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On the ERN and the significance of errors

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Abstract

The error-related negativity (ERN) is an event-related brain potential observed when subjects commit errors. To examine whether the ERN is sensitive to the value of errors, the motivational significance of errors was manipulated in two experiments. In Experiment 1, low and high monetary value errors were compared to evaluate the effect of trial value on the ERN. In Experiment 2, subjects performed a flanker task both while their performance was being evaluated and during a control condition. Consistent with the notion that the error-detection system is sensitive to the significance of errors, the ERN was significantly larger on high-value trials in Experiment 1 and during evaluation in Experiment 2. There were no corresponding effects on the correct response negativity, and no behavioral differences between conditions were evident in either experiment. These results are discussed in terms of the functional role of the ERN in response monitoring.

Descriptors: Motivation, Event-related potentials (ERPs), Error-related negativity (ERN), Ne, Value, Affect

Effective action monitoring involves appropriate performance adjustments in terms of task demands, and a crucial component of this process is the ability to detect errors and adjust performance accordingly (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). Studies that measure response-locked event-related brain potentials (ERPs) have described fronto-centrally maximal negative components that appear relevant to response monitoring. Perhaps most notably, when subjects make a mistake, the response-locked ERP at fronto-central recording sites is characterized by a negative deflection known as the error-related negativity (ERN or Ne) that peaks approximately 50 ms post-response (Falkenstein et al., 2000; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Coles, Meyer, & Donchin, 1990; Gerhing, Goss, Coles, Meyer, & Donchin, 1993; Holroyd & Coles, 2002; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001).

Because the ERN has been observed across different stimulus and response modalities, it is thought to reflect the activity of a generic response monitoring system (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 utilizing source localization suggest that the ERN is generated in the medial frontal cortex, most likely the anterior cingulate cortex (ACC; Dehaene et al., 1994; Holroyd et al., 1998; Miltner et al., 1997).

In addition to the ERN, a small negative deflection has also been observed in the response-locked ERP on correct trials. This correct response negativity (CRN) appears to have morphological and topographical properties similar to the ERN (Vidal, Burle, Bonnet, Grapperon, & Hasbroucq, 2003; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). Although the functional significance of the CRN is unknown, the similarities between the ERN and CRN suggest that both components may index response monitoring processes. In support of this possibility, Ridderinkhof, Nieuwenhuis, Hajcak, van den Wildenberg, and Burle (2004) recently showed that CRN amplitude was related to performance measures that index response control. Specifically, trials that followed large-CRN trials were characterized by increased accuracy and reduced reaction time (RT) interference from incompatible stimuli. Taken together, the ERN and CRN are potentially similar medial frontal negativities that are related to evaluative functions during response monitoring.

Although many contemporary response monitoring theories discuss ERP components such as the ERN in terms of information processing signals (e.g., related to reinforcement learning or the detection of response conflict; Holroyd & Coles, 2002; Yeung, Botvinick, & Cohen, 2004) there is a growing body of literature describing potential affective and motivational influences on these components. For instance, a number of studies have reported enhanced ERNs in affectively distressed subjects such as patients with obsessive-compulsive disorder (OCD; Gehring, Himle, & Nisenson, 2000; Johannes, Wieringa, Nager, Rada,

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et al., 2001), subjects who score high in OC symptoms (Hajcak & Simons, 2002), worried subjects (Hajcak, McDonald, & Simons, 2003), and subjects who report high negative affective experience (Hajcak, McDonald, & Simons, 2004; Luu, Collins, & Tucker, 2000). Additionally, Johannes, Wieringa, Nager, Dengler, and Munte (2001) found that administration of an anxiolytic drug (Oxazepam) reduced the amplitude of the ERN, despite having no effect on performance measures.

Based on these data, several authors have now assigned an important role for affective and motivational factors in their conceptualization of the ERN (Gehring & Willoughby, 2002; Hajcak, McDonald, et al., 2004; Luu et al., 2000; Luu, Tucker, Derryberry, Reed, & Pulsen, 2003; Luu & Tucker, in press; Pailing & Segalowitz, 2004). Rather than indexing error or conflict detection as such, it has been suggested that the ERN may by sensitive to dynamically established goal states, and reflect the negative affective response to errors (Luu et al., 2000, 2003; Luu & Tucker, in press).

If the ERN reflects the affective evaluation of response outcomes and is sensitive to motivational factors, then a straightforward prediction is that the ERN should be larger when errors are more valuable or significant. In an early study, Gehring et al. (1993) found that the ERN was larger when subjects were rewarded for being accurate rather than fast. One interpretation of this finding is that instructions to be accurate made errors more salient and therefore negatively valent when they occurred. However, the conflict model of the ERN can explain these ERP differences based on behavioral data alone (e.g., differences in error rate and reaction time; Yeung, 2004; Yeung et al., 2004).

More recently, Pailing and Segalowitz (2004) attempted to directly manipulate the motivational value of response errors by selectively rewarding one type of correct response over another in a four-choice reaction-time task. Although they did not find that more costly types of errors were associated with larger ERNs in all participants, Pailing and Segalowitz did show that the motivational influence on the ERN was negatively related to conscientiousness and positively related to neuroticism. Pailing and Segalowitz argue that their results demonstrate the importance of taking both motivational and personality factors into account when analyzing an individual's response to an error.

Although there have been suggestions that the ERN reflects the motivational or affective evaluation of events based on between-groups studies on affective distress, more valuable errors have not consistently been related to enhanced ERN magnitude in within-subject designs. In an attempt to further examine the role of motivational factors on the ERN, the main goal of the present research was to evaluate the ERN while manipulating the motivational salience of errors in two experiments. In Experiment 1, we sought to make errors more or less significant by manipulating the monetary value of each trial. In Experiment 2, subjects performed a task while their performance was being evaluated, and again when they were simply encouraged to be fast and accurate. We hypothesized that more significant errors (those committed on more valuable trials and those committed during evaluation) would be associated with larger ERNs than less significant errors.

A second goal of the present study was to determine whether the effect of motivational manipulations would be confined to erroneous trials. Considering the potential functional, morphological, and topographical similarities of the ERN and CRN, more significant trials may modulate both of these components. Despite this possibility, the influence of motivation on the CRN has not yet been evaluated. If the general response monitoring activity of the ACC is sensitive to affective and motivational factors, then the manipulations in the present study might exert an influence on both the CRN and ERN. However, if the motivational manipulations selectively increase the ERN, these data would suggest that only error detection, as such, is sensitive to situational affective and motivational factors, and indicate some functional differentiation between the ERN and CRN.

EXPERIMENT 1

In this experiment, we employed a simple motivational manipulation: varying monetary value on a trial-by-trial basis. To this end, participants performed an arrowhead version of a flanker task in which each trial was preceded by either a "5" or "100" signal, indicating the number of points they could earn on the upcoming trial. Participants were instructed to earn as many points as possible and were told that points would be converted to money at the conclusion of the experiment. If the ERN is sensitive to the value of errors, then the response ERN should be larger on 100-point than on 5-point errors.

Methods

Participants

Twenty-two undergraduate students (21 women) in an upper level psychology class participated in the current experiment for extra credit. Participants were told that they could earn bonus money based on their performance, but, in fact, performance was unrelated to subject remuneration. All participants received $5 for their participation. Data from 2 participants were excluded because of near perfect task performance.

Task

An arrowhead version of the flanker task 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.

Throughout 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. In this way, there were two congruent conditions ("< < < < <" and "> > > > >") and two incongruent conditions ("< < > < <" and "> > < > >"). Arrowheads were presented in white font against a black background, and were positioned in the center of the screen. 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. The stimuli were presented randomly such that 50% of trials were congruent. Finally, each trial was preceded randomly by either a "5" or "100" cue indicating the point value of the upcoming trial. The cue remained on the screen for 2 s, and the imperative stimulus appeared between 200 and 800 ms following cue offset and remained on the screen for 200 ms. The interval between the offset of the imperative stimulus and the onset of the following cue varied between 1700 and 2300 ms.

Procedure

After a brief description of the experiment, EEG sensors were attached and the participants were given detailed task instructions. They were then given two blocks of 48 practice trials during which both speed and accuracy were emphasized. Although there was no explicit RT deadline during the actual experiment, participants were instructed that they could earn points during the actual experiment if they were both fast and accurate. Participants were told that each trial would be preceded by a "5" or "100" signal indicating the value of the upcoming trial. Participants were told to earn as many points as possible and that points would be converted to money at the conclusion of the experiment. Following this brief set of instructions, the experiment commenced and consisted of 12 blocks of 48 trials (576 total trials) with each block initiated by the participants at their own pace. The entire procedure lasted approximately 40 min.

Psychophysiological Recording, Data Reduction, and Analysis

The electroencephalogram (EEG) was recorded using a Neurosoft Quik-Cap with tin electrodes. Recordings were taken from three locations along the midline: frontal (Fz), central (Cz), and parietal (Pz). In addition, tin disk electrodes were placed on the left and right mastoids. During the recording, all activity was referenced to Cz. The electrooculogram (EOG) generated from blinks and vertical eye movements was recorded using Med-Associates miniature Ag-AgCl 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 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 presentation 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 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 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 response-locked CRN and ERN, averages were separately computed for correct and erroneous trials. The data were then baseline corrected by subtracting from each data point after response onset the average activity in the 100-ms window prior to the response. Both the ERN and CRN were then defined as the average voltage in the window from 0 to 100 ms post-response. To rule out possible stimulus-locked differences on the response-locked ERPs, the P300 was also evaluated. The P300 was defined in the averaged ERP of each participant as the most positive peak in a 300–600-ms window following stimulus onset, relative to the first data point as a baseline. Behavioral measures were analyzed via analysis of variance (ANOVA) and all ERP components were statistically evaluated using an ANOVA with the Greenhouse–Geisser correction applied to p values associated with multiple df repeated-measures comparisons.

Results

Behavioral Measures

Table 1 presents RT and accuracy data for 5- and 100-point trials. The number of excluded trials did not differ between 5- ($M = 10.94$, $SD = 10.34$) and 100-point ($M = 9.23$, $SD = 11.76$) trials, $F(1,19) < 1$. Because the number of rejected trials could vary between conditions, the number of errors and percentage correct are not redundant behavioral measures, and both are reported. Subjects did not differ in terms of the total number of 5- and 100-point errors, $F(1,19) = 2.79$, $p > .10$, nor did they differ in average error rate on 5- and 100-point trials, $F(1,19) = 2.11$, $p > .10$. All subjects made at least five errors on both 5- and 100-point trials. In terms of RT on valid trials, a 2 (Trial Type) × 2 (Trial Value) ANOVA indicated that subjects were faster on error trials, $F(1,19) = 5.91$, $p < .05$, than on correct trials, but not faster on 100-point trials than on 5-point trials, $F(1,19) < 1$. The interaction between Trial Type and Trial Value also did not reach significance, $F(1,19) < 1$.

Table 1 also presents the RTs for both congruent and incongruent correct trials. A 2 (Congruency) × 2 (Trial Value) ANOVA confirmed the impression that congruent trials were characterized by faster RTs than incongruent trials, $F(1,19) = 148.89$, $p < .001$; there was no main effect of Trial Value, $F(1,19) < 1$, and no interaction between Congruency and Trial Value, $F(1,19) < 1$. These data demonstrate the traditional flanker interference effect such that incongruent trials are associated with slower RTs than congruent trials; however, this flanker interference effect was comparable on both 5- and 100-point trials.

Response-Locked ERPs

Figure 1 presents the response-locked ERP averages for errors and correct trials at Fz (top), Cz (middle), and Pz (bottom) for 5- and 100-point trials. The ERN is evident as a sharp negative deflection on error trials, peaking roughly 50 ms after the response. A smaller negativity, the CRN, is apparent on trials with correct responses.

A 2 (Trial Type) × 2 (Trial Value) × 3 (Electrode Site) ANOVA confirmed that the ERN was larger than the CRN, $F(1,19) = 25.79$, $p < .001$. Additionally, there was a main effect for Electrode Site, confirming the impression from Figure 1 that both the ERN and CRN were fronto-centrally maximal, $F(2,38) = 11.76$, $p < .001$, $\varepsilon = .68$. Importantly, there was also a significant interaction between Trial Type and Trial Value, $F(1,19) = 7.59$, $p < .05$, suggesting that the motivational manipulation differentially influenced correct and erroneous trials. In post hoc analyses, we separately compared the CRN and ERN for 5- and 100-point trials. In terms of the CRN, a 2 (Trial Value) × 3 (Electrode Site) ANOVA confirmed the impression that the CRN did not differ between 5- and 100-point trials, $F(1,19) < 1$.

Table 1. *Mean RT and Accuracy Measures (and Standard Deviations) for Experiment 1*

	5-point trials	100-point trials
Number of errors	15.2 (9.0)	17.7 (12.7)
Accuracy (% correct)	93.0 (4.1)	93.7 (4.6)
Error RT (ms)	360.2 (36.0)	358.8 (29.1)
Correct RT (ms)	421.6 (27.3)	422.4 (27.7)
Congruent RT (ms)	391.2 (23.8)	390.8 (24.2)
Incongruent RT (ms)	464.5 (38.4)	462.8 (33.2)

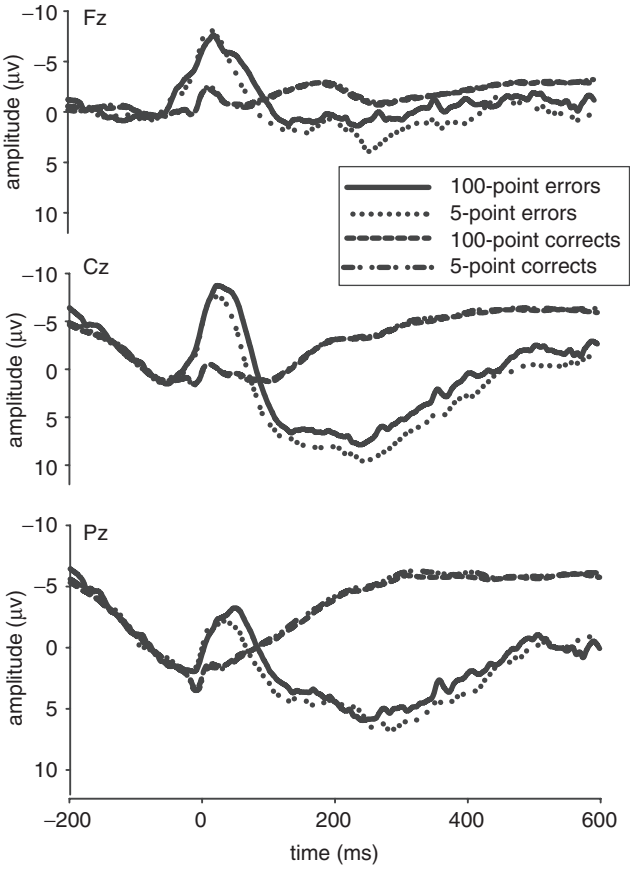

Figure 1. Response-locked ERPs at Fz (top), Cz (middle), and Pz (bottom) for 5- and 100-point correct and error trials.

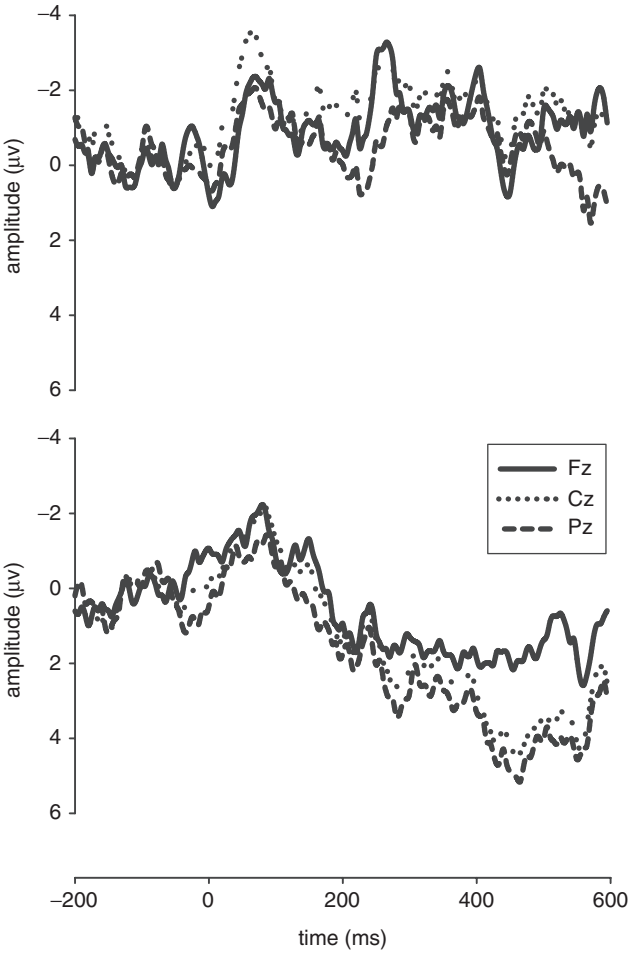

Figure 2. Difference waveforms at Fz, Cz, and Pz for 100-point minus 5-point errors (top) and evaluation-control errors (bottom).

However, a similar analysis of the ERN yielded a main effect for Trial Value, $F(1,19) = 7.53$, $p < .05$, such that 100-point errors were associated with a significantly greater ERN. The interaction of Trial Value and Electrode Site on error trials approached significance, $F(2,38) = 3.19$, $p < .10$, $\varepsilon = .75$, with the ERN difference between error values appearing largest at the central recording site. Seventeen of the 20 subjects had larger ERNs at Cz on 100-point than 5-point errors ($p < .005$ by sign test).

Because the enhancement of the negative activity on 100-point trials appeared to be prolonged, especially at the Cz recording site, we subtracted correct trial averages from error trial averages to yield a difference wave (Figure 2, top). The difference wave is characterized by a general increase in negative activity, but shows a clear negative peak around 75 ms post-response, maximal at the Cz recording site. Thus, the enhanced negativity on 100-point errors was largest around the time window of the ERN—suggesting that the motivational manipulation enhanced error-related activity in the response-locked ERP.[1]

Stimulus-Locked ERPs

To ensure that response-locked ERP differences were not due to stimulus-related differences or to a general increase in orienting or arousal, we evaluated the stimulus-locked P300 for both 5- and 100-point trials. Because the P300 for congruent flankers has been shown to peak earlier than P300 on incongruent flankers (Coles, Scheffers, & Fournier, 1995; Masaki, Takasawa, & Yamazaki, 2000; Scheffers & Coles, 2000), we evaluated congruent and incongruent trials separately; these data are presented in Figure 3.

A 2 (Trial Value) × 2 (Congruency) × 3 (Electrode Site) ANOVA yielded a main effect of Electrode Site, $F(2,38) = 97.03$, $p < .001$, $\varepsilon = .87$, which was consistent with the fact that the P300 was largest at central and parietal recording sites; overall P300s were not larger on congruent trials, $F(1,19) = 2.64$, $p > .10$, and there was no interaction between Congruency and Electrode Site, $F(2,38) = 2.38$, $p > .10$. Importantly, there was no effect of trial value on the P300, $F(1,19) < 1$, and the Value × Congruency, $F(1,19) < 1$, Value × Site, $F(2,38) = 1.03$, $p > .30$, and the Value × Congruency × Site, $F(2,38) < 1$, interactions were not significant.

Discussion

By manipulating the value of errors on a trial-by-trial basis, the present study found that 100-point errors were characterized by a larger ERN than 5-point errors. An interesting feature of the present results was that the effect of value on the ERN was observed despite this manipulation having no systematic effect

[1]In an analysis not reported in detail here, we performed a principal components analysis (PCA) to further examine this issue. The PCA yielded a factor showing a sharp negative peak just after the response that corresponded to the ERN and was significantly greater at Cz for 100-point errors than 5-point errors.

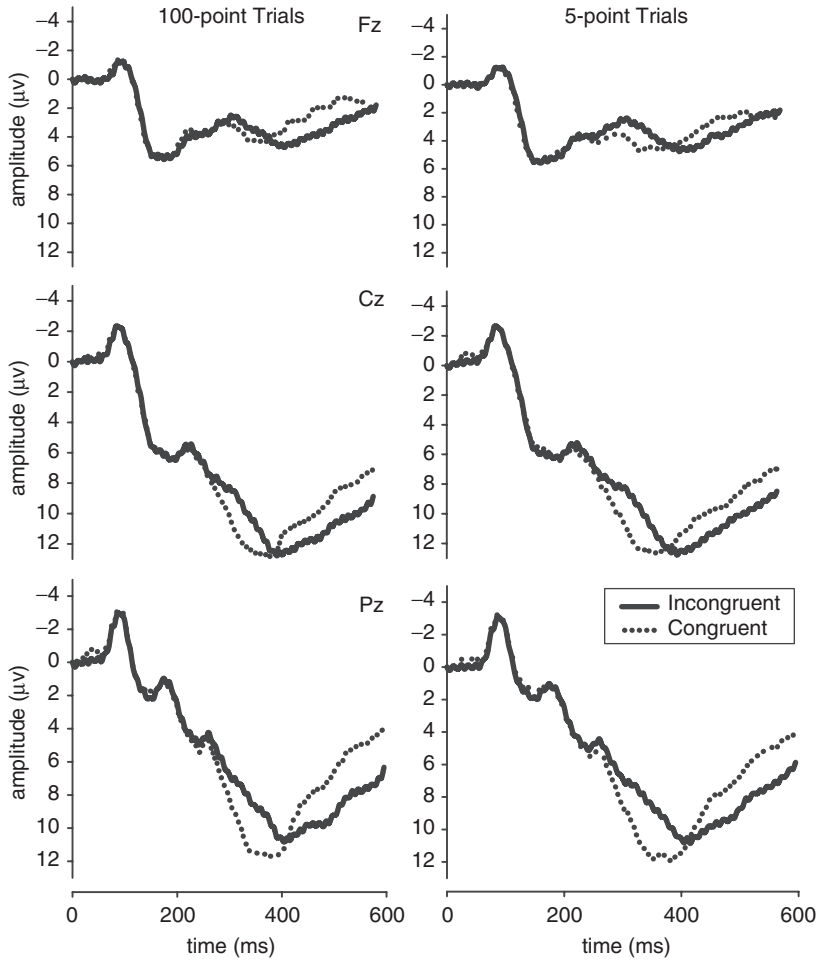

Figure 3. Stimulus-locked ERPs at Fz (top), Cz (middle), and Pz (bottom) for congruent and incongruent correct 100-point (left) and 5-point (right) trials.

on subjects' performance. That is, subjects were equally accurate on both 5- and 100-point trials, and RTs did not differ between 100- and 5-point trials on either error or correct trials.

Because 5- and 100-point trials did not differ with respect to any behavioral measures, it is unlikely that the enhanced ERN on more valuable trials could be driven by performance-related differences, such as those related to response conflict or error-detection as described by Yeung (2004), or by differential stimulus-component overlap as described by Hajcak, Vidal, and Simons (2004). The absence of behavioral differences strongly suggests that the larger ERN on 100-point trials is specifically related to the higher value of these errors.

In addition, the enhanced ERN associated with 100-point trials appeared to be specific to response-locked data on erroneous trials. First, trial value had no corresponding effect on the CRN. Insofar as trial value selectively influenced the ERN, these data suggest some functional difference between the ERN and CRN. Second, the P300 was similar on both 5- and 100-point trials. This suggests that the effect of trial value on the ERN was not related to general increases in arousal or to stimulus-related artifact.

The present data indicate that if subjects are given an appropriate cue, the magnitude of the ERN is enhanced for more valuable errors. Whereas Pailing and Segalowitz (2004) found that motivational factors only influenced ERN magnitudes in some subjects, the present data found a reliable increase in the ERN for more valuable errors. One possible explanation for this discrepancy is that the present study used a simpler manipulation of trial value. Overall, the finding that the magnitude of the ERN was sensitive to the value of errors is consistent with the view that the ERN reflects the motivational significance of errors.

EXPERIMENT 2

In an effort to examine the robustness of motivational influences on the ERN, we conducted a second experiment that evaluated the effects of error significance, by using a very different manipulation. In this experiment, participants again performed the arrowhead version of the flanker task; in this case, however, there were no cues indicating points that could be earned. Rather, participants performed the flanker task in both an evaluation and control condition. During the evaluation condition, the participants were told that their performance was being evaluated online by a research assistant, and that the research assistant would compare the participant's performance to other students who had performed the task. Participants were also told that they would receive feedback about how they compared to others who

had performed the task (for similar task instructions to ensure engagement in a response monitoring task, see Luu et al., 2000). During the control condition, participants were told that no evaluation would take place and were simply encouraged to be both fast and accurate.

We hypothesized that errors committed during the evaluation condition would be more significant to participants, and therefore, predicted that errors in the evaluation condition would be associated with a larger ERN relative to the control condition. Because more valuable correct trials in Experiment 1 were not characterized by an enhanced CRN, we expected this effect to be specific to the ERN and that the magnitude of the CRN would not differ between the two experimental conditions.

Method

Participants

Nineteen undergraduate students (12 men) were recruited through the University of Delaware Psychology Department subject pool to participate in the current study. All participants received course credit for their participation. Data from 1 participant were not included due to a technical malfunction. Thus, 18 participants comprised the final study sample and data from these participants were included in all subsequent analyses.

Task

The arrowhead version of a flanker task described in Experiment 1 was also utilized for this experiment. Because there were no "5" or "100" cues in this experiment, the interval between offset of the imperative stimulus and the following imperative stimulus varied between 1700 and 2300 ms.

Procedures

All procedures for this experiment were identical to those described in Experiment 1, except that participants received 12 blocks of 48 trials (576 trials) in each of the two experimental conditions (1152 total trials). Each condition lasted approximately 20 min and a 5-min break was taken between conditions; the order of conditions was counterbalanced between participants such that half the participants (9) performed the task in the control condition first.

In the evaluation condition, participants performed the flanker task seated approximately 0.5 m from a male research assistant. The evaluator was seated at a desk with a laptop computer that was connected to the participant's computer. Participants were told that the evaluator would receive information about the speed and accuracy of their performance throughout the task, and would compare their performance to that of other University of Delaware students who had completed the task before. Participants were also told that they would receive feedback about how they compared to others at the completion of the study. However, no actual evaluation was performed; at the conclusion of the experiment, all participants were told that they performed above average.

During the control condition, the evaluator was not present in the room and the participant was simply encouraged to be both fast and accurate. If the control condition was second, the participant was told explicitly that no formal performance evaluation would take place.

Psychophysiological Recording, Data Reduction, and Analysis

All psychophysiological recording, data reduction, and data analysis procedures for this study were identical to those utilized in Experiment 1.

Results

Behavioral Measures

Table 2 presents RT and accuracy data for trials in the evaluation and control conditions. The number of valid trials did not differ between experimental conditions, $F(1,17) < 1$. Subjects did not differ in terms of either the total number of errors, $F(1,17) = 2.94$, $p > .10$, or average error rate between conditions, $F(1,17) = 3.37$, $p > .08$. In terms of RT on valid trials, a 2 (Trial Type) $\times$ 2 (Condition) ANOVA indicated that subjects were faster on error trials, $F(1,17) = 164.22$, $p < .001$, than on correct trials, but did not differ between evaluation and control conditions, $F(1,17) < 1$; the interaction between Trial Type and Condition also did not reach significance, $F(1,17) < 1$.

Table 2 also presents the RTs for both congruent and incongruent correct trials. A 2 (Congruency) $\times$ 2 (Condition) ANOVA confirmed the impression that congruent trials were characterized by faster RTs than incongruent trials, $F(1,17) = 213.06$, $p < .001$. There was no main effect of Trial Value, $F(1,19) < 1$, and no interaction between Congruency and Trial Value, $F(1,19) < 1$. Thus, the flanker interference effect was comparable in both the evaluation and control conditions.

Response-Locked ERPs

Figure 4 presents the response-locked ERP averages for errors and correct trials at Fz (top), Cz (middle), and Pz (bottom) in both the evaluation and control conditions. The ERN is evident as a sharp negative deflection on error trials, peaking roughly 50 ms after the response; the smaller CRN is also apparent as a negative deflection on correct trials.

A 2 (Trial Type) $\times$ 2 (Condition) $\times$ 3 (Electrode Site) ANOVA confirmed that the ERN was significantly larger than the CRN, $F(1,17) = 41.43$, $p < .001$. Additionally, there was a main effect for Electrode Site, consistent with the impression from Figure 4 that both the ERN and CRN were fronto-centrally maximal, $F(2,34) = 36.02$, $p < .001$, $\varepsilon = .68$. Importantly, there was also a significant interaction between Trial Type and Condition, $F(1,17) = 8.47$, $p < .01$, suggesting that the correct and erroneous trials were differentially affected in the control and evaluation conditions. In post hoc analyses, CRNs and ERNs were separately compared.

A 2 (Condition) $\times$ 3 (Electrode Site) ANOVA confirmed that the ERN was significantly larger during the evaluation condition than in the control condition, $F(1,17) = 6.44$, $p < .05$. Consistent with the fact that the ERN was most negative at fronto-central

Table 2. *Mean RT and Accuracy Measures (and Standard Deviations) for Experiment 2*

	Control	Evaluation
Number of errors	33.2 (18.8)	29.9 (18.7)
Accuracy (% correct)	94.1 (3.4)	94.7 (3.3)
Error RT (ms)	340.6 (41.2)	338.7 (30.8)
Correct RT (ms)	407.9 (31.1)	407.0 (36.1)
Congruent RT (ms)	381.4 (32.2)	380.2 (32.5)
Incongruent RT (ms)	437.3 (32.5)	436.1 (42.0)

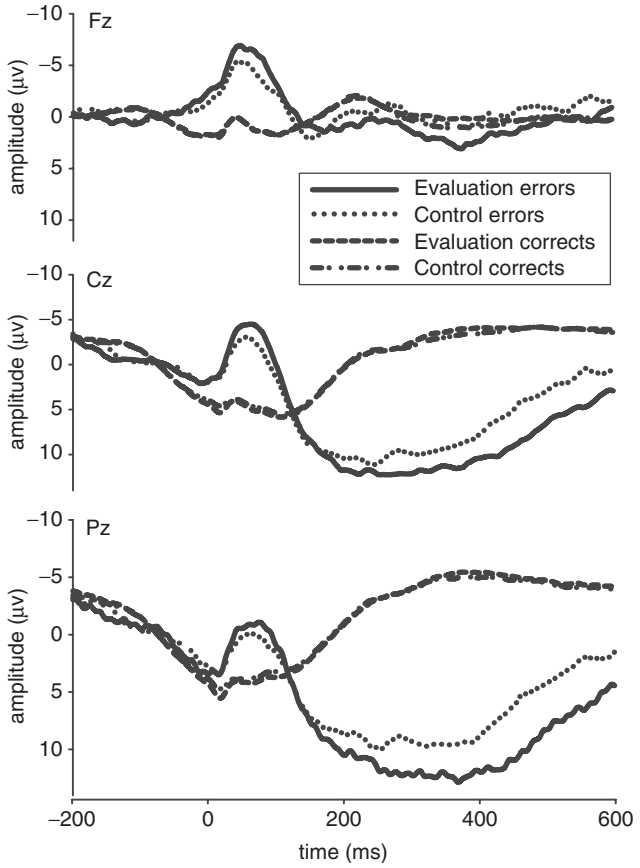

Figure 4. Response-locked ERPs at Fz (top), Cz (middle), and Pz (bottom) for errors and correct trials during the evaluation and control conditions.

recording sites, there was a main effect of Electrode Site, $F(2,34) = 34.40$, $p < .001$, $\varepsilon = .75$. There was no interaction between Electrode Site and Condition, $F(2,34) < 1$.

In contrast with the ERN, A 2 (Condition) × 3 (Electrode Site) ANOVA confirmed the impression that the CRN did not differ between experimental conditions, $F(1,17) < 1$. There was a main effect for Electrode Site, $F(2,34) = 20.27$, $p < .001$, $\varepsilon = .56$, consistent with the notion that the CRN was frontally maximal. There was no interaction between Electrode Site and Condition, $F(2,34) < 1$.

Difference waves were computed by subtracting ERP averages obtained on error trials in the control condition from ERP averages obtained on error trials in the evaluation condition, and are presented in Figure 2 (bottom). As in Experiment 1, these difference waves demonstrate a negative peak around 75 ms after response onset—consistent with an enhancement of the ERN. Unlike the data in Experiment 1, the negative enhancement on more significant errors did not continue beyond the window of the ERN.

Stimulus-Locked ERPs

The stimulus-locked ERP averages derived from congruent and incongruent correct trials in both the evaluation and control conditions are presented in Figure 5.

A 2 (Condition) × 2 (Congruency) × 3 (Electrode Site) ANOVA confirmed the impression that P300s were largest at central and parietal recording sites, $F(2,34) = 51.62$, $p < .001$, $\varepsilon = 1.0$; the magnitude of the P300 did not differ overall between congruent and incongruent trials, $F(1,17) = 3.72$, $p > .05$. However, there was a significant interaction between Congruency and Electrode Site, $F(2,34) = 3.97$ $p < .05$, $\varepsilon = .96$, indicating that incongruent trials were characterized by larger P300s at the central and parietal recording sites. These differences were confirmed by post hoc analyses, $F(1,17) = 6.74$, $p < .05$, and $F(1,17) = 7.02$, $p < .05$, at the central and parietal recording sites, respectively. Although congruency appeared to influence the magnitude of the P300, the effect of Condition on the P300 was not significant, $F(1,17) = 3.65$, $p > .05$, nor were the interactions between Condition and Congruency, $F(1,17) = 1.47$, $p > .20$, Condition and Electrode Site, $F(2,34) < 1$, and the three-way interaction of Condition, Congruency, and Electrode Site, $F(2,34) = 1.10$, $p > .30$. Although the magnitude of the P300 was influenced by the congruency of the trials at central and parietal recording sites, the motivation manipulation did not affect the P300.

Discussion

The results of Experiment 2 provide further support for the notion that the magnitude of the ERN is sensitive to error significance. Specifically, when subjects made mistakes in the evaluation condition, ERNs were reliably larger than ERNs in the control condition. Like Experiment 1, this effect was not found on correct trials, and was not accompanied by behavioral differences between conditions. Furthermore, analyses of the P300 suggested that the ERN difference was not related to differences in arousal or orienting in the evaluation condition.

Curiously, incongruent trials were characterized by an enhanced P300 at the central and parietal recording sites in Experiment 2, although this effect was not observed in Experiment 1. In fact, although differences did not reach statistical significance, the P300 appeared larger at Pz on congruent trials in Experiment 1. The difference in congruency effects on the P300 might be explained by methodological differences between the studies, although the precise mechanism underlying this difference is unclear. It is important to note that Experiments 1 and 2 also differed with respect to the sustained activity following the peak of the ERN. In Experiment 1, the enhanced negative activity on more valuable trials was sustained following the ERN. In Experiment 2, however, the enhanced ERN in the evaluation condition was followed by greater positive activity. These differences may reflect variation in the error positivity (Pe), a centroparietally maximal event-related brain potential that follows the ERN and has been related to processes that follow error detection, such as error awareness or error salience (cf. Falkenstein et al., 2000; Nieuwenhuis et al., 2001). In light of these results, future studies might be designed to further examine the role of motivational factors on this later error-related brain potential.

General Discussion

In the present study, the role of error significance was evaluated in two experiments. In Experiment 1, the value of errors was manipulated on a trial-by-trial basis. In Experiment 2, errors were made more relevant in a performance evaluation condition. In both experiments, more significant errors were associated with an enhanced ERN. These effects on ERN amplitude were observed in the absence of performance differences between conditions. That is, subjects were equally as fast and accurate

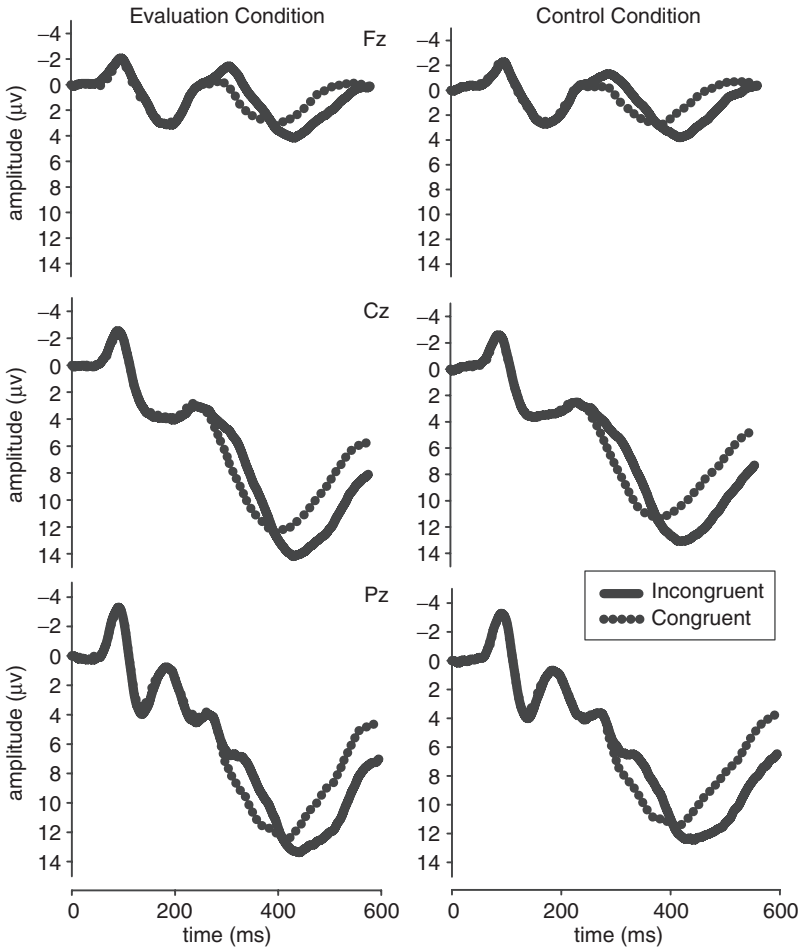

Figure 5. Stimulus-locked ERPs at Fz (top), Cz (middle), and Pz (bottom) for congruent and incongruent correct trials during the evaluation (left) and control (right) conditions.

on 5- and 100-point trials in Experiment 1, and demonstrated comparable performance in both evaluation and control conditions in Experiment 2. Thus, the observed effects of motivational significance on the ERN cannot be explained in terms of confounding effects of between-condition performance differences (cf. Yeung, 2004). Moreover, we conclude that the observed modulations of the ERN amplitude are not attributable to differential overlap with stimulus-locked components (cf. Hajcak, Vidal, et al., 2004) or differences in orienting/arousal that would be reflected in the P300. Instead, our findings provide clear evidence of the sensitivity of the ERN to the motivational significance of errors.

These findings suggest the need to extend contemporary theories of the ERN that have been formalized in computational models of specific cognitive functions, such as reinforcement learning (Holroyd & Coles, 2002) and response conflict monitoring (Yeung et al., 2004). To date, these theories have not placed emphasis on the kind of motivational factors studied here. Instead, the present results add to the growing body of evidence indicating the importance of affective and motivational factors on the evaluative functions reflected in the ERN.

For instance, a number of studies have reported enhanced ERNs in affectively distressed subjects (Gehring et al., 2000; Hajcak & Simons, 2002; Hajcak et al., 2003; Hajcak, McDonald, et al., 2004, Johannes, Wieringa, Nager, Rada, et al., 2001; Luu, Collins et al., 2000). Based on these data, Luu and Tucker (in press) suggest that the ERN indexes the affective distress associated with making an error. Insofar as more significant errors in Experiment 1 and 2 could be associated with greater affective distress, the present data appear consistent with this suggestion. In fact, affective distress has also been related to increased concern about mistakes, suggesting that errors are more significant for subjects high in affective distress (Cox, Enns, & Clara, 2002; Frost, Heimberg, Holt, Mattia, & Neubauer, 1993). Thus, one possibility is that the kind of motivational influences reported here may explain the relationship between affective distress and the ERN.

The present results also suggest potentially important dissociations between the response-locked ERN and other medial frontal negativities that have been implicated in evaluative functions. In particular, we found no effects of our motivational manipulations on the amplitude of the CRN, another response-locked component thought to be related to action monitoring. This finding suggests that the effect of motivation is specific to response-locked error-related brain activity. Insofar as the ERN is an index of ACC engagement, these data suggest that the ACC may become more engaged when processing more significant errors. Although the CRN may reflect similar ACC activity (Ridderinkhof et al., 2004; Vidal et al., 2000, 2003), the present data suggest that the ACC is not more engaged

in response monitoring during more significant correct trials. In this way, the present data suggest some functional differentiation between the CRN and ERN in terms of response monitoring.

In addition to the response-locked ERN, a number of studies have described the existence of a medial frontal negative deflection following negative performance feedback (Gehring & Willoughby, 2002; Hajack, Holroyd, Moser, & Simons, in press; Holroyd & Coles, 2002; Luu et al., 2003; Miltner et al., 1997). In fact, Holroyd and Coles suggest that both the response- and feedback-locked ERN reflect the activation of the same reinforcement learning system. Interestingly, recent studies have found that the feedback ERN is relatively insensitive to the value of negative feedback (Luu et al., 2003; Yeung & Sanfey, 2004). To the extent that the response-locked ERN does appear to be sensitive to error value, the present data suggest some functional dissimilarity between the response-locked and feedback-locked ERN (cf. Gehring & Willoughby, 2004).

Overall, the present study provides support for the notion that the ERN conveys information beyond simple error detection. The present study provides experimental evidence that the ERN is larger when errors are more significant. This effect was unrelated to performance differences, and was not accompanied by differences in the CRN or P300. These data suggest that the magnitude of the ERN reflects the motivational significance of errors.

REFERENCES

Bernstein, P. S., Scheffers, M. K., & Coles, M. G. H. (1995). "Where did I go wrong?" A psychophysiological analysis of error detection. *Journal of Experimental Psychology: Human Perception & Performance*, *21*, 1312–1322.

Coles, M. G. H., Scheffers, M. K., & Fournier, L. (1995). Where did I go wrong? Errors, partial errors, and the nature of human information-processing. *Acta Psychologica*, *90*, 129–144.

Cook, E. W., III (1999). *VPM reference manual*. Birmingham, AL: Author.

Cox, B. J., Enns, M. W., & Clara, I. P. (2002). The multidimensional structure of perfectionism in clinically distressed and college student samples. *Psychological Assessment*, *14*, 365–373.

Dehaene, S., Posner, M. I., & Tucker, D. M. (1994). Localization of a neural system for error detection and compensation. *Psychological Science*, *5*, 303–305.

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of cross-modal divided attention on late ERP components: II. Error processing in choice reaction tasks. *Electroencephalography and Clinical Neurophysiology*, *78*, 447–455.

Falkenstein, M., Hoormann, J., Christ, S., & Hohnsbein, J. (2000). ERP components on reaction errors and their functional significance: A tutorial. *Biological Psychology*, *51*, 87–107.

Frost, R. O., Heimberg, R. G., Holt, C. S., Mattia, J. I., & Neubauer, A. L. (1993). A comparison of two measures of perfectionism. *Personality and Individual Differences*, *14*, 119–126.

Gehring, W. J., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1990). The error-related negativity: An event-related brain potential accompanying errors. *Psychophysiology*, *27*, S34.

Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science*, *4*, 385–390.

Gehring, W. J., Himle, J., & Nisenson, L. G. (2000). Action-monitoring dysfunction in obsessive-compulsive disorder. *Psychological Science*, *11*, 1–6.

Gehring, W. J., & Willoughby, A. R. (2002). The medial frontal cortex and the rapid processing of monetary gains and losses. *Science*, *295*, 2279–2282.

Gehring, W. J., & Willoughby, A. R. (2004). Are all medial frontal negativities created equal? Toward a richer empirical basis for theories of action monitoring. In M. Ullsperger & M. Falkenstein (Eds.), *Errors, conflicts, and the brain: Current opinions on response monitoring* (pp. 14–20). Leipzig: MPI of Cognitive Neuroscience.

Gratton, G., Coles, M. G. H., & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalography and Clinical Neurophysiology*, *55*, 468–484.

Hajcak, G., McDonald, N., & Simons, R. F. (2003). Anxiety and error-related brain activity. *Biological Psychology*, *64*, 77–90.

Hajcak, G., McDonald, N., & Simons, R. F. (2004). Error-related psychophysiology and negative affect. *Brain & Cognition*, *56*, 189–197.

Hajcak, G., Holroyd, C. B., Moser, J. S., & Simons, R. F. (in press). Brain potentials associated with expected and unexpected good and bad outcomes. *Psychophysiology*.

Hajcak, G., & Simons, R. F. (2002). Error-related brain activity in obsessive-compulsive undergraduates. *Psychiatry Research*, *110*, 63–72.

Hajcak, G., Vidal, F., & Simons, R. F. (2004). Difficulties with easy tasks: ERN/Ne and stimulus component overlap. In M. Ullsperger & M. Falkenstein (Eds.), *Errors, conflicts, and the brain: Current opinions on response monitoring* (pp. 204–211). Leipzig: MPI of Cognitive Neuroscience.

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, *109*, 679–709.

Holroyd, C. B., Dien, J., & Coles, M. G. H. (1998). Error-related scalp potentials elicited by hand and foot movements: Evidence for an output-independent error-processing system in humans. *Neuroscience Letters*, *242*, 65–68.

Johannes, S., Wieringa, B. M., Nager, W., Dengler, R., & Munte, T. F. (2001). Oxazepam alters action monitoring. *Psychopharmacology*, *155*, 100–106.

Johannes, S., Wieringa, B. M., Nager, W., Rada, D., Dengler, R., & Emrich, H. M., et al. (2001). Discrepant target detection and action monitoring in obsessive-compulsive disorder. *Psychiatry Research: Neuroimaging*, *108*, 101–110.

Luu, P., Collins, P., & Tucker, D. M. (2000). Mood, personality, and self-monitoring: Negative affect and emotionality in relation to frontal lobe mechanisms of error monitoring. *Journal of Experimental Psychology: General*, *129*, 43–60.

Luu, P., Flaisch, T., & Tucker, D. M. (2000). Medial frontal cortex in action monitoring. *Journal of Neuroscience*, *20*, 464–469.

Luu, P., & Tucker, D. M. (in press). Self-regulation by the medial frontal cortex: Limbic representation of motive set-points. In M. Beauregard (Ed.), *Consciousness, emotional self-regulation and the brain*. Amsterdam: John Benjamin.

Luu, P., Tucker, D. M., Derryberry, D., Reed, M., & Poulsen, C. (2003). Electrophysiological responses to errors and feedback in the process of action regulation. *Psychological Science*, *14*, 47–53.

Masaki, H., Takasawa, N., & Yamazaki, K. (2000). An electrophysiological study of the locus of the interference effect in a stimulus-response compatibility paradigm. *Psychophysiology*, *37*, 464–472.

Miller, G. A., Gratton, G., & Yee, C. M. (1988). Generalized implementation of an eye movement correction procedure. *Psychophysiology*, *25*, 241–243.

Miltner, W. H. R., Braun, C. H., & Coles, M. G. H. (1997). Event-related brain potentials following incorrect feedback in a time-estimation task: Evidence for a "generic" neural system for error detection. *Journal of Cognitive Neuroscience*, *9*, 788–798.

Nieuwenhuis, S., Ridderinkhof, K. R., Blom, J., Band, G. P. H., & Kok, A. (2001). Error-related brain potentials are differentially related to awareness of response errors: Evidence from an antisaccade task. *Psychophysiology*, *38*, 752–760.

Pailing, P. E., & Segalowitz, S. J. (2004). The error-related negativity as a state and trait measure: Motivation, personality, and ERPs in response to errors. *Psychophysiology*, *40*, 84–95.

Ridderinkhof, K. R., Nieuwenhuis, S., Hajcak, G., van den Wildenberg, W., & Burle, B. (2004). Suboptimal action monitoring in mediofrontal cortex results in performance declines. Poster presented at the 11th

annual meeting of the Cognitive Neuroscience Society, San Francisco, March.
Scheffers, M. K., & Coles, M. G. H. (2000). Performance monitoring in a confusing world: Error-related brain activity, judgments of response accuracy, and type of errors. *Journal of Experimental Psychology: Human Perception and Performance*, *26*, 141–151.
Van 't Ent, D., & Apkarian, P. (1999). Motoric response inhibition in finger movement and saccadic eye movement: A comparative study. *Clinical Neurophysiology*, *110*, 1058–1072.
Vidal, F., Burle, B., Bonnet, M., Grapperon, J., & Hasbroucq, T. (2003). Error negativity on correct trials: A reexamination of available data. *Biological Psychology*, *64*, 265–282.
Vidal, F., Hasbroucq, T., Grapperon, J., & Bonnet, M. (2000). Is the 'error negativity' specific to errors? *Biological Psychology*, *51*, 109–128.
Yeung, N. (2004). Relating cognitive and affective theories of the error-related negativity. In M. Ullsperger & M. Falkenstein (Eds.), *Errors, conflicts, and the brain: Current opinions on response monitoring*. Leipzig: MPI of Cognitive Neuroscience.
Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review*, *111*, 931–959.
Yeung, N., & Sanfey, A. G. (2004). Independent coding of reward magnitude and valence in the human brain. *Journal of Neuroscience*, *24*, 6258–6264.

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