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Frontal Monitoring and Parietal Evidence: Mechanisms of Error Correction

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Abstract

■ When we respond to a stimulus, our decisions are based not only on external stimuli but also on our ongoing performance. If the response deviates from our goals, monitoring and decision-making brain areas interact so that future behavior may change. By taking advantage of natural variation in error salience, as measured by the RT taken to correct an error (RT_{EC}), here we argue that an evidence accumulation framework provides a potential underlying mechanism for this variable process of error identification and correction, as evidenced by covariation of frontal monitoring and parietal decision-making processes. We study two early EEG signals linked to monitoring within medial PFC—the error-related negativity (ERN) and fronto-central theta activity—and a third EEG signal, the error positivity (Pe), that is thought to share the same parietal substrates as a signal (the P3b) proposed to reflect evidence accumulation. As predicted, our data show that on slow RT_{EC} trials, frontal monitoring resources are less strongly employed, and the latency of the Pe is longer. Critically, the speed of the RT_{EC} also covaries with the magnitude of subsequent neural (intertrial alpha power) and behavioral (post-error slowing) adjustments following the correction. These results are synthesized to describe a timing diagram for adaptive decision-making after errors and support a potential evidence accumulation mechanism in which error signaling is followed by rapid behavioral adjustments. ■

INTRODUCTION

Activity in parietal and frontal brain regions is critical for adaptive decision-making. Studies in nonhuman primates suggest a mechanism for decision-making in which information accumulates in parietal (and other) areas until neural activity reaches the threshold level required to make a behavioral response (Gold & Shadlen, 2007; Mazurek, Roitman, Ditterich, & Shadlen, 2003). Importantly, frontal areas including the medial PFC (mPFC) play an important role in this process, monitoring decisions and signaling the need for behavioral adjustments such as the allocation of greater attentional resources (Ito, Stuphorn, Brown, & Schall, 2003; see Heekeren, Marrett, & Ungerleider, 2008; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004, for reviews).

Recently, the idea has arisen that an evidence accumulation framework may also explain error processing (Murphy, Robertson, Allen, Hester, & O'Connell, 2012; Steinhauser & Yeung, 2010, 2012; Wessel, Danielmeier, & Ullsperger, 2011; Ullsperger, Harsay, Wessel, & Ridderinkhof, 2010). By analogy to the case in which sensory evidence is evaluated, evidence for an erroneous response integrates until a threshold is reached, at which point behavioral corrections can be undertaken. Notably, studies of evidence accumulation in other contexts, such as perceptual decision-making, typically take advantage of parametric variation in stimulus salience—for example, the motion coherence of a moving dot stimulus (Gold & Shadlen, 2007)—to manipulate the amount of sensory evidence. However, the salience of an error cannot be modulated as directly as stimulus salience in perceptual decision-making studies. To account for this issue, here we take advantage of natural variation in the RT taken to correct the error (RT_{EC}) by dividing instances of error correction into short and long RT_{EC} trials. We hypothesize that the salience of an error correlates inversely with RT_{EC}, an intuition that recent data suggest can be useful: The Pe peak latency, for example, is correlated with the latency at which error awareness is indicated (Murphy et al., 2012).

With this approach, implications of the evidence accumulation hypothesis can also be addressed for other processes necessary for behavioral corrections after errors, including predictions that vary in their degree of certainty. On the one hand, more rapid accumulation of evidence for an error and faster corrections should likely be preceded by stronger correlates of error commission, as indexed by greater response conflict at the time of the initial response (Rodriguez-Fornells, Kurzbuch, & Munte, 2002). On the other hand, the effect of the speed of evidence accumulation on subsequent processes is potentially unclear. Under one scenario, more rapid accumulation, indicating greater evidence for an error, might be associated with more demand on the cognitive processes responsible for deciding whether to initiate post-error

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behavioral adjustments. However, the opposite might also be true: Because it indicates more confidence about the existence of an error in the context of a time-limited decision (Kiani, Corthell, & Shadlen, 2014), more rapid accumulation might place less demand on the decision to initiate such compensations. Specifically, when greater certainty exists that an error was committed (or that the response was correct, for that matter), the decision about whether to engage post-error behavioral adjustments would be easier, compared with the intermediate case in which lesser, more uncertain evidence for an error accumulates more slowly to threshold. Finally, the rapidity of evidence accumulation might have no effect: If such processes are not triggered until evidence for an error reaches a threshold, identical thresholds should trigger the same behavioral adjustments, irrespective of speed.

Of course, a great deal of work has previously investigated the neural correlates of error processing, including EEG studies that have identified candidate electrophysiological correlates for many of these processes. When an error occurs, monitoring signals originating in the mPFC are represented in initial signals (both error-related negativity [ERN] and theta power; e.g., Endrass, Klawohn, Preuss, & Kathmann, 2012; Endrass, Reuter, & Kathmann, 2007; Luu, Tucker, & Makeig, 2004) that may act as one of the sources of information indicating a potential error (Ullsperger et al., 2010). A later EEG signal, the error positivity (Pe), is evoked by errors in comparison with correct responses in parietal areas (Navarro-Cebrian, Knight, & Kayser, 2013; Shalgi, Barkan, & Deouell, 2009; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). This Pe signal has been suggested to share the same neural substrates as the P3b (Ullsperger et al., 2010; Ridderinkhof, Ramautar, & Wijnen, 2009). Because the P3b has recently been shown to reflect evidence accumulation in decision-making (Kelly & O'Connell, 2013; O'Connell, Dockree, & Kelly, 2012), the Pe may also represent the accumulation of evidence for error occurrence (Steinhauser & Yeung, 2010, 2012). Finally, cognitive (i.e., intertrial alpha power) and behavioral (post-error slowing) adjustments are commonly observed after errors. In particular, intertrial alpha suppression after errors in comparison with correct responses has been considered a measure of post-error modulations in cortical arousal (Navarro-Cebrian et al., 2013; Compton, Arnstein, Freedman, Dainer-Best, & Liss, 2011; Carp & Compton, 2009) and post-error slowing (Rabbitt, 1966), defined as a longer RT in the trial following an error (RT_{n+1}), is often used to measure behavioral adjustments after errors.

Thus, in the current study, we take advantage of natural variation in the RT_{EC} as a proxy for parametric variation in the salience of an error, permitting us to evaluate whether the evidence accumulation framework might account for various aspects of error correction. Specifically, as linked by RT_{EC} values, we analyze whether early error-monitoring mechanisms are associated with subsequent variation of Pe latency and whether later post-error adjustments covary with error-monitoring and evidence accumulation processes. We predicted the following: (1) faster (slower) RT_{EC} will be associated with an increased (decreased) amplitude of early ERN monitoring processes, (2) faster (slower) RT_{EC} will be associated with shorter (longer) Pe latencies, and (3) faster (slower) RT_{EC} will be associated with both increased (reduced) intertrial alpha power and reduced (increased) behavioral slowing on the next trial. In testing these predictions via the RT_{EC}, we seek to determine whether previous error-related findings within the literature can be linked to an accumulator model of error correction.

METHODS

Participants

Fifteen University of California, Berkeley, students participated in the experiment in exchange for monetary compensation. One participant was excluded for failing to follow task instructions. Two more participants were rejected from the EEG analysis because of excessive motion and muscle artifacts. Twelve students were included in the final analysis (nine women) ages 18–25 years (mean, 20.3 ± 2.0 years). All participants gave written informed consent in accordance with the Committee for the Protection of Human Subjects at the University of California, San Francisco and University of California, Berkeley. Participants were right-handed and had normal or corrected-to-normal vision.

Procedure

A total of 100 pictures of male and female faces unknown to our participants were used in the experiments (Navarro-Cebrian et al., 2013). They were converted to black and white and resized to 113 × 151 pixels using Adobe Photoshop CS4 (Adobe Systems, San Jose, CA). Targets subtended 2.55° of visual angle. Pictures were obtained from one of the authors (ANC) and from the Face Database of the Max-Planck Institute for Biological Cybernetics in Tübingen, Germany (faces.kyb.tuebingen.mpg.de; Troje & Bulthoff, 1996). The task was presented using Presentation software (Neurobehavioral Systems, Inc., Berkeley, CA).

Participants were presented with a previously validated version of the Flanker task (Navarro-Cebrian et al., 2013) designed to produce intermittent errors (Figure 1). All trials contained five high contrast (black and white) human faces, and participants were asked to respond via button press with their right hand (index and middle fingers) whether the target (middle) face was upright or upside down. The trial started with a fixation cross for 200 msec. After the cross disappeared, four flanker faces were shown alone for 80 msec and then together with the target (middle face) for an additional 30 msec. To make the task more difficult, the flanker faces could be congruent or incongruent with the target regarding the orientation of the face. After the flanker and target faces disappeared, a

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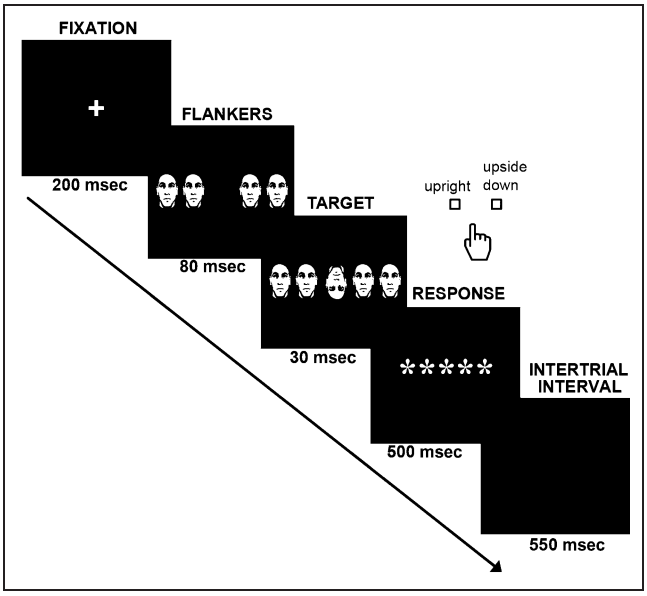

Figure 1. Paradigm. After a fixation cross, flanker faces (congruent or incongruent with the target) were shown alone for 80 msec and then together with the target for 30 msec more. Participants were instructed to indicate with a button press whether the target was upright or upside down. They were asked to correct themselves as fast as possible if an error was made. An ITI of 550 msec was presented before the next trial started.

group of five asterisks was presented in the middle of the screen for 500 msec to indicate when to respond. Importantly, participants were required to correct themselves as quickly as possible whenever they realized they had made an error by pressing the correct button (giving rise to a second RT denoted as the RT taken to correct the error or RT_{EC}; see below). An additional intertrial interval (ITI) of 550 msec preceded the next trial.

Behavioral Analysis

Only those trials in which participants made an error and corrected themselves were used for analysis. Trials were sorted by the RT for the second motor response in each error trial—that is, the RT_{EC}, as determined by the latency of the correction minus the latency of the initial (erroneous) response. We created two groups of trials for which the RT_{EC} values were well differentiated. On the basis of the RT_{EC} distribution for each participant, in one group, trials with an RT_{EC} equal to or shorter than the 25th percentile within participants were included; in the other group, trials with an RT_{EC} equal to or greater than the 75th percentile within participants were included. These quartiles were chosen for analysis to maximize potential differences in error salience while retaining an adequate number of trials for comparisons. Additionally, we evaluated possible differences in RTs in the trials following errors (post-error trials; RT_{n+1}) to study the relationship to post-error slowing. We predicted that the uncertainty generated in some trials by reduced evidence for the error should lead to slower RTs in the trials after the errors (i.e., greater post-error slowing).

EEG Procedure

As noted above, two participants were rejected from the EEG analysis because of excessive motion and muscle artifacts. EEG data were collected from 64 channels at a sampling rate of 512 Hz (Biosemi, Amsterdam, The Netherlands; www.biosemi.com). The Biosemi ActiveTwo system allows low-noise recordings free of interference consisting of a feedback loop between an electrode (CMS) and a passive electrode (DRL; see www.biosemi.com/faq/cms&drl.htm for more details). Vertical and horizontal eye movements were recorded using four electrooculography electrodes placed at the outer canthi of the right and left eyes, and above and below the right eye, aligned with the pupil. EEG data analysis was performed using MATLAB scripts based on functions from the EEGLAB toolbox (Delorme & Makeig, 2004). The data were re-referenced offline to an average reference and filtered to remove frequencies >50 Hz and <0.3 Hz. Portions of continuous data with excessive artifacts were rejected after visual inspection. In addition, independent components were estimated for individual participants using the filtered but unsegmented EEG data to identify and remove eye movement and muscular artifacts. ICA decomposition relied on the independent component analysis functions of EEGLAB (runica algorithm). Sixty-four ICA components were identified for each participant, and IC scalp topographies, time courses, and spectral characteristics were inspected visually to identify and reject components related to blinks and eye movements (Jung et al., 1998).

ERP Analysis

Data were segmented and time locked to the onset of the first motor response for analysis of the ERN and the Pe components. Epochs were created starting 500 msec before the onset of the error and finishing 1000 msec after it. A prestimulus time window from −500 msec to the onset of the initial stimulus was used as a baseline. First, we calculated the amplitude of the ERN, an ERP originating in the mPFC and associated with conflict monitoring and uncertainty (Navarro-Cebrian et al., 2013; Van Veen & Carter, 2002). For the ERN component, three frontocentral electrode sites were selected for analysis (Fz, FCz, and Cz). As in our previous research (Navarro-Cebrian et al., 2013), the average minimum amplitude between −20 and 50 msec around the onset of the response was calculated to measure the activity of the ERN component. Next, we analyzed the error-related positivity (Pe) to examine whether the RT for the error correction (RT_{EC}) varied with the Pe latency. The Pe latency was defined as the time to the most positive point in the epoch for the average of three centroparietal electrodes (CP1, CPz, and CP2). Statistical analyses were performed in SPSS (version

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15.0 for Windows, IBM, Armonk, NY). Comparisons were made using the Fisher's least significant difference test and *t* tests.

Time–Frequency Analysis

Time–frequency calculations were computed using MATLAB scripts based on functions from the EEGLAB toolbox. Specifically, we used the *newtimef* function available for EEGLAB to calculate the event-related changes in spectral power relative to baseline (event-related spectral perturbation). The use of the Morlet wavelet decomposition in this function allows power changes to be observed in both the time and frequency domains, in contrast with fast Fourier transform methods that only have resolution in the frequency domain. Wavelet cycles began at 3 and increased with frequency, reaching half the number of cycles in the equivalent fast Fourier transform window at its highest frequency. Data were filtered to remove frequencies >50 Hz and <0.3 Hz. We were interested in the power of the theta band as a measure of error monitoring (Navarro-Cebrian et al., 2013; Trujillo & Allen, 2007; Debener et al., 2005; Luu et al., 2004). To study frontal theta band power, epochs were created starting 2000 msec before the onset of the error and finishing 2000 msec after it. Values from 1000 to 500 msec before the error were used as a baseline to avoid border and edge artifacts. As for the ERPs, RT_{EC} duration was used to evaluate other segments of the trial by separating epochs into two conditions (fast and slow RT_{EC}). Frequencies of 4–7 Hz were used to study the theta band in the FCz channel, as the electrode site FCz is in the most representative location for post-error monitoring activity and has been used in previous research (Navarro-Cebrian et al., 2013; Cavanagh, Cohen, & Allen, 2009) to measure post-error theta power. Time series were plotted and differences in latency and amplitude of frontocentral theta activity were calculated for the fast and slow RT_{EC} conditions by analyzing the maximum amplitude and the latency at which this maximum occurred from −500 to 500 msec around the error.

In addition to the frontocentral theta power analysis, we calculated whether frontocentral theta phase values were consistent over trials (intertrial coherence [ITC]) in the FCz channel. ITC was calculated with the *std_itc* function of EEGLAB. Phase coherence values range from 0 to 1, "0" indicating random phases and "1" indicating perfect phase locking to the response across trials. We compared the ITC values for fast and slow RT_{EC} in the same time window as the ERN and for the theta band (4–7 Hz, as above). Because the ERN has been suggested to emerge from the theta activity phase locked to the response (Luu et al., 2004), we predicted that the ITC results would agree with those obtained for the ERN. A greater ITC value at the time of the response would demonstrate more synchronization between activity within the mPFC and the manual response (error).

Lastly, in addition to post-error behavioral changes (post-error slowing), we studied post-error neural adjustments by analyzing the ITI for differences in alpha band power. Here we evaluated whether alpha power varies with the degree of error salience. Epochs were created starting 2000 msec before stimulus onset and finishing 2000 msec afterward. Values from 1000 to 500 msec before the onset of the stimulus were used as a baseline. The time window from 300 to 500 msec within the ITI, during which decisions and manual responses (corrections) no longer took place, was used for analysis. Specifically, although participants could take longer than the 500 msec correction time window to correct themselves, no error correction was detected within the ITI after an additional 300 msec on any trial. On the basis of previous research into alpha suppression (Carp & Compton, 2009), frequencies of 10–14 Hz were included in the analysis, and one posterior midline channel location (Pz) was chosen as a representative electrode (Navarro-Cebrian et al., 2013; Carp & Compton, 2009). As for the other ERP and time–frequency analyses, two groups were created and included in the analysis based on the RT to the error (fast and slow RT_{EC}).

RESULTS

Behavioral Results

Participants completed an average of 895 trials (*SD* = 5.45, range = 882–900). They made an average of 148 errors (16.5% of the total number of trials, *SD* = 40.46, range = 91–211). The average RT for error correction (RT_{EC}) was 212 msec (*SD* = 45, range across participants = 154–284). In keeping with previous literature, these error correction RTs were quite rapid, suggesting that the process for movement correction begins very early in the trial (Christensen, Kristiansen, Rowe, & Nielsen, 2008; Fiehler, Ullsperger, & Von Cramon, 2004; Schmidt & Gordon, 1977; Higgins & Angel, 1970). In contrast, the average RT for the initial erroneous response in the error trials (i.e., the instigating error [RT_E]) was 349 msec (*SD* = 41, range = 266–420), and the average RT for correct trials was 417 msec (*SD* = 34, range = 358–478). Consistent with other work (Navarro-Cebrian et al., 2013; Pailing, Segalowitz, Dywan, & Davies, 2002; Ratcliff & Rouder, 1998; Gehring, Goss, Coles, Meyer, & Donchin, 1993), the RT_E was significantly shorter than the RT for correct responses ($t(11) = 7.58$, $p < .0001$).

On the basis of the RT_{EC}, trials were then sorted to create two groups: one with trials in which RT_{EC} was equal to or less than the 25th percentile (within participants) and one with trials in which the RT_{EC} was equal to or greater than the 75th percentile. The average RT_{EC} for the group of trials with the faster error corrections was 126 msec (*SD* = 43; range across participants = 64–183 msec) and for the group of trials with the slower error corrections was 326 msec (*SD* = 69; range across

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participants = 243–467 msec). The corresponding RT_E for the group of trials with the faster error corrections was 370 msec (SD = 35; range = 326–447 msec) and for the group of trials with the slower error corrections was 322 msec (SD = 54; range = 183–397 msec), both of which remained faster than for correct responses ($t(11) = 5.45$; $p < .001$ and $t(11) = 7.07$; $p < .0001$, respectively) and differed significantly from each other ($t(11) = 4.1$; $p = .002$).

One theory suggests that a single evidence accumulation process begins at stimulus onset (Rabbitt, 2002; Maylor & Rabbitt, 1989) and that the error correction response reflects not a separate error-related process, but rather the correct outcome of evidence accumulation related to the initial stimulus. Under such a theory, calculating the RT_{EC} from the time of stimulus onset ($RT_{EC\text{-}SO}$), rather than from the time of the initial erroneous response, might equalize average RT values for the fast and slow RT_{EC} trials. In contrast, if the RT_{EC} represents the outcome of a separate (or interacting) error-related process, fast and slow RT_{EC} trials, when locked to the stimulus, should continue to give rise to different $RT_{EC\text{-}SO}$ values. To test this idea, we calculated this new stimulus-locked $RT_{EC\text{-}SO}$ value relative to the onset of the stimulus for the original fast and slow RT_{EC} groups, rather than relative to the onset of the error. The $RT_{EC\text{-}SO}$ remained significantly slower for the slow RT_{EC} condition in comparison with the fast RT_{EC} condition (fast $RT_{EC\text{-}SO}$ = 496 msec; slow $RT_{EC\text{-}SO}$ = 647 msec; $t(11) = 10.18$; $p < .0001$).

Additionally, the combination of congruent and incongruent trials in the current experiment, which was motivated by the need to collect as many trials as possible in each condition, raises the possibility that fast (slow) RT_{EC} trials might consist of a disproportionate number of congruent (incongruent) trials. To rule out this concern, we analyzed the number of each type of trial for each condition. No significant differences were found between the two conditions in the number of congruent ($t(11) = 1.39$; $p = .19$) and incongruent ($t(11) = 0.29$; $p = .78$) trials.

ERP Results

To study whether the ERN or Pe activities were related to the RT_{EC}, we compared the fast RT_{EC} and slow RT_{EC} conditions for the frontal and parietal components of interest (ERN and Pe) evoked by errors—specifically, the amplitude of the ERN and the latency of the Pe. We found significant differences between RT_{EC} conditions (Figure 2): pairwise comparisons (Fisher's least significant difference test) showed that the fast and slow RT_{EC} conditions were significantly different at the time of the ERN ($p = .027$) and the Pe ($p = .048$) signals. This finding was manifest as enhanced ERN activity and shorter Pe latency for the fast RT_{EC} condition.

Time–Frequency Results

We then analyzed changes in theta power after errors. Statistical analyses demonstrated significant differences

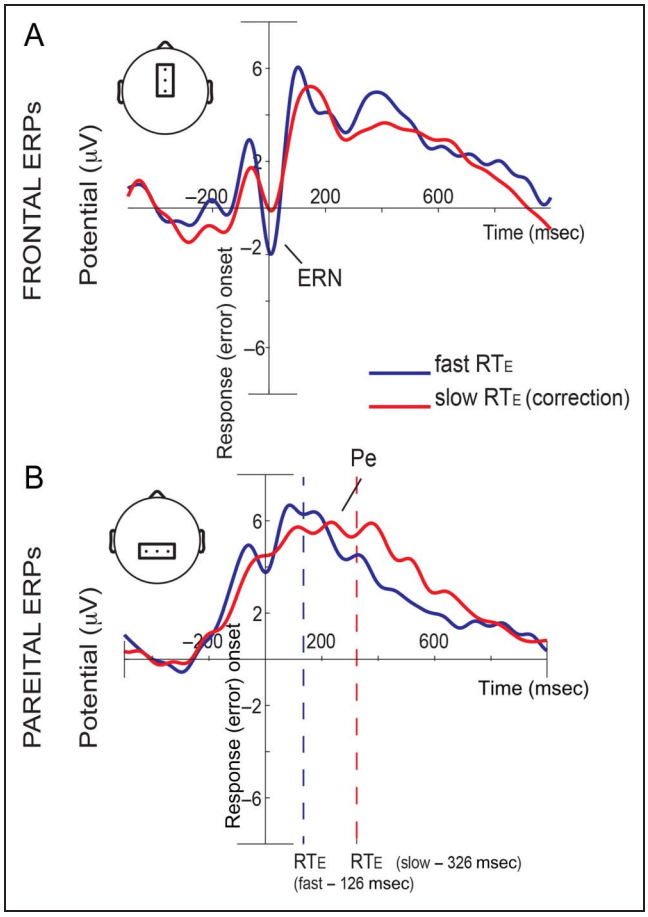

Figure 2. ERP responses. The response-locked average in frontal electrodes (Fz, FCz, Cz) is shown in the upper figure and response-locked average in parietal electrodes (CP1, CPz, CP2) is shown in the lower figure. Fast RT_{EC} (error correction) is shown in blue, and slow RT_{EC} is shown in red. Panels show a significantly greater ERN amplitude (A) and a significantly shorter Pe latency (B) for the fast RT_{EC} condition.

in the latency ($t(11) = 4.16$; $p = .002$), but not in the amplitude ($t(11) = 0.61$; $p = .56$ (ns)) of the theta power in FCz at the time of the error. Figure 3A shows an earlier peak of the theta power for the fast RT_{EC} (20 msec after the error) compared with the slow RT_{EC} (100 msec after the error). We also found that compared with the slow RT_{EC} condition, the fast RT_{EC} condition showed increased ITC for theta activity locked to the response across trials ($t(11) = 9.93$; $p = .002$; Figure 3B).

A diminished alpha power after errors, compared with correct responses, has been suggested to be indicative of higher alertness and reflective of neural adjustments (Navarro-Cebrian et al., 2013; Carp & Compton, 2009). Time–frequency analyses were performed to test whether there was less post-error alpha power in those cases in which reduced frontal activity in monitoring areas led to greater uncertainty about performance and a slower RT to the error. Significant differences in power in the alpha band were found between the fast and slow RT_{EC} in the ITI ($t(11) = 5.21$; $p = .0003$; Figure 4, dashed box), indicating less alpha power in the slow RT_{EC} condition.

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Post-error Behavioral Adjustments

Finally, we calculated the RTs in the trials following the errors (post-error trials) to assess differences in post-error slowing (Figure 4). Trials following correct responses (average RT_{n+1} of 399 msec) did not differ significantly from trials following faster error corrections (average RT_{n+1} of 402 msec; correct responses vs. fast correction trials: $t(11) = 0.77$, $p = .46$). In contrast, trials following slower error corrections had an average RT_{n+1} of 418 msec, which was significantly longer than RTs following correct trials ($t(11) = 2.75$; $p = .019$) and fast correction trials ($t(11) = 2.99$; $p = .012$). These results indicate increased post-error slowing in the cases in which participants took a longer time to respond to the error.

DISCUSSION

To investigate the role of evidence accumulation in error correction, we took advantage of natural variation in error salience to define fast and slow error correction trials and to evaluate differences in conflict monitoring, decision-making, and post-error adjustments. Previous EEG studies (Kelly & O'Connell, 2013; O'Connell et al., 2012) have characterized a centroparietal electrical signal that increases with incoming evidence and peaks at the time of response execution. This signal shares characteristics with the classical P3b and, based on similarities between the P3b and the Pe (Ullsperger et al., 2010; Ridderinkhof et al., 2009), has led to proposals that the Pe indexes the accumulation of evidence for the existence of an error (Steinhauser & Yeung, 2012). Consistent with this notion, here we found that the amplitude of early monitoring signals was greater and the latency of the Pe was shorter when participants rapidly corrected themselves. Furthermore, we determined that post-error cognitive and behavioral adjustments depended upon this decision-making process and, along with the latency of the Pe, varied with the RT_{EC}. Together these signals define a potential sequence of events that underlies error correction.

Error Monitoring Processes

The earliest signal in this sequence of EEG events—activity of the mPFC—has long been a focus of attention in EEG studies that analyzed activity in frontocentral electrodes after errors using ERPs (ERN signal; Endrass et al., 2007, 2012; Shalgi et al., 2009; O'Connell et al., 2007; Gehring et al., 1993) or time–frequency analysis, specifically in the theta band (Cavanagh, Zambrano-Vazquez, & Allen, 2012; Cohen & Cavanagh, 2011; Cavanagh

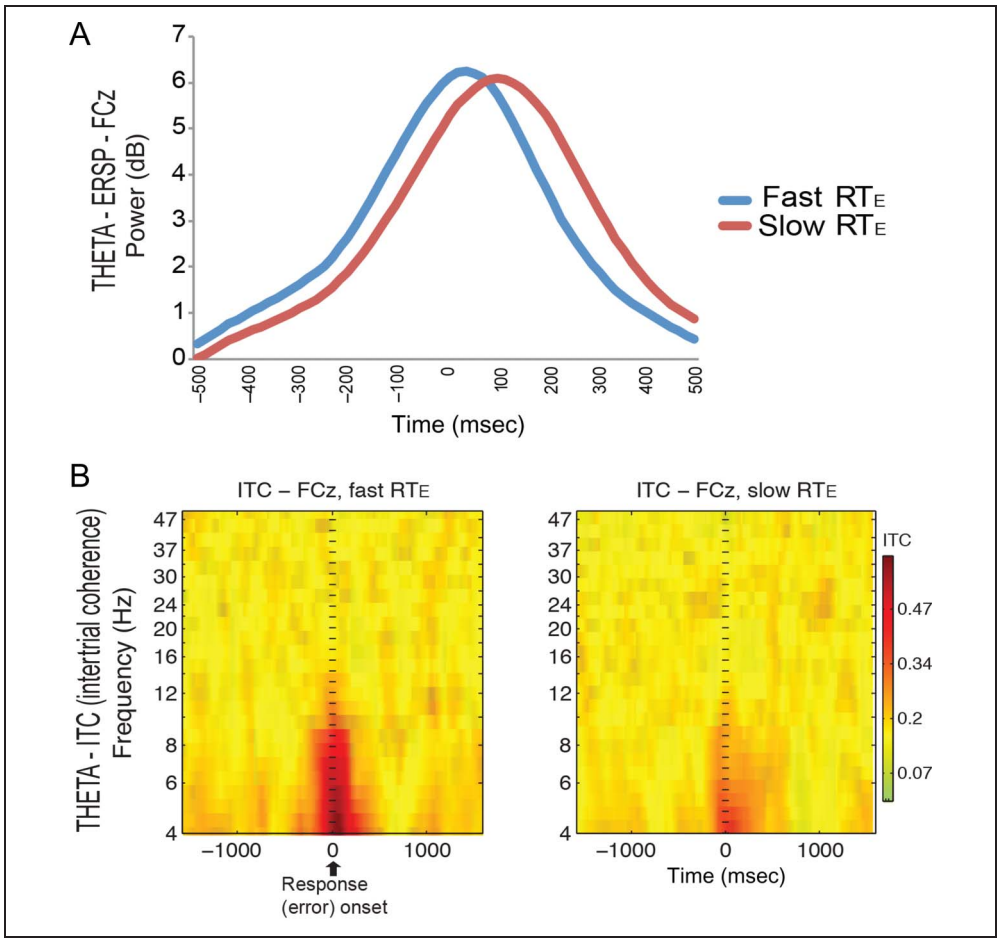

Figure 3. Event-related changes in theta power. (A) Event-related changes in theta power in the FCz channel. A decreased latency of the theta amplitude for the fast RT_{EC} condition (fast error correction, in blue) can be seen in comparison with the slow RT_{EC} condition (red). (B) A greater intertrial phase coherence (ITC) can be seen in the theta band for the fast RT_{EC} condition (left) compared with the slow RT_{EC} condition (right) at the time of error onset.

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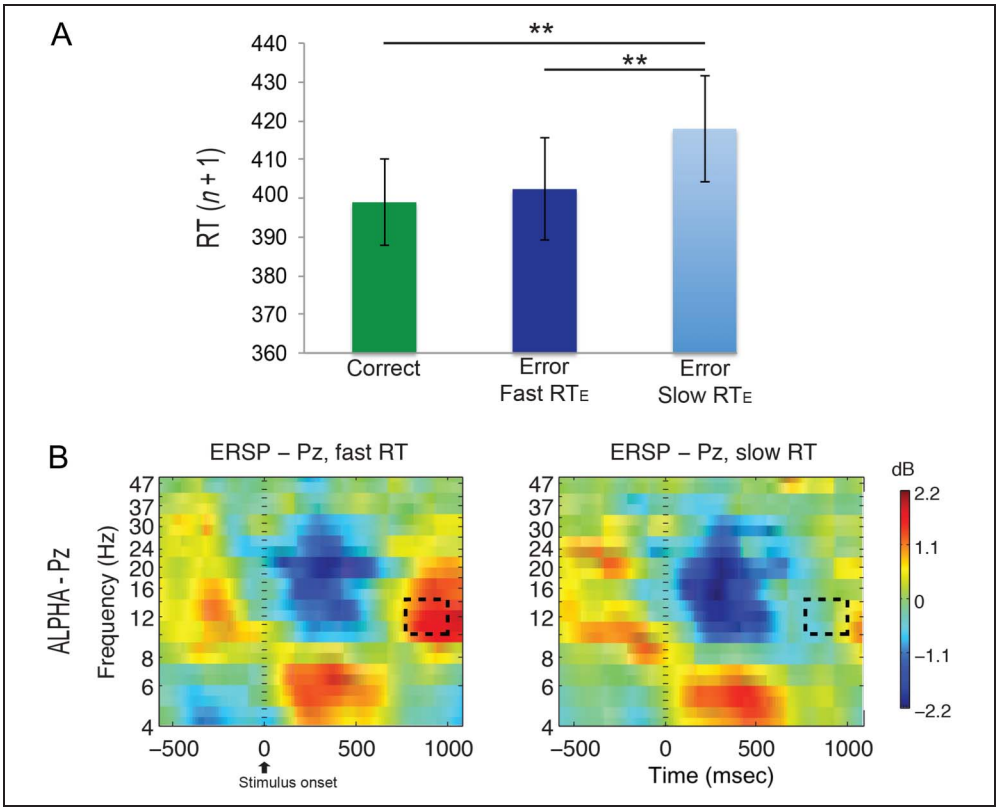

Figure 4. Post-error adjustments. (A) The RT (RT_{n+1}) for trials following correct responses, trials following fast RT_{EC} errors (fast correction), and trials following slow RT_{EC} errors (slow correction), respectively. The plot indicates a greater post-error slowing (RT in the trial following the error) after trials with a slow RT_{EC} error. (B) Less alpha power can be seen in the ITI (dashed rectangle) in the slow RT_{EC} condition (right) compared with the fast RT_{EC} condition (left).

et al., 2009; Cohen, Ridderinkhof, Haupt, Elger, & Fell, 2008; Trujillo & Allen, 2007; Debener et al., 2005; Luu et al., 2004). Here we demonstrate that the amplitude of the ERN differs between fast and slow RT_{EC} conditions, suggesting a relationship between this signal and error correction. As in a previous study (Rodriguez-Fornells et al., 2002), our data showed a larger amplitude of the ERN in fast corrections compared with slow corrections, whereas the latency of this EEG signal was not affected.

Contrary to the results for the ERN amplitude, we did not find differences in the magnitude of the theta power. This discrepancy in our results may be explained by the fact that, although the magnitude of the theta power is similar in both conditions (Figure 3A), a greater amount of the theta activity is phase-locked to the response in the fast RT_{EC} trials. This result agrees with the idea that the ERN component emerges from phase locking of theta band EEG activity (Luu & Tucker, 2001). Although early viewpoints argued that ERPs result from an evoked neural response to an event (Hillyard & Picton, 1987), recent theories suggest that environmental events induce partial "phase resetting" of ongoing neural activity (Makeig, Debener, Onton, & Delorme, 2004; Makeig et al., 2002). Such phase locking has been suggested to correlate with performance (Yamagishi, Callan, Anderson, & Kawato, 2008) and to be affected in neurological conditions such as schizophrenia (Ford, Krystal, & Mathalon, 2007) and Alzheimer's disease (Pijnenburg et al., 2004). Consistent with the importance of such synchronization to behavior, intra-area intertrial phase synchrony (ITC) provided a direct measure of neural synchrony (Makeig et al., 2002) that identified greater phase locking of ongoing theta activity in mPFC for fast versus slow RT_{EC}. Thus, for those trials in which the monitoring activity of the mPFC and the motor response are more synchronized, there is improved error signaling and faster error correction.

Lastly in relation to the ERN results, it is important to note that, supporting previous findings (Pailing et al., 2002), larger ERN amplitudes were preceded by slower RT_E, Pailing and colleagues suggested that the magnitude of ERN monitoring activity may reflect individual differences in response control or impulsive behavior. Following that logic, the authors showed that participants with larger mean ERN amplitudes had slower RT_E, suggesting a less impulsive response strategy by those individuals. In our data, the fact that larger ERN amplitudes across trials, rather than individuals, are preceded by significantly slower RT_E and followed by faster RT_{EC} further suggests that differences in response control vary significantly from trial to trial within individuals.

The Error-related Positivity (Pe) and Performance Decision-making

This initial medial prefrontal signal (ERN) has been hypothesized to relate to the detection of response conflict (Van Veen & Carter, 2002), among other theories of mPFC

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function after errors (i.e., Holroyd & Coles, 2002). If true, greater response conflict should correspond with greater likelihood of an error, and evidence accumulation processes should integrate more rapidly to an error detection threshold when greater response conflict is present (Ullsperger et al., 2010). The error positivity or error-related positivity (Pe) is a parietal electrical signal evoked by errors in comparison with correct responses (Shalgi et al., 2009; Falkenstein et al., 2000). Recent studies (Ullsperger et al., 2010; Ridderinkhof et al., 2009) have argued that this signal is related to the motivational significance of the error in the same way that another well-known parietal ERP, the P3b, is related to the motivational significance of infrequent stimuli (e.g., oddballs). Our results show that the latency of the Pe varies with the RT_{EC}, possibly reflecting the accumulation of evidence for the error that is needed to make a correction. This theory would state that, at the time of the Pe, different sources of information signaling the error contribute to integration in parietal areas until the evidence becomes strong enough to make a decision about the performance (Ullsperger et al., 2010). These results support previous data that found a modulation of the Pe amplitude when the level of the decision criterion was varied (Orr & Carrasco, 2011; Steinhauser & Yeung, 2010).

Interestingly, this link, via the RT_{EC}, between earlier frontal and later parietal signals agrees with previous EEG studies (Philiastides, Ratcliff, & Sajda, 2006; Philiastides & Sajda, 2006) of perceptual decision-making that detected a frontal ERP component occurring approximately 220 msec after the onset of the stimulus (possibly reflecting the typical N2 ERP). This component varied with the level of the perceptual difficulty and predicted the onset of a later component related to evidence accumulation (which could reflect the CPP or typical P3b ERP). Together, these findings suggest that future work might further explore interactions between earlier frontal and later parietal signals as a general mechanism in both the processing of errors (ERN and Pe) and perceptual stimuli (N2 and P3b; Wessel, 2012).

More broadly, these data agree with other EEG work supportive of a centroparietal evidence accumulation mechanism. Recent EEG studies (Kelly & O'Connell, 2013; O'Connell et al., 2012), for example, have characterized a brain electrical signal that shares properties described by models of this process in primates and humans (Ratcliff & McKoon, 2008). Interestingly, this signal, called the centroparietal positivity, shares characteristics with the P3b signal (O'Connell et al., 2012). Although early studies touched on decision-making in relation to the P3b (Smith, Donchin, Cohen, & Starr, 1970), the most influential theory, the context-updating model (Donchin, 1981), states that the P3b is related to a memory update process. According to this theory, the brain constantly generates hypotheses about the environment, with the P3b signal generated every time that the environment changes and the context representation needs to be updated. However, newer studies (Kelly & O'Connell, 2013; O'Connell et al., 2012; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001) suggest a reevaluation of previous P3b results to consider this slow EEG potential as a dynamically evolving component rather than the culmination of a unitary event (Kelly & O'Connell, 2013).

Post-error Adjustments: Alpha Power and Post-error Slowing

Once conflict is detected and evidence for an error has reached threshold, not only must a correction be made, but behavioral modifications must also be implemented to avoid the occurrence of further errors in the future. In EEG studies, suppression of the ITI alpha power, for example, is used to measure an increase in cortical arousal (Compton, Bissey, & Worby-Selim, 2014; Compton, Hofheimer, Kazinka, Levinson, & Zheutlin, 2013; van Driel, Ridderinkhof, & Cohen, 2012; Compton et al., 2011; Carp & Compton, 2009) that may be interpreted as cognitive adjustments occurring after an error is made (Navarro-Cebrian et al., 2013). An increase in alpha power, mainly in posterior areas, often occurs when participants finish a trial and wait for the next one to start. This alpha in the ITI is considered a sign of task disengagement, and its increase is suppressed when one's behavior deviates from a goal. In other words, greater alpha suppression after errors suggests higher alertness. In this study, those trials accompanied by longer RT_{EC} were associated with less alpha power, suggestive of higher alertness following these more uncertain errors. Such findings are consistent with the reduced confidence associated with trials in which evidence accumulates more slowly (Kiani & Shadlen, 2009).

In accordance with these results, participants also slowed down after more uncertain errors (i.e., errors that took longer to correct). Post-error slowing is often defined as longer RTs in trials following errors in comparison with correct responses (Rabbitt, 1966). In our study, the fact that RT_{EC} values covaried with activity of mPFC and parietal cortex, as well as the degree of post-error slowing, suggests that signaling in the frontal and parietal cortices may influence these later processes. Interestingly, not all studies have found the same relationship between the ERN and the degree of post-error slowing. In contrast with our finding of an inverse relationship (i.e., faster RT_{EC} is associated with both a greater ERN and lesser post-error slowing), Gehring and colleagues found a greater ERN to be followed by greater post-error slowing (Gehring et al., 1993). One possibility for this discrepancy relates to the nature of the ERN itself. As shown previously, ERN amplitude is increased by perceptually difficult stimuli (Philiastides et al., 2006), possibly reflecting uncertainty at the stimulus phase (Navarro-Cebrian et al., 2013); in contrast, internal fluctuations in monitoring areas at the time of the response may result in a smaller ERN. As a result, including trials following errors related to stimulus uncertainty rather than motor failures might predispose to finding a direct relationship.

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A second possibility is that the nature of the relationship between ERN amplitude and post-error slowing may be driven by a more proximate signal—that is, the latency of the Pe. A third possibility has to do with our task design: because the ITI was relatively brief (550 msec), slower error correction may simply have impacted participants' abilities to attend to the next stimulus. We note that even the slowest of error correction responses had been completed by 300 msec into the ITI and that subsequent alpha power also distinguished these trials. Nonetheless, future work to explicitly vary the duration of the ITI would be helpful.

Timing Diagram for Adaptive Decision-making

Taken together, our data link together several findings from previous studies (Murphy et al., 2012; Steinhauser & Yeung, 2012; Wessel et al., 2011; Steinhauser & Yeung, 2010; Ullsperger et al., 2010; Pailing et al., 2002; Rodriguez-Fornells et al., 2002) under a potential evidence accumulation framework and provide a hypothetical timing diagram for adaptive decision-making after errors (Figure 5). Specifically, more cognitive control at the time of the selection of the response (slower RT_E; Pailing et al., 2002) may lead to a more accurate comparison between the representation of the correct response and the efference copy of the (incorrect) motor commands. As a consequence, a greater mismatch and a larger ERN will be observed in those trials (Pailing et al., 2002). Therefore, stronger error signaling in these responses will lead to more rapid accumulation of evidence for the erroneous response and a faster RT_{EC}.

It is important to note that the RT_{EC} may occur quickly (average for fast RT_{EC} condition: 126 msec) following the initial erroneous response. These fast corrections, replicating previous studies (e.g., Rodriguez-Fornells et al., 2002), may represent accumulation of evidence for the error if they occur after the comparison of an efference copy of the erroneous motor commands with the correct commands (Figure 5; Christensen et al., 2008; Rodriguez-Fornells et al., 2002; Coles, Scheffers, & Holroyd, 2001; Higgins & Angel, 1970). Moreover, if this comparison between representations of correct and error responses is triggered by the arrival of the efference copy (Coles et al., 2001), it may occur even before the actual motor response (error) takes place. This last possibility may help us to understand both the early latency of the ERN observed in this and previous studies (i.e., Maier & Steinhauser, 2013; Navarro-Cebrian et al., 2013; Wiersema, Van Der Meere, & Roeyers, 2007) and the fast error-correcting responses (RT_{EC}).

An alternative account would argue that the processes necessary for conscious error-signaling responses and subsequent corrections require more than 150 msec (Rabbitt, 2002) and that this type of fast correction may instead represent "delayed correct responses" that are

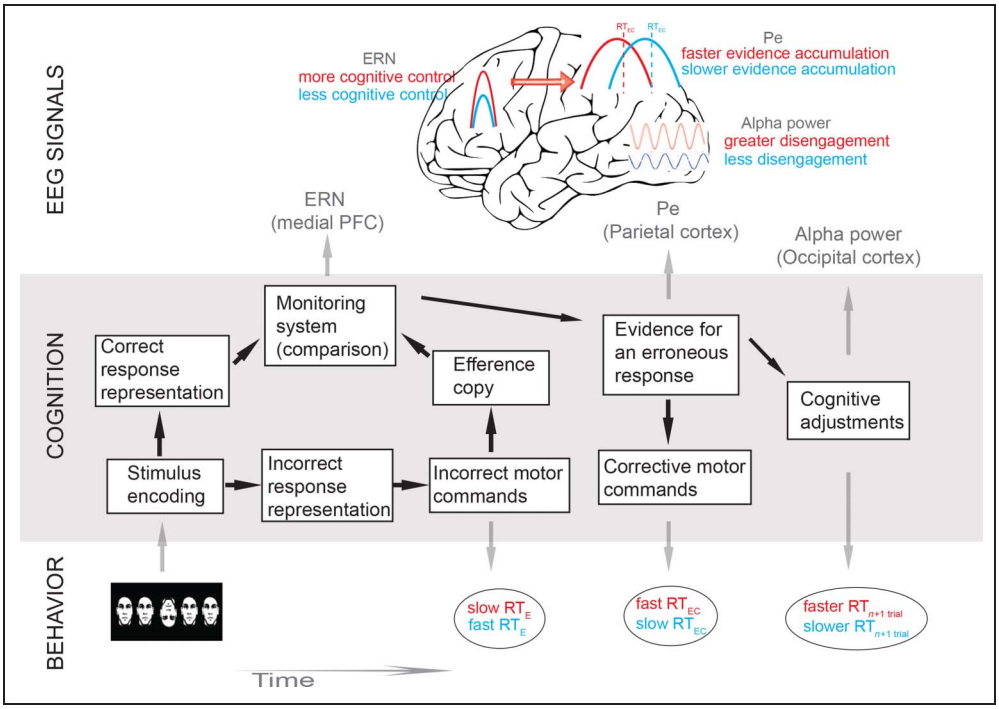

Figure 5. Timing diagram for adaptive decision-making. This diagram represents the different cognitive processes (boxes), behavioral signatures (ellipses), and EEG signals that derive from error processing, error correction, and post-error adjustments. We hypothesize that after the incorrect motor commands are selected, an efference copy of these commands is generated and compared with the predicted correct response representation. An ERN signal will be elicited when a mismatch is produced, and a greater ERN will lead to more rapid corrections (Rodriguez-Fornells et al., 2002). In addition, the ERN signal will correlate with the speed with which evidence for an error accumulates (Ullsperger et al., 2010) so that a greater ERN signal will lead to a faster evidence accumulation process, as reflected in the latency of the Pe signal (Murphy et al., 2012). Lastly, these differences in cognitive control and decision-making after the initial error will influence post-error cognitive and behavioral adjustments. When participants more easily identify their errors (more cognitive control) and correct them quickly, they are better able to disengage during the ITI (more alpha power), and these errors will have a reduced influence on the next trial (reduced post-error slowing).

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completed after impulsive errors (Rabbitt, 2002; Maylor & Rabbitt, 1989). Following this idea, our rapid RT_{EC} values may be due to the continuation of the accumulation of the stimulus information without the explicit detection of the initial error; they would therefore not reflect accumulation of evidence for having made an error, and the Pe in the present example would simply be the continuation of the P3b in relation to the processing of the stimulus. This account would be supported by the fact that our task reflects error corrections rather than error signaling (Ullsperger & von Cramon, 2006). Our data suggest, however, that this possibility may not explain the early latency found for the ERN, which in our and other work (i.e., Maier & Steinhauser, 2013; Navarro-Cebrian et al., 2013; Wiersema et al., 2007) can precede the motor response. An additional complication for this account may come from the results of the RT_{EC} relative to the onset of the stimulus ($RT_{EC\text{-}SO}$). We continue to observe significant differences between the slow and fast $RT_{EC\text{-}SO}$ conditions when those RTs are calculated relative to the onset of the stimulus. One might expect no differences between those two $RT_{EC\text{-}SO}$ values if evidence accumulation is related to the processing of the stimulus alone (i.e., irrespective of the erroneous response) in both cases.

Similarly, the argument might be made that fast and slow errors represent qualitatively distinct cognitive processes—specifically, that unlike long values, fast RT_{EC} values reflect a motor plan that has previously been initiated and that is ultimately implemented without awareness of the error. In our previous work using this paradigm (Navarro-Cebrian et al., 2013), we demonstrated that correct and unaware errors are characterized by small ERN amplitudes that differ significantly from the large ERN amplitudes accompanying aware errors. If rapid RT_{EC} values reflected a lack of error awareness, we would expect that ERN amplitudes would be significantly smaller for fast RT_{EC} trials than for slow RT_{EC} trials. However, we find the opposite result, arguing that error awareness cannot explain the difference between these trial types in this data set.

Conclusions

The current data demonstrate how variation in error salience, as indexed by the RT_{EC}, implicates areas previously associated with evidence accumulation in both the evaluation of decisions and the implementation of compensations when those decisions deviate from goals. Importantly, the study of error-related EEG signals within a decision-making context allows us to describe a potential timing diagram for adaptive decision-making when errors are made. Moreover, mechanisms of error correction, beyond the initial detection of an error, may be relevant to therapies that address behavioral change in patient populations. Future work might therefore investigate how this sequence of events differs when the ability to monitor and then alter behavior is impaired.

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