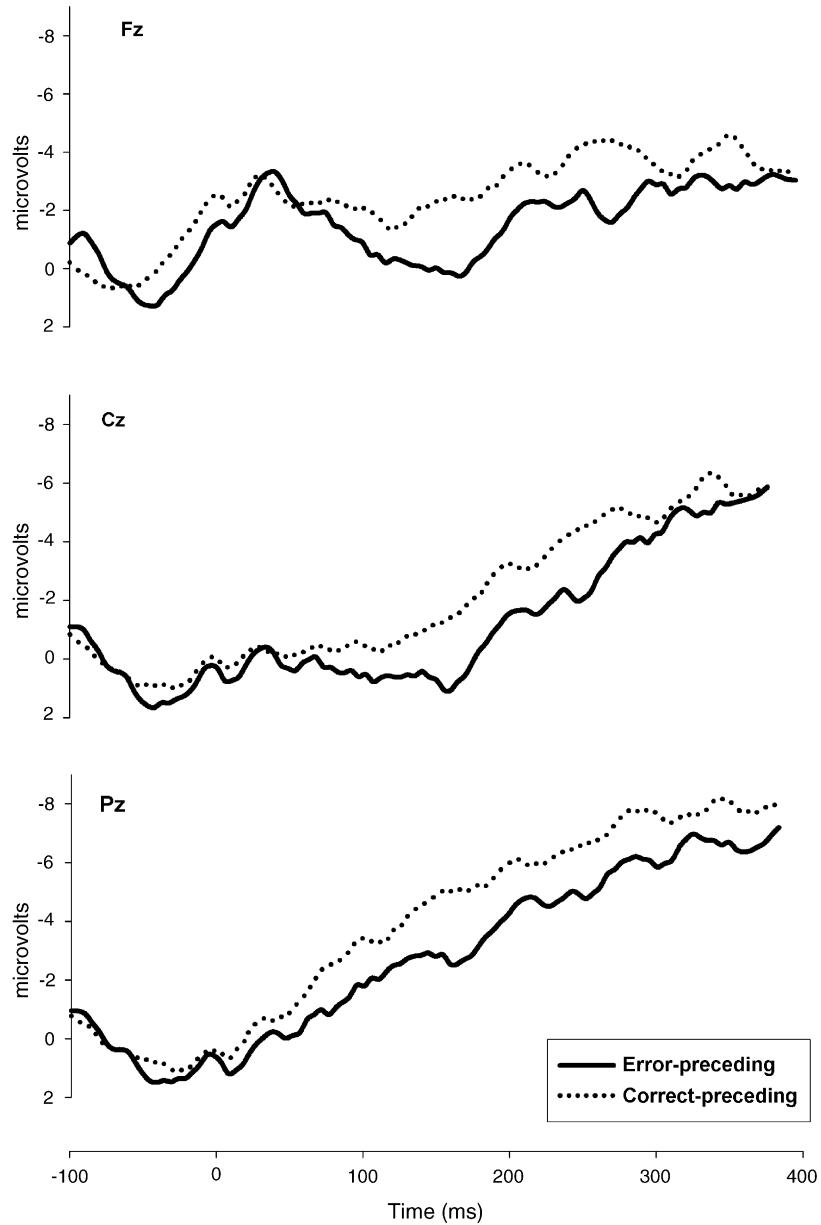


Fig. 6. Response-locked ERPs at Fz, Cz, and Pz for error-preceding trials and RT-matched correct-preceding trials in Study 3.

underlying the error-preceding modulation may be sustained over relatively long time intervals. It may be important to note, in addition, that the positive modulation observed in Studies 2 and 3 started somewhat later and continued for a longer duration than it did in Study 1 (cf. Allain et al., 2004b). It is possible that the modulations observed in Study 1 and in Studies 2 and 3 reflect the same cognitive process but that the manifestation of this process is influenced by contextual factors unique to each experimental setting.

Describing the ERP modulation that precedes errors as the “error-preceding positivity” conveys the notion that there may be a specific process that occurs before errors. While this may be the case, it is also plausible that ERP differences on error-preceding trials represent, to some degree, a specific modulation of the response-locked CRN—

the small negative deflection that closely follows the response on correct trials. It has been proposed that the CRN reflects activity of the same neural system for performance monitoring that underlies the ERN following errors (Vidal et al., 2000, 2003; Allain et al., 2004a,b). Indeed, fMRI studies suggest that the circumstances and events that give rise to CRNs and ERNs result in activation of a common brain region in the posterior medial frontal cortex (see Ridderinkhof et al., 2004b, for a review). Thus, the EPP may, at least in part, reflect a reduction of the CRN on trials that precede errors.

Relevant to this possibility, a recent article by Allain et al. (2004b) used spatial filtering (i.e., Laplacian transformation) and found that the ERP modulation on error-preceding trials was accounted for, at least in part, by a reduction in the CRN.

In fact, the notion that the EPP reflects CRN-modulation seems consistent with the original proposal that the EPP reflects the transient disengagement of the performance monitoring system and that this disengagement eventually causes an error to occur (Ridderinkhof et al., 2003). It also seems consistent with recent observations that fluctuations in the amplitude of the CRN are predictive of various indices of future performance (Ridderinkhof et al., 2004a).

However, the present data suggest that the CRN and EPP have different scalp topographies: Whereas the CRN has been reported to be largest over fronto-central midline electrodes (Vidal et al., 2000, 2003; Bates et al., 2002, 2004; Mathalon et al., 2003), the error-preceding modulations in each of the studies reported here were similar in magnitude at frontal, central, and parietal midline electrodes. Additionally, error-preceding activity was rather sustained in Study 2 and 3, as well as in the Allain et al. (2004b) paper—continuing well beyond the duration of the CRN. Thus, the ‘error-preceding positivity’ may actually reflect some combination of CRN-modulation and other, more sustained, positive activity.

In sum, the present study replicated and extended previous observations of a positive modulation of scalp-recorded electrophysiological activity on trials preceding performance errors (Ridderinkhof et al., 2003; Allain et al., 2004b). The finding that this modulation was most evident on error – 1 trials but appeared irrespective of the inter-trial interval duration suggests that this modulation reflects neural activity predictive of errors—consistent with the notion of an incidental disengagement of a performance monitoring system. Although the precise timing of this modulation differed between studies, it remains to be determined how meaningful these differences in timing are and what factors these differences reflect. In any case, the current results add to the growing literature on brain activity associated with performance monitoring. The present studies underscore the notion that the performance monitoring system can be studied both by measuring brain activity related to error and correct responses and by correlating fluctuations in this activity with subsequent performance (cf. Gehring et al., 1993; Botvinick et al., 2001; Kerns et al., 2004).

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