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Oops!.. I did it again: An ERP and behavioral study of double-errors

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Abstract

To understand the sequealae of action monitoring failures, most previous studies have focused on neural (e.g., the ERN and Pe) and behavioral (e.g., post-error slowing) measures associated with correct trials that precede and follow errors. However, trials that precede and follow errors are not always correct, and no study to date has examined RT and ERP indices in double-error sequences that could shed additional light on multiple response monitoring failures. In the present study, we examined ERP and behavioral data surrounding double-errors to explore the possibility that double-errors could either result from the failed detection of the first error, or from a reduction in compensatory post-error behavioral adjustments. Results indicate a normal ERN and Pe surrounding double-errors; however, errors that followed errors were characterized by reduced post-error reaction time slowing. These data are discussed in terms of existing response monitoring data, and in terms of the utility of double-errors to shed light on distinct types of response monitoring failures.

Keywords: ERN; Error-related negativity; CRN; Correct response negativity; ACC; Errors; Response monitoring; Post-error slowing

1. Introduction

The ability to detect errors and prevent their future occurrence is crucial for successful response monitoring, and recent studies have begun to integrate behavioral and neurobiological data in order to understand action monitoring. Researchers are increasingly focusing on activity of the anterior cingulate cortex (ACC) in studies of response monitoring. Most notably, when subjects commit errors in speeded response tasks, the response-locked ERP is characterized by a distinctive negative deflection at fronto-central recording sites that begins around the time of the mistake and peaks approximately 50 ms later (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Gehring, Coles, Meyer, & Donchin, 1990; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001). This error-related negativity or error negativity (ERN/Ne) has been observed both when subjects make an incorrect response choice, and when subjects commit errors of action (e.g., responding when a response should be withheld; Scheffers, Bernstein, & Donchin, 1996); additionally, the ERN appears independent of stimulus and response modality (Holroyd, Dien, & Coles, 1998; Nieuwenhuis et al., 2001; Van't Ent and Apkarian, 1999). In this way, the ERN appears to be an electrophysiological index of the activity of a generic error detection system (Falkenstein et al., 1991; Falkenstein et al., 2000). Using whole head recording systems, the ERN has been source-localized to the anterior cingulate cortex (ACC; Dehaene, Posner, & Tucker, 1994; Holroyd et al., 1998).

Following the ERN, the error positivity (Pe) also appears specific to error trials. The Pe is a positive deflection that occurs 200–400 ms after the commission of an error (Falkenstein, Hoormann, Hohnsbein, & Kleinsorge, 2003; Nieuwenhuis et al., 2001) and has a more posterior midline scalp distribution than the ERN (Falkenstein et al., 2000). Van Veen and Carter (2002) noted that the

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Pe consisted of two subcomponents—a Pe closely linked to the ERN and a later Pe with a more posterior scalp distribution. There are several ideas as to what the Pe represents functionally, such as error salience or an orienting response to errors (Hajcak, McDonald, & Simons, 2003; Nieuwenhuis et al., 2001).

In addition to the error-specific ERN and Pe, a number of studies have focused on an ERN-like component that is evident in the response-locked ERP on correct trials. This correct response negativity (CRN; Ford, 1999) has a similar morphology and scalp topography as the ERN, and may index the response monitoring activity of the ACC on correct trials (Bartholow et al., 2005; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). In fact, several studies have reported a reduction in the CRN on correct trials that precede errors. Ridderinkhof and colleagues first reported that error-preceding trials were associated with an increased positivity in the response-locked ERP in a similar time window as the ERN (Ridderinkhof, Nieuwenhuis, & Bashore, 2003). Subsequent studies replicated this pattern of findings (Allain, Carbonnell, Falkenstein, Burle, & Vidal, 2004; Hajcak, Nieuwenhuis, Ridderinkhof, & Simons, 2005), and have suggested that this error-preceding positivity reflects a reduction in the CRN. Thus, the available data suggests that some errors may result from transient deficiencies in the response monitoring system on preceding correct trials, and that these impending failures of action monitoring can be indexed by a reduction in the CRN.

Behaviorally, errors tend to be characterized by relatively fast reaction times (RTs) compared to correct responses. However, trials immediately following errors have relatively long RTs—an effect loosely referred to as *post-error slowing*. Because error trials tend to be especially fast, some increase in RT following errors is likely a simple regression back toward the mean. Reaction time on correct trials that follow errors, however, actually tends to be slower than the average RT on *all correct trials* and, therefore, must reflect more than simple regression. This true post-error slowing has been interpreted as a compensatory adjustment that minimizes the risk of subsequent errors; support for this possibility comes from studies that report increased performance accuracy following errors (Laming, 1979; Rabbitt, 1966; Rabbitt & Rodgers, 1977). Just as the ERN, CRN, and Pe have been attributed to the response monitoring activity of the ACC, multiple functional neuroimaging studies have found that post-error RT adjustments relate to activation in the dorsolateral prefrontal cortex (PFC; Garavan, Ross, Murphy, Roche, & Stein, 2002; Kerns et al., 2004).

These data raise the possibility that in addition to 'monitoring' errors, some errors might also result from a failure to implement compensatory adjustments once an error has occurred. Thus, two possibilities present themselves with regard to why errors might occur following errors. First, double-errors may result from transient deficits in action monitoring, much like data on errors that follow correct trials (Allain et al., 2004; Hajcak et al., 2005; Ridderinkhof et al., 2003). That is, the first error in a double-error sequence might not be properly detected. Based on the work described above on the ERN and Pe, this failure of error detection should be associated with a reduced ERN and/or Pe elicited by the first error. Alternatively, double-errors may not be due to failures of error detection, but rather, might result from a failure to implement control processes following the initial error. If this were the case, post-error compensatory processes, such as post-error RT slowing might be reduced or absent following the initial error. It is unclear why double-errors occur, and no study to date has examined behavioral and ERP indices surrounding this unique type of action monitoring failure.

The primary aim of the present study is to identify double-errors and to explore the possibility that errors might result either from action monitoring failures or from the failure to implement post-error RT adjustments. The ERN, Pe, and post-error slowing were measured in the context of both single and double-errors during a speeded response task. If double-errors result from deficient error monitoring activity, the first error should be characterized by a reduced or absent ERN and/or Pe. On the other hand, double-errors may occur because of a failure to implement post-error behavioral adjustments—in which case, post-error slowing should be reduced or absent following the initial error.

2. Method

2.1. Subjects

ERP data from forty low-anxious (control) subjects who participated in previous studies were re-analyzed in the present investigation. These subjects were all students in an introductory psychology course. All subjects received course credit for their participation, and all subjects provided informed consent prior to the experiment.

2.2. Task

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

2.3. Procedure

After a brief description of the experiment, EEG/EOG sensor electrodes were attached and the subject was given detailed task instructions. Each subject was seated .5 m directly in front of the computer monitor and given 2 blocks of 24 practice trials. In one condition, the subjects were told to press the left button on the mouse when the color word was written in red, and the right mouse button when the word was written in green. In the other condition, counter-balanced across subjects, the correspondence between mouse button and word color was reversed. The subjects were told to place equal emphasis on speed and accuracy in their responses. Following two practice blocks, the subjects received 24 blocks of 48 trials (1152 total trials) with each block initiated by the subject. Word stimuli were presented for 200 ms at random intervals between 2000 and 2400 ms.

2.4. Psychophysiological recording, data reduction and analysis

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

Fz, Pz, A1, A2, and EOG were recorded by a Grass Model 7D polygraph with Grass Model 7P1 F preamplifiers (bandpass = 0.05–35 Hz). The EEG was digitized on a laboratory microcomputer at 200 samples per second, using VPM software (Cook, 1999). Data collection began at stimulus presentation and continued for 1500 ms.

Off-line, the EEG for each trial was corrected for vertical EOG and artifacts using the method developed by Gratton, Coles, and Donchin (1992; 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 the data fell out of A/D conversion range, or if the there was a 'flat' analog signal, exceeding 25 ms in duration; in addition, trials were rejected if the reaction time fell outside of a 200–800 ms window.[1] Single trial EEG data were lowpass filtered at 20 Hz with a 19 weight FIR digital filter as per Cook and Miller (1992). 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. Post-error slowing was defined by comparing RTs that followed the error of interest to the mean RT on correct trials.

To quantify the response-locked 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 average activity in the baseline-corrected ERP from 0 to 100 ms post-response. An area measure of the ERN was chosen because peak measures might be especially sensitive to noise, or low trial numbers.[2] The Pe was quantified as the average activity in a 200–400 ms baseline-correct post-response window. Behavioral and ERP measures were statistically evaluated using SPSS (Version 10.1) General Linear Model software with the Greenhouse–Geisser correction applied to p values associated with multiple df repeated measures comparisons.

For the duration of the paper, sequences of trials and the trial of interest will be referred to using the following notation: cEc, ceC, cEe, and ceE. In each case, c = correct and e = error; the capitalized letter reflects the trial of interest. For instance, cEe trials would refer to error trials that are followed by a subsequent error (e.g., the first error in a double-error sequence), whereas ceE trials would refer to error trials that were preceded by an error (e.g., the second error in a double-error sequence).

3. Results

3.1. Accuracy

Overall, subjects made an average of 84.2 mistakes ($SD = 39.8$); expressed as a percentage of valid trials, accuracy was 92.4% ($SD = 3.6\%$). Consistent with previous studies, RTs on correct trials that followed errors ($M = 422$ ms; $SD = 52$) were reliably slower than overall correct trial RT ($M = 406$ ms; $SD = 52$; $t(39) = 3.05$, $p < .01$—participants evinced the typical pattern of post-error slowing. Subjects made 5.2 ($SD = 5.2$) double-errors on average, and accuracy following all errors was 93.2% ($SD = 4.1\%$). The increase in performance accuracy following errors, however, did not reach statistical significance when compared to overall accuracy ($t(39) = .91$, $p > .35$). Thus, despite robust post-error slowing, we did not find reliably improved accuracy following errors. Because some subjects made relatively few double-errors, all subsequent

[1] About 200 ms was more than four standard deviations faster than the mean RT on correct trials, and accounted for 11.1 trails per subject on average, out of 986 trials per subject, on average; thus, about 1% of trials were excluded because of RT that fell below 200 ms.

[2] We also analyzed the ERN using a baseline to peak (in the 0–100 ms post-response window) measurement, and obtained an identical pattern of results.

behavioral and ERP analyses utilize only subjects who made five or more double-errors ($n = 20$).[3]

Within this sample of subjects, overall accuracy was 89.9% ($SD = 3.1\%$), and accuracy following errors was 90.2% ($SD = 3.4\%$). Consistent with the fact that this group was selected on their number of double-errors and number of double-errors was inversely related to overall accuracy ($r = -.84$, $p < .001$), this group had significantly more double-errors [$F(1,38) = 31.87$, $p < .001$], worse performance [$F(1,38) = 45.73$, $p < .001$], and worse post-error accuracy [$F(1,38)-43.51$, $p < .001$] than the other subjects in the initial sample.

3.2. ERPs

The response-locked ERPs at Fz, Cz, and Pz for single errors and the first error during a double-error sequence (cEc and cEe trials, respectively) are presented in Fig. 1, and ERN amplitude at Fz, Cz, and Pz are presented in Table 1. Both errors were characterized by a distinct negative deflection that began around the time of response and peaked approximately 50 ms later. We evaluated single errors compared to the first error in a double-error sequence with a 2 (error type: single error, first error in a double-error sequence) × 3 (location) using a repeated measures analysis of variance (ANOVA). As illustrated in the figure, the ERN was predominantly frontal [$F(2,38) = 31.27$, $p < .001$]. More importantly, there was no effect of trial type [$F(1,19) = 1.19$, $p > .25$] and no interaction between location and trial type [$F(2,38) < 1$].[4] Thus, the magnitude of ERNs observed on cEc trials (single errors) did not differ from ERNs on the cEe trials (the first of double-errors).

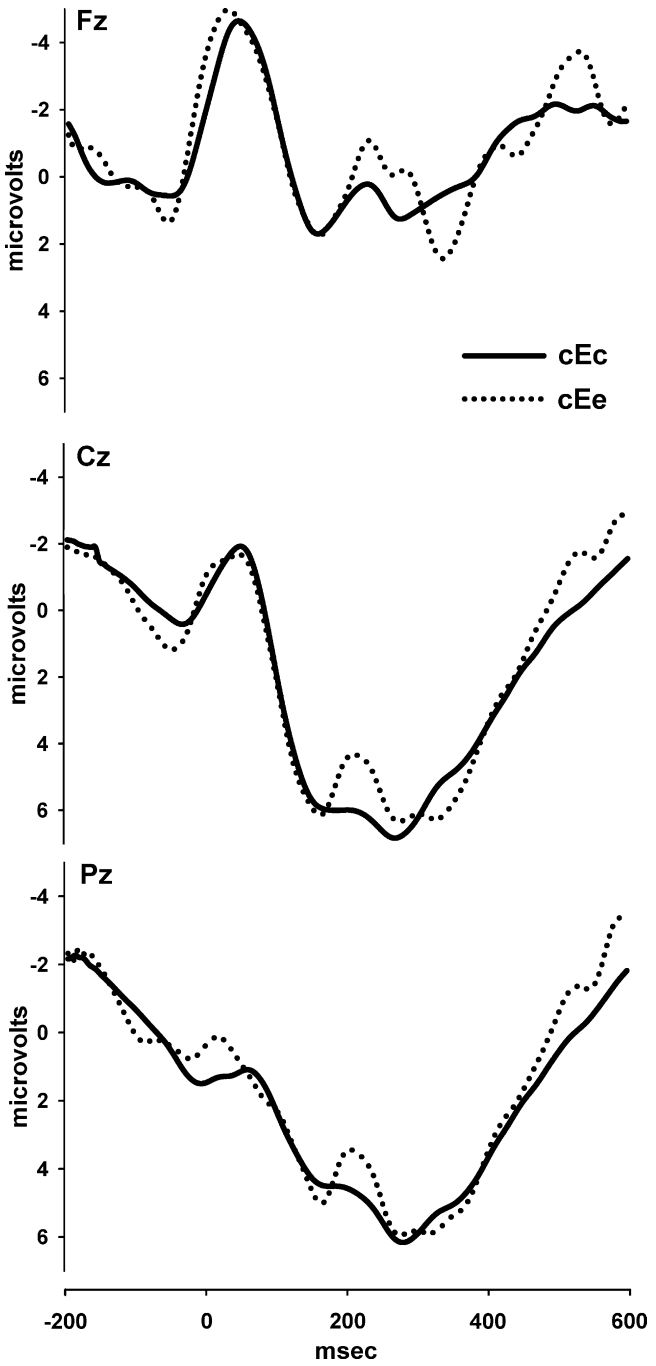

Fig. 1. Response-locked ERPs at Fz (top), Cz (middle), and Pz (bottom) for single errors (cEc) and the first error in a double-error sequence (cEe). Response onset occurred at 0 ms; negative is plotted upwards.

Table 1
Mean (and standard deviation) of ERN and Pe amplitudes at Fz, Cz, and Pz for single error (cEc) and double-error sequences (cEe) in micorvolts

	ERN		Pe	
	cEc	cEe	cEc	cEe
Fz	−3.65 (2.10)	−3.00 (2.86)	−.69 (3.88)	.88 (5.63)
Cz	−.84 (2.47)	−.43 (3.22)	3.20 (3.95)	3.44 (5.70)
Pz	.82 (2.25)	1.29 (3.87)	5.64 (6.20)	6.70 (10.44)

[3] In this group, the average number of double-errors included in the ERP analyses was 12.3 ($SD = 8.4$; minimum = 5, maximum = 38); the number of single errors included was 78.1 ($SD = 23.9$). Although such a small number of trials in the average ERP might raise questions about the reliability of the observed ERN component, it is important to note several things. First, there are no published data on how many trials are required to obtain a stable ERN. Although Polich (1986) found that 20 trials were required for a stable P300, it is possible that the ERN may be more reliably measured and therefore might require fewer trials to achieve stability. In fact, a number of published studies have reported ERN averages comprised of trials numbers similar to those reported here. For instance, Amodio et al. (2004) also used five trials as a minimum, and in one condition, the ERN was based on approximately 14 trials. In another recent study on the effects of haloperidol on the ERN, comparisons were made using an average of 10 and 6.25 trials (Zirnheld, Carroll, Kieffaber, O'Donnell & Hetrick, 2004). Collectively, these results suggest that meaningful ERN differences can be detected with a relatively small number of trials.To evaluate whether the effect of the different number of trials that formed ERPs for single (cEc) and the first error in a double-error sequence (cEe), we also analyzed a subset cEc trials that were matched to cEe trials on RT. The pattern of ERP results was identical in the two analyses.

[4] The ERN did not differ between cEc and cEe trials at Fz, where the ERN was maximal [$t(19) = 1.16$, $p > .25$]; similarly, the Pe did not differ between cEc and cEe trials at Pz, where the Pe was maximal [$t(19) = .63$, $p > .50$].

However, the peak latency of the ERN (measured at the Fz maximum) was prolonged for single errors ($M = 56.1$ ms, $SD = 4.4$) compared to the first of double-errors ($M = 50.8$, $SD = 6.9$; $t(19) = 3.38$, $p < .01$). Thus, the first error in a double-error sequence was characterized by an ERN that had comparable amplitude, but was earlier-peaking, compared to single error trials.

The magnitudes of the Pe at Fz, Cz, and Pz for single errors (cEc) and for the first error in a double-error sequence (cEe) are presented in Table 1. A 2 (error type: single error, first error in a double-error sequence) × 3 (location) repeated measures ANOVA confirmed the Pe was larger at posterior sites [$F(2, 38) = 19.56$, $p < .001$]. However, like the ERN, the Pe did not vary as a function of error type [$F(1, 19) < 1$], and the interaction between error type and location did not reach significance [$F(2, 38) = 2.71$, $p > .10$].[4]

3.3. Post-error slowing

The reaction times (RTs) for single- and double-error sequences are presented in Fig. 2. On single-error sequences (cEc trials: $M = 355$ ms, $SD = 57$; ceC trials: $M = 429$ ms, $SD = 46$), correct trials that followed errors were characterized by reliably slower RT than the mean correct RT [$F(1, 19) = 4.42$, $p < .05$]. Thus, single-errors were characterized by reliable post-error slowing.

Unlike single-errors where true post-error slowing occurred, double-error sequences (cEe trials: $M = 342$ ms, $SD = 60$; ceE trials: $M = 386$ ms, $SD = 44$) were not characterized by RTs slower than the mean RT on correct trials. Specifically, although the second errors in double-error sequences were reliably slower than single error trials [$F(1, 19) = 5.45$, $p < .05$], error trials that followed errors (e.g., ceE trials) were still significantly *faster* than both the average correct RT [$F(1, 19) = 5.00$, $p < .05$] and the RT on correct trials that followed single errors [e.g., ceC trials; $F(1, 19) = 12.43$, $p < .01$]. Thus, the double-error sequence provides evidence of some RT slowing following the first error, but the magnitude of this slowing is significantly less than that observed following single errors, and these trials are still characterized by reliably faster RT than the average correct trial.

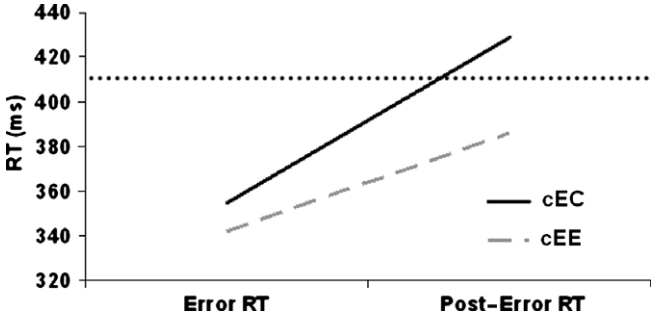

Fig. 2. Reaction time (milliseconds) for error and post-error trials in single error (cEC; solid black) and double-error (cEE; dashed grey) sequences; the dotted black line reflects the average correct trial reaction time.

4. Discussion

The present study evaluated ERPs and RT in single- and double-error sequences to evaluate possible mechanisms by which errors might be produced. It was hypothesized that errors might result from a failure in response monitoring or a failure to implement cognitive control and behavioral adjustment. In the present study, errors that were followed by subsequent errors (i.e., cEe sequences) were associated with ERN and Pe amplitudes that were virtually identical to single-error trials (i.e., cEc sequences). Insofar as the ERN and Pe reflect error detection and error awareness, respectively, these data suggest normal electrophysiological indices of response monitoring on error trials that were followed by a subsequent error. These data stand in contrast to a growing body of research indicating that single errors (e.g., errors that follow correct trials) might be due to lapses in response monitoring activity on the preceding correct trial (Allain et al., 2004; Hajcak et al., 2005; Ridderinkhof et al., 2003). Unlike single errors, then, double-errors do not appear to occur following a lapse in response monitoring.

Rather, the present study suggests that double-errors may result from a failure to appropriately implement post-error RT adjustments. Although both single and double-errors were associated with some post-error RT slowing, the magnitude of this effect was larger in single- than double-error sequences and only in the single-error sequence did the post-error RT exceed the average RT computed from all correct trials. These data indicate that double-errors may represent a distinct type of error, and occur due to failures to implement controlled post-error compensatory behavioral adjustments following the initial error. The notion that there might be two causal routes to error commission is consistent with functional neuroimaging data that posits a differential role of the ACC and PFC in error detection and compensatory behavioral adjustments, respectively (Kerns et al., 2004; Garavan et al., 2002). Within this framework, we have shown in a previous study (Hajcak et al., 2005; Study 2) that single errors most likely reflect ACC-related failures, and in the present study that double-errors might result from failures of the PFC. Further investigation of double-errors using functional neuroimaging could substantiate this possibility.

It is important to note that double-errors were relatively frequent—accuracy after errors was not better than overall accuracy, and occurred after approximately 7% of errors in the full sample of 40 subjects. Although accuracy was not better following errors, correct trials that followed errors were characterized by a robust post-error RT slowing. Thus, when simply comparing post-error accuracy to post-error RT, the data appear to suggest that post-error slowing is not compensatory: RT was increased following errors but accuracy was not improved.

However, this comparison involves different trials: Only correct trials are included in the post-error slowing computation whereas both correct and error trials are used to compute post-error accuracy. When post-error trials were separately analyzed based on accuracy (e.g., ceC versus ceE), it becomes clear that the degree of post-error slowing differs as a function of accuracy: RTs were longer following single than double-errors. These data provide support for the notion that post-error RT adjustments are functionally related to increased performance accuracy.

Interestingly, although ERN amplitudes did not differ, the ERN peaked *earlier* on the first error of double-error sequences compared to single errors. The meaning of ERN latency variation is less understood than amplitude-related differences. One study, however, found an earlier-peaking ERN for errors when participants were instructed to correct errors, suggesting that cEe trials in the present study might have been associated with earlier or more frequent error-correction (Fiehler, Ullsperger, & von Cramon, 2005). Possibly, error correction could interfere with the implementation of subsequent post-error RT adjustments.

In terms of current computational accounts of ERN magnitude, both the reinforcement learning theory of the ERN and the conflict monitoring theory of the ERN predict that the ERN should relate to increased cognitive control on subsequent trials (Holroyd & Coles, 2002; Yeung, Cohen, & Botvinick, 2004). In fact, early ERN studies suggested that the ERN may be *directly* related to compensatory post-error RT slowing and post-error performance adjustments to increase accuracy (Gehring et al., 1993). Consistent with this possibility, recent work by Debener and colleagues related trial-by-trial variation in the ERN to subsequent behavioral measures (Debener et al., 2005). Because cEc and cEe trials had comparable ERN and Pe amplitudes, but were associated with different post-error behavior, the present data are consistent with the possibility that the ERN and Pe relate *indirectly* to subsequent performance adjustments. In this view, ACC-related activity is necessary, but not sufficient, for behavioral adjustment. This is consistent with recent data indicating that the PFC mediates the relationship between error-related ACC activity and behavioral adjustments (Garavan et al., 2002; Kerns et al., 2004). An intact ERN/Pe may signal the need to increase cognitive control; however, such adjustments may not be properly implemented on some trials (i.e., double-error sequences).

Limitations of the present study might be acknowledged at this point. For example, the present ERN analyses are based on relatively few trials. Sensitive to this issue, we chose to employ an area measure of the ERN in order to reduce noise that may have unduly influenced a peak or base-to-peak measure. Nonetheless, future studies that investigate double-errors would be well-advised to utilize an experimental paradigm designed specifically to increase the number of observed double-errors.

A second limitation of the present data set arises from the fact that the data analyzed was obtained from 20 subjects who had reliably worse performance than the 20 subjects whose data were not included. Thus, it is not clear to what extent the present data will generalize to better performers. This issue is fairly typical in ERN studies, where subjects who commit low number of errors are excluded from analyses, and experimental results are assumed to generalize to even the best performers. This concern reinforces the need for experimental procedures in which double-errors are more frequent.

In sum, the present data indicates normal ACC function in the context of double-errors, and reduced implementation of subsequent control processes reflected in post-error RT. In this way, double-errors may be a useful tool for investigating the inter-relationship among those action monitoring systems that detect the need for enhanced cognitive control, and those that implement such behavioral adjustments.

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