

Subthreshold error corrections predict adaptive post-error compensations

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Abstract

Relatively little is known about the relation between subthreshold error corrections and post-error behavioral compensations. The present study utilized lateralized beta power, which has been shown to index response preparation, to examine subthreshold error corrections in a task known to produce response conflict, the Simon task. We found that even when an overt correction is not made, greater activation of the corrective response, indexed by beta suppression ipsilateral to the initial responding hand, predicted post-error speeding, and enhanced post-error accuracy at the single-trial level. This provides support for the notion that response conflict associated with errors can be adaptive, and suggests that subthreshold corrections should be taken into account to fully understand error-monitoring processes. Furthermore, we expand on previous findings that demonstrate that post-error slowing and post-error accuracy can be dissociated, as well as findings that suggest that frontal midline theta oscillations and the error-related negativity (ERN) are dissociable neurocognitive processes.

KEYWORDS

action-monitoring, conflict, EEG, ERN, errors, lateralized beta suppression, theta power, time-frequency

1 | INTRODUCTION

During difficult decision-making tasks, a resource intensive process is engaged that enables active monitoring of ongoing task performance. Following an erroneous response, a neural processing cascade is initiated that enables detection of and compensation for the error (Wessel, 2017; Wessel & Aron, 2017). When investigating the relation between error processing and post-error behavior, researchers often observe increases in post-error response time (post-error slowing; PES). Although there is considerable evidence suggesting that PES reflects increased caution, resulting in improved performance on the subsequent trial (Beatty et al., 2020; Botvinick et al., 2001; King et al., 2010; Maier et al., 2011; Marco-Pallarés et al., 2008; Steinhauser & Andersen, 2019), there is

also evidence suggesting that PES reflects distraction, resulting in a decline in performance on the subsequent trial (Beatty et al., 2018; Buzzell et al., 2017; Jentsch & Dudschig, 2009; Notebaert et al., 2009; Steinhauser et al., 2018; Ullsperger & Danielmeier, 2016; Van der Borgh et al., 2016). These disparate findings for adaptive and maladaptive PES can partially be explained by the duration of the response-stimulus interval (Danielmeier & Ullsperger, 2011), as well as differences in the method chosen to compute PES (Schroder et al., 2020).

Although the majority of studies investigating post-error compensations compare the magnitude of error processing with changes in post-error response time and accuracy, there are also some studies that investigate immediate error corrections. However, given that it is difficult to measure subthreshold error corrections, there is a paucity of studies that report

or interpret them. Of the psychophysiological studies that have examined error corrections, the majority focused on determining whether corrections occur due to differences in ongoing motor conflict (Fiehler et al., 2005; Rodríguez-Fornells et al., 2002; Ullsperger & von Cramon, 2006; however, see Burle et al., 2008) or differences in error salience (Kalfaoğlu et al., 2018; Kieffaber et al., 2016; Navarro-Cebrian et al., 2016) across trials. While these studies have contributed to our understanding as to why corrections occur, only a handful of studies have investigated how making a correction—or the propensity to make a correction—impacts subsequent trial behavior such as PES (Crump & Logan, 2013; Fiehler et al., 2005; Kalfaoğlu et al., 2018; Navarro-Cebrian et al., 2016). Although the interpretation of PES remains a subject of debate (Danielmeier & Ullsperger, 2011; Schroder et al., 2020), there is a general consensus among error correction studies that individuals tend to exhibit slowing following uncorrected errors (Crump & Logan, 2013; Fiehler et al., 2005; Kalfaoğlu et al., 2018; Navarro-Cebrian et al., 2016). However, there is evidence for both slowing (Fiehler et al., 2005; Kalfaoğlu et al., 2018; Navarro-Cebrian et al., 2016) and speeding (Crump & Logan, 2013) following corrected errors. Based on these findings, one proposal is that PES on an uncorrected error trial occurs due to the inhibition of the error correction process itself (Crump & Logan, 2013; Kalfaoğlu et al., 2018). Moving beyond studies that investigate response accuracy and error correction as binary processes, other work has assessed subthreshold error tendencies that take place prior to overt responses on correct trials (partial errors) (Allain et al., 2009) as a way to investigate online error corrections that occur prior to making overt responses. For example, a few studies (Burle et al., 2008; Cohen & van Gaal, 2014; Ficarella et al., 2019; Meckler et al., 2017; Roger et al., 2014) have utilized electromyography (EMG) recordings to investigate how subjects detect and correct for subthreshold error responses prior to initiating an overt response. Among these studies, three of them investigated the role of partial errors when investigating post-error behavior, in which they either observed (Allain et al., 2009; Ficarella et al., 2019) or failed to observe (Cohen & van Gaal, 2014) post-partial error slowing relative to pure correct trials. Collectively, the studies regarding error correction and partial errors suggest that the relation between error corrections and post-error compensations needs to be further investigated.

Given that only a handful of studies have investigated error corrections, there is not a unifying theory as to how they fit into existing theoretical frameworks describing error processing and post-error behavior (Danielmeier & Ullsperger, 2011; Wessel, 2017). However, an interpretation of response corrections (Fiehler et al., 2005; Rodríguez-Fornells et al., 2002; Ullsperger & von Cramon, 2006), which is consistent with the fundamental assumptions outlined in conflict monitoring

theory (Botvinick et al., 2001; Yeung et al., 2004), suggests that corrections occur when a competing motor program is simultaneously activated and exceeds the motor threshold required to elicit a response. For instance, following stimulus presentation, the response selection process is initiated, prompting for a degree of motor activation to build for each response option. Once the motor threshold for a trial has been reached, a response will be made. Specifically, during an error trial that is corrected, instead of the subthreshold correction immediately returning to baseline, its magnitude is sustained and eventually elicits a correction.

A recent study by Steinhauser and Andersen (2019) suggests that immediate corrective activity might occur more commonly following error commissions than is typically assumed. Utilizing a global motion paradigm with steady-state visual evoked potentials (SSVEP), these authors determined that up until the point of error commission, attention was focused on the incorrect stimuli. However, following the error, there was a rapid shift in attention toward the correct stimuli, as well as improved performance on the subsequent trial. Therefore, this shift in attention was interpreted as an immediate, adaptive post-error adjustment that occurs in the brain even though an overt corrective button press was not performed. This phenomenon is important to consider when investigating post-error compensations. While many studies have investigated how the magnitude of error processing influences post-error compensations such as PES, they omit a critical step—the tendency to salvage the ongoing trial (see Figure 1). For example, while some studies explicitly investigate error corrections by experimentally modulating their likelihood (Crump & Logan, 2013; Fiehler et al., 2004, 2005; Steinhauser, 2010; Ullsperger & von Cramon, 2006), other studies conclude that error corrections are potential confounds to the research question being proposed, and therefore, contend with them by either forbidding them altogether (Amengual et al., 2013; Buzzell et al., 2017; de Visser et al., 2018; van Meel et al., 2007) or removing them post hoc (Beatty et al., 2018, 2020). Thus, long-standing questions regarding the functional significance of error monitoring might be clarified by investigating corrections that occur between trial N and trial $N + 1$. Empirical research has shown that (1) error corrections modulate post-error behavior and (2) subthreshold corrective activity occurs in the brain despite the absence of an overt correction. We propose that, similar to investigations of partial errors on correct trials, it would be informative to investigate subthreshold corrective activity (that occurs after the response) on uncorrected error trials.

Although researchers have found that event-related potentials such as the error-related negativity (ERN), the correct response negativity (CRN), and the error positivity (Pe) could index error corrections, findings have been inconsistent (ERN: Crump & Logan, 2013; Fiehler et al., 2004; Meckler et al., 2017; Navarro-Cebrian et al., 2016; Rodríguez-Fornells

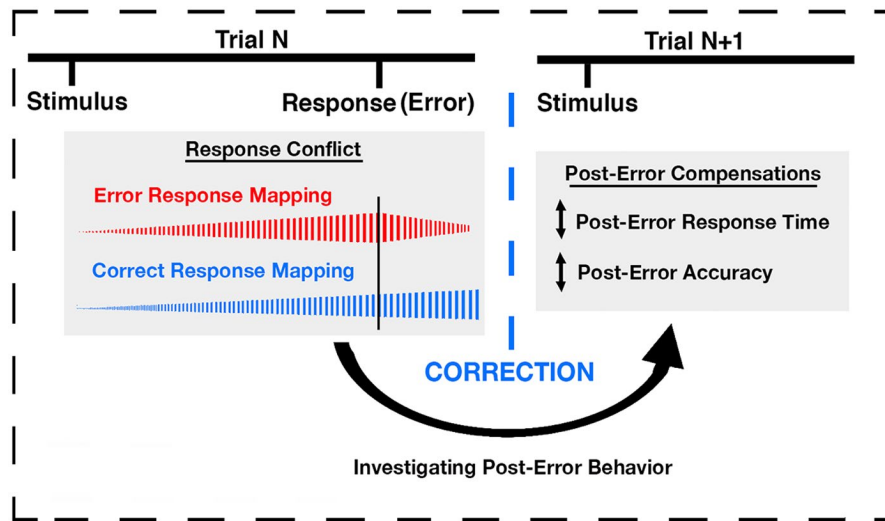


FIGURE 1 Conceptual model of immediate error corrections. Following stimulus presentation, the response selection process is initiated, prompting for a degree of motor activation to build for each response option (i.e., red and blue oscillations). Once the motor threshold for that trial has been reached, the response will be made. In this example, the error response reached threshold prior to the correct response. We propose that during an error trial in which a response correction occurs, instead of the subthreshold error correction immediately returning to baseline, its magnitude is sustained and eventually elicits a correction. While many studies have investigated how the magnitude of error processing influences post-error compensations (i.e., black arrow), they omit a critical step, the tendency to salvage the ongoing trial (i.e., correct for the error). Thus, long-standing questions regarding the functional significance of error monitoring might be clarified by investigating response corrections that occur *between* trial N and trial $N + 1$

et al., 2002; Ullsperger & von Cramon, 2006; Pe: Ficarella et al., 2019; Kieffaber et al., 2016) suggesting that these components may not be a reliable index of subthreshold error corrections. Recently, two studies (Beatty et al., 2020; Valadez & Simons, 2017) have suggested that induced, transient, rhythmic cortical activity, is more closely linked to post-error compensations than evoked cortical activity such as event-related potentials (ERPs). More specifically, while the ERN was predictive of post-error slowing, frontal midline theta power (4–7 Hz) predicted both increases in post-error slowing and post-error accuracy (Beatty et al., 2020; Valadez & Simons, 2017). We propose that, similar to the findings for post-error adjustments that occur from one trial to the next, it is possible that induced rhythmic oscillatory activity would be able to predict subthreshold error corrections. For instance, researchers have observed increases in theta power (Kieffaber et al., 2016) as well as shifts in peak latency of theta power (Navarro-Cebrian et al., 2016) on corrected error trials. We note however that analyses for theta power predicting post-error behavior were not performed during these studies. Frontal midline theta power is believed to arise, at least in part, from the medial frontal cortex and serves to recruit dorsolateral prefrontal cortex in response to increased task demands (Cavanagh & Frank, 2014). However, while theta has been demonstrated to be *modulated* by error corrections, we suggest that it is important to further assess motor activation itself (via beta oscillatory activity) to further understand the cascade of processing that links responses on one trial and behavior on the subsequent trial.

Beta oscillations (13–25 Hz) can be used as a direct neural index of motor activation (Neuper et al., 2006). For instance, researchers have observed beta power suppression when participants were planning (Tzagarakis et al., 2010) or imagining to perform (Pfurtscheller et al., 1997) a motor response, which provides compelling evidence that even in the absence of an explicit motor response, it is possible to detect the intention to perform an action. One study in particular (Fischer et al., 2018) investigated how beta power lateralization (BPL) can index the response preparation process. BPL is the difference in the magnitude of beta power suppression between the two hemispheres, which is calculated by subtracting activity contralateral to the response from activity ipsilateral to the response. Fischer et al. (2018) observed greater suppression in beta power contralateral to the response hand. In the present study, we used lateralized beta suppression to detect motor activation associated with subthreshold error corrections. Critically, the role for beta suppression as an index of subthreshold error corrections can be verified given that the association between beta and an explicit correction can be evaluated. During the initial response, we would expect to observe greater beta suppression over electrode sites contralateral to the responding hand. However, if the response was incorrect, a response correction could be observed as relatively greater beta suppression for the response that was not initially selected (ipsilateral to the initial incorrect response).

The current study sought to determine whether subthreshold error corrections, as indexed by BPL, are associated with adaptive or maladaptive post-error behavior (PES and PEA).

However, conflicting evidence for the role of subthreshold error corrections on post-error behavior do not favor a particular prediction. When using lateralized beta suppression as an index of subthreshold corrections (on uncorrected error trials) to predict post-error behavior at the single-trial level, one possibility is that greater activation for subthreshold corrections might increase motor conflict. If this were indeed the case, such a finding would be in line with the prediction of conflict monitoring theory (Botvinick et al., 2001; Yeung et al., 2004). In response to an increase in motor conflict, participants might slow down on the subsequent trial due to either increased caution (increased PES; increased PEA) or distraction—the error would interrupt the task set (increased PES; decreased PEA). Alternatively, some authors have suggested that PES only occurs when participants are required to suppress an implicit urge to correct their responses, and that when allowed to make spontaneous corrections, it is possible to observe not PES, but post-error speeding instead (Crump & Logan, 2013; Kalfaoğlu et al., 2018). Thus, it is possible that more subtle variation for subthreshold error corrections (indexed via BPL) may be associated with either the absence of PES or post-error speeding, as well as greater PEA.

2 | METHOD

2.1 | Participants

Fifty-three healthy, young adults (Mean Age = 20.151; $SD = 2.214$; 35 female) participated in exchange for course credit at George Mason University. Three participants were excluded due to either inadequate ($< 70\%$ accuracy) task performance (two participants) or not following task instruction (one participant), leaving 50 participants (Mean Age = 20.040; SD Age = 2.109; 33 female) to be incorporated

into subsequent analyses. All participants were right-handed and had normal or corrected-to-normal vision. The George Mason University Office of Integrity and Assurance approved all research procedures.

2.2 | Experiment design

Participants completed a color Simon task (Figure 2), which was presented using Psychtoolbox (Brainard, 1997; Kleiner et al., 2007; Pelli, 1997) as well as custom scripts and functions in MATLAB (MathWorks, Natick, MA). On a standard LCD monitor, participants were presented with a light gray, central fixation cross on a darker gray background along with two light gray boxes, each subtending 3.75 by 3.75 degrees, located 4.25 degrees to the left and right of the center of the screen. A red (sRGB: $r = 105.85$, $g = 0$, $b = 0$) or green (sRGB: $r = 0$, $g = 53.05$, $b = 0$) colored circle, subtending 2 degrees, was presented for 200 ms in the center of one of the two boxes. All stimuli were equiprobable and perceived luminance of the stimuli was equated using the sRGB gamma function.

Participants were instructed to weigh the speed and accuracy of their responses equally and to indicate which color was presented by pressing either the “2” key (using their left index finger) or the “8” key (using their right index finger). These response mappings were counterbalanced across participants. If the participant responded within 150 ms of stimulus onset, responded after the 500 ms response deadline, or did not provide a response, the trial (as well as the following trial) was removed from all analyses. Each response was followed by a response-stimulus interval drawn from a random uniform distribution between 750 ms and $1,000$ ms in duration. Response corrections were recorded from the time of the initial response until the beginning of the next trial. In

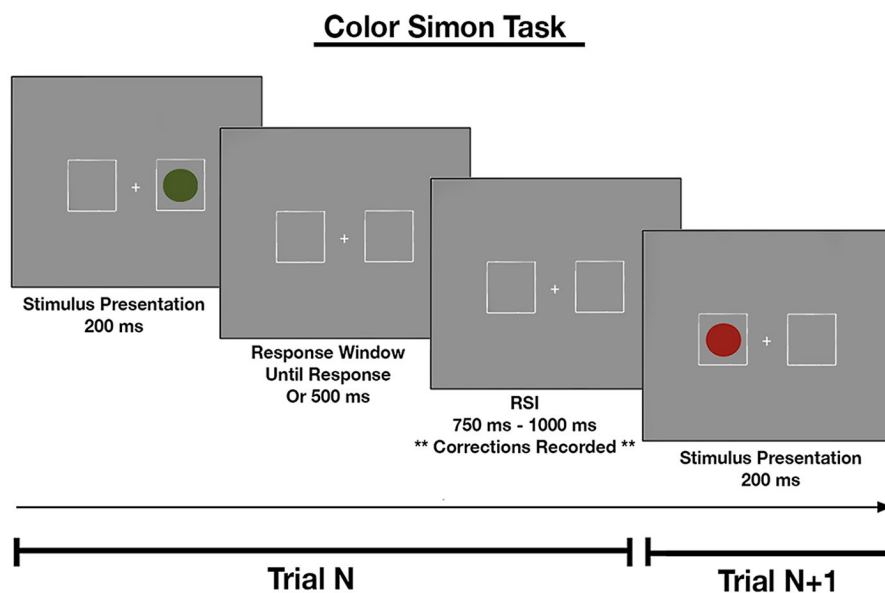


FIGURE 2 Task design. Participants completed a Color Simon Task. Stimulus contrasts have been increased for presentation purposes

order to update participants on their overall task performance, and in order to combat fatigue, participants were informed of their block accuracy in-between blocks and were required to take self-paced breaks for a minimum of 30 s prior to starting the next block. The main experiment consisted of 3,200 trials (20 blocks of 160 trials). To familiarize themselves with the task, participants completed a practice block, in which they were provided with trial-by-trial feedback of their performance. Critically, no trial-level accuracy feedback was presented during the actual experiment.

2.3 | EEG data acquisition and processing

EEG data were collected using a Brain Vision Brain Amp amplifier and Brain Vision Recorder 1.2 acquisition software (Brain Products Inc.). Data were recorded using 64 actiCAP electrodes (positioned according to the extended 10–20 system), which included two in-cap electrodes corresponding to the left and right mastoids (TP9 and TP10). An additional in-cap ground electrode was positioned anterior to electrode Fz. Although electrode Cz was used as the online reference for the other 63 in-cap electrodes, the data for electrode Cz were recovered offline through re-referencing. Offline, the data were re-referenced to the average of the left and right mastoids (electrodes TP9 and TP10). The data were recorded using a 500 Hz sampling rate, with an online band-pass filter of .1–250 Hz. In concordance with impedance recommendations for high-impedance active EEG systems, impedance for all electrodes was maintained below 25 k Ω .

The continuous EEG data were filtered offline using a 30 Hz low-pass filter and subjected to independent components analysis (ICA) using Brain Vision Analyzer software (Brain Products Inc.) to identify and reject components corresponding to blinks and saccades. Data were then exported to EEGLAB (Delorme & Makeig, 2004), a toolbox for MATLAB, for all subsequent processing steps. The data were epoched from –1,000 ms to 1,500 ms for all stimulus and response markers. To remove residual artifacts following rejection of ocular IC components, the data were subjected to an automated rejection procedure using a voltage rejection threshold of ± 100 μ V and a spectral rejection threshold of 50 dB within the 20–40 Hz band using the `pop_rejspec` function. If more than 20% of epochs were marked for rejection for a given electrode, that electrode was removed from the data set. In order to ensure that the trial N stimulus and trial N response epochs were matched, if either epoch was rejected during artifact rejection, the corresponding epoch was also rejected. All rejected electrodes were then interpolated (using a spherical spline interpolation). If the maximum number of electrodes that were interpolated for a participant was more than 10% of all electrodes, that participant was removed from all analyses. To better estimate cortical sources and reduce

the impact of volume conduction, we conducted a Laplacian (current source density; CSD) transformation of the data (Kayser & Tenke, 2006).

When conducting time-frequency analyses, we conducted a technique pioneered by Cohen and Donner (2013), which is believed to remove the ERP from the continuous EEG signal. Empirically, it has been demonstrated that induced oscillatory theta power acts as a robust neural index of behavior that indexes unique characteristics of the data when compared with evoked signals (Beatty et al., 2020). For a comprehensive review of this technique, please see Cohen and Donner (2013) and Beatty et al., (2020). Briefly, the averaged ERPs were subtracted from each individual epoch prior to computing the time-frequency spectrum, thus, theoretically removing the phase-locked component of the EEG signal. This subtraction was performed in the time domain (using the full –1,000 ms to 1,500 ms epoch) and was completed separately at each electrode for each condition of interest. Following subtraction of the ERP, the data were convolved with a series of complex Morlet wavelets at the single-trial level. The frequency of each wavelet increased from 2 to 30 Hz in 20 linearly spaced steps, while the number of cycles increased from 3 to 6 in 20 logarithmically spaced steps. Power values at each frequency were separately baseline normalized using condition-specific baseline relative to the average power from –400 to –100 relative to stimulus onset ($10 * \log_{10}$ (trial N response power/trial N stimulus baseline period)).

All ERP analyses were conducted using the same trials that were used in the time-frequency analyses. In order to conform the epoch range to be more suitable for analyzing ERPs, the trial N stimulus epochs were re-epoched from –200 ms to 800 ms, and the trial N response epochs were re-epoched from –400 ms to 800 ms. Similar in concept to what was done for time-frequency analyses, baseline correction for response-locked epochs was implemented using the prestimulus period (–200 ms to 0 ms) from the trial-matched stimulus-locked epoch.

2.4 | Averaged-level subset analyses for fully executed error corrections

In order to investigate error corrections, statistical analyses for averaged-level behavior and electrophysiology were conducted on a subset of 19 participants (mean age = 21.158, $SD = 2.693$, 14 female). The analyses based on the subset of participants with enough error corrections are complementary or supplementary to the primary analyses that utilize 50 participants to test hypotheses regarding single-trial relations. Participants were selected based on their task performance (if they completed at least 20 fully executed error corrections per congruency condition) as well as to optimize both the number of trials and the size of the sample incorporated into

the analyses (Boudewyn et al., 2018). Although the minimum number of trials to obtain a sufficient signal to noise ratio for ERP components such as the ERN has been suggested to be as few as 4–6 trials (Steele et al., 2016) or 6–8 trials (Oivet & Hajcak, 2009), a more recent analysis based on a large sample (863 participants) suggests that a reliable ERN would require at least 16 trials for a within-subjects design (Fischer et al., 2017). We chose a relatively conservative cutoff of 20 trials. The number of trials for each condition that were included in all subset analyses were the following: correct congruent (mean = 1,224.050, $SD = 122.002$, range = 960–1,407); correct incongruent (mean = 1,033.421, $SD = 160.543$, range = 728–1,284); corrected congruent errors (mean = 46.526, $SD = 33.827$, range = 20–154); corrected incongruent errors (mean = 85.211, $SD = 66.380$, range = 24–253); uncorrected congruent errors (mean = 147.842, $SD = 76.547$, range = 48–299); uncorrected incongruent errors (mean = 282.895, $SD = 131.858$, range = 63–581).

2.4.1 | Behavior

For current trial response time, the analyses were conducted using a 2 (Accuracy: Corrects, Errors) \times 2 (Congruency: Congruent, Incongruent) ANOVA (collapsing across corrected and uncorrected errors), as well as an error-only 2 (Congruency: Congruent, Incongruent) \times 2 (Correction: Corrected, Uncorrected) ANOVA. Analysis of current trial *correction* response times (as a function of trial congruency) was conducted using a repeated samples *t* test. For all analyses, post-error slowing was computed by subtracting current trial error RTs from next trial (post-error) RTs (Schroder et al., 2020). Post-error accuracy during averaged-level subset analyses was computed as a percentage change between (1) the accuracy for trials following an error and (2) the accuracy for trials following a correct response (Beatty et al., 2018, 2020; Buzzell et al., 2017), while post-error accuracy during single-trial analyses was computed as the accuracy on post-error trials. Analyses for post-error slowing and post-error accuracy were conducted using separate error-only 2 (Congruency: Congruent, Incongruent) \times 2 (Correction: Corrected, Uncorrected) ANOVAs.

2.4.2 | Electrophysiology

Statistical analyses for theta and the ERN took place at electrode Cz (which is where both components were found to be maximal). Statistical analyses for beta took place where it was found to be maximal at separate clusters of electrodes over the left and right side of the scalp: the average of electrodes C3, C5, CP3, CP5 on the left and the average of electrodes C4, C6, CP4, CP6 on the right. Ipsilateral and

contralateral beta power magnitudes were computed by accounting for which hand completed the response relative to which cluster of electrodes was utilized.

Prior to constructing analysis windows to investigate the magnitude of each component, time-series plots were calculated by collapsing across the data originating from wavelets between 4 to 7 Hz for theta and 13 to 25 Hz for beta. In order to determine if there were statistically significant latency shifts for each of these indices as a function of condition, we conducted a peak amplitude latency analysis (see supplementary materials). During this analysis, the latencies for the peak amplitude were computed for each person, for each condition of interest. For theta and the ERN, the analyses were conducted using a 2 (Accuracy: Corrects, Errors) \times 2 (Congruency: Congruent, Incongruent) ANOVA (collapsing across corrected and uncorrected errors), as well as an error-only 2 (Congruency: Congruent, Incongruent) \times 2 (Correction: Corrected, Uncorrected) ANOVA. The analyses investigating latency shifts in beta power were similar to those that were conducted for theta and the ERN with the exception that an additional factor of laterality (Ipsilateral, Contralateral) was included for each ANOVA.

In order to create data driven analysis windows, window-widths were based on the 50% amplitude of each component (Hassall et al., 2019). For theta, the latency analysis determined that there were significant latency shifts between correct trials, corrected error trials, and uncorrected error trials. In order to compensate for these shifts, we computed a 50% amplitude window for each of these conditions, collapsing across congruency (Corrects: –230 ms to 110 ms; Corrected Errors: –162 ms to 238 ms; Uncorrected Errors: –144 ms to 276). For the ERN, since the latency analysis demonstrated that there were not any significant latency shifts as a function of condition, we selected a window based on the 50% amplitude of the ERN collapsing across all conditions (–12 ms to 60 ms). For beta, the latency analysis demonstrated that there were significant latency shifts for beta as a function of correct and error trials (corrected and uncorrected errors were not significantly different in their latency). In order to compensate for these shifts, we computed separate 50% amplitude windows for corrects (collapsing across congruency and laterality) and errors (collapsing across congruency, corrections, and laterality) (Corrects: –234 ms to 238 ms; Errors: –218 ms to 338 ms).

For analyses regarding component magnitude, the windows were created based on the latency analyses. For each component, the analyses were conducted using a 2 (Accuracy: Corrects, Errors) \times 2 (Congruency: Congruent, Incongruent) ANOVA (collapsing across corrected and uncorrected errors), as well as an error-only 2 (Congruency: Congruent, Incongruent) \times 2 (Correction: Corrected, Uncorrected) ANOVA. The analyses investigating beta magnitude were similar to those that were conducted for theta and the ERN

with the exception that an additional factor of laterality (Ipsilateral, Contralateral) was included for each ANOVA. The values being compared were the average voltage (for the ERN) or average power (for theta and ipsilateral/contralateral beta) within their respective analysis windows.

2.5 | Single-trial analyses

Since statistical analyses at the single-trial level were conducted to investigate subthreshold corrections (i.e., ipsilateral beta power) on uncorrected errors, we were permitted to utilize the full sample of 50 participants using the same windows that were constructed in the averaged-level analyses, collapsing across the windows for corrected and uncorrected errors (Theta: -154 ms to 258 ms; ERN: -12 ms to 60 ms; Beta: -218 ms to 338 ms). The number of trials for each condition that were included in all single-trial analyses were the following: uncorrected congruent errors (mean = 125.520 , $SD = 66.730$, range = $31-319$); uncorrected incongruent errors (mean = 232.220 , $SD = 110.066$, range = $63-581$).

A series of mixed-effects models were performed using the R statistical software, version 3.3.1 (R Core Team, 2016), utilizing the lmerTest package, version 2.0–32 (Kuznetsova et al., 2016) and the lme4 package, version 1.1–12 (Bates et al., 2014). Prior to carrying out each analysis, models were constructed such that the continuous variables (ERN, Theta, Ipsilateral/Contralateral Beta, Post-Error Slowing) were centered and scaled to have a mean of 0 and SD of 1. These variables were fit using linear mixed-effects analysis using the lmer function, with restricted maximum likelihood estimation. The categorical variables (Post-Error Accuracy) were examined using sum contrasts and fit using generalized linear mixed-effects models using the glmer function with logit link with maximum likelihood estimation. Variation in intercept was treated as a random effect, while all remaining variables were treated as fixed effects. Statistical significance for each fixed effect was calculated via lmerTest (Kuznetsova et al., 2016), using the Satterthwaite's approximation to denominator degrees of freedom. The most critical single-trial analyses investigated the relation between frontal midline theta power magnitude, ipsilateral and contralateral beta power magnitude, and ERN magnitude, predicting the magnitude of post-error slowing/speeding or post-error accuracy on the subsequent trial. An additional model was created to investigate the relation between lateralized beta power and next trial error RTs. Since we were interested specifically in error processing, we limited what types of trials would be included in the models. When predicting post-error slowing/speeding, we limited the trials that were included in the model to “error trials followed by correct trials.” When predicting post-error accuracy, we limited the trials such that the current trial must be an error.

3 | RESULTS

3.1 | Averaged-level subset analyses for fully executed error corrections

3.1.1 | Behavior

For the subset analyses (which included 19 participants that executed at least 20 corrected errors per congruency condition), overall task accuracy was 80.089% (congruent trials: 86.373%, incongruent trials: 73.738%). When investigating the current trial response time, a 2 (Accuracy: Correct, Error) by 2 (Congruency: Congruent, Incongruent) repeated measures ANOVA, revealed a main effect of accuracy ($F(1,18) = 93.337$, $p < .001$, $\eta^2 = .838$), in which errors ($M = 317.129$; $SE = 4.693$) were faster than corrects ($M = 336.054$; $SE = 4.806$), but failed to reveal a main effect of congruency ($F(1,18) = .044$, $p = .835$, $\eta^2 = .002$). However, there was an accuracy by congruency interaction ($F(1,18) = 186.894$, $p < .001$, $\eta^2 = .912$). Paired samples t tests revealed that congruent trials ($M = 325.485$; $SE = 4.895$) were faster than incongruent trials ($M = 346.623$; $SE = 4.789$) for corrects ($p < .001$, $d = -1.002$), but incongruent trials ($M = 306.841$; $SE = 4.606$) were faster than congruent trials ($M = 327.416$; $SE = 5.122$) for errors ($p < .001$, $d = .954$). In addition to this analysis, limiting the included trials to only errors, an additional 2 (Congruency: Congruent, Incongruent) by 2 (Corrections: Corrected, Uncorrected) repeated measures ANOVA, revealed a main effect of congruency ($F(1,18) = 62.151$, $p < .001$, $\eta^2 = .775$), in which incongruent errors ($M = 306.841$; $SE = 4.606$) were faster than congruent errors ($M = 327.416$; $SE = 5.122$), but failed to reveal a main effect of corrections ($F(1,18) = 1.628$, $p = .218$, $\eta^2 = .083$), or a congruency by corrections interaction ($F(1,18) = 1.432$, $p = .247$, $\eta^2 = .074$).

Among the errors that were committed during the task, 34.060% of errors took place on congruent trials (congruent error correction rate = 24.837%), while 65.940% of errors took place on incongruent trials (incongruent error correction rate = 24.194%). When investigating the current trial correction response time, a paired samples t test revealed a significant effect of congruency ($t(18) = -4.060$, $p = .001$, $d = -.200$), in which congruent corrections ($M = 141.796$; $SD = 28.620$) were faster than incongruent corrections ($M = 149.820$; $SD = 33.969$).

With regard to effects on post-error behavior (Figure 3), a 2 (Congruency: Congruent, Incongruent) by 2 (Corrections: Corrected, Uncorrected) repeated measures ANOVAs was separately conducted for post-error RT and post-error accuracy. Analyses of post-error RT revealed a main effect of congruency ($F(1,18) = 43.221$, $p < .001$, $\eta^2 = .706$), in which post-incongruent errors ($M = 34.921$; $SE = 3.710$) were slower than post-congruent errors ($M = 10.950$;

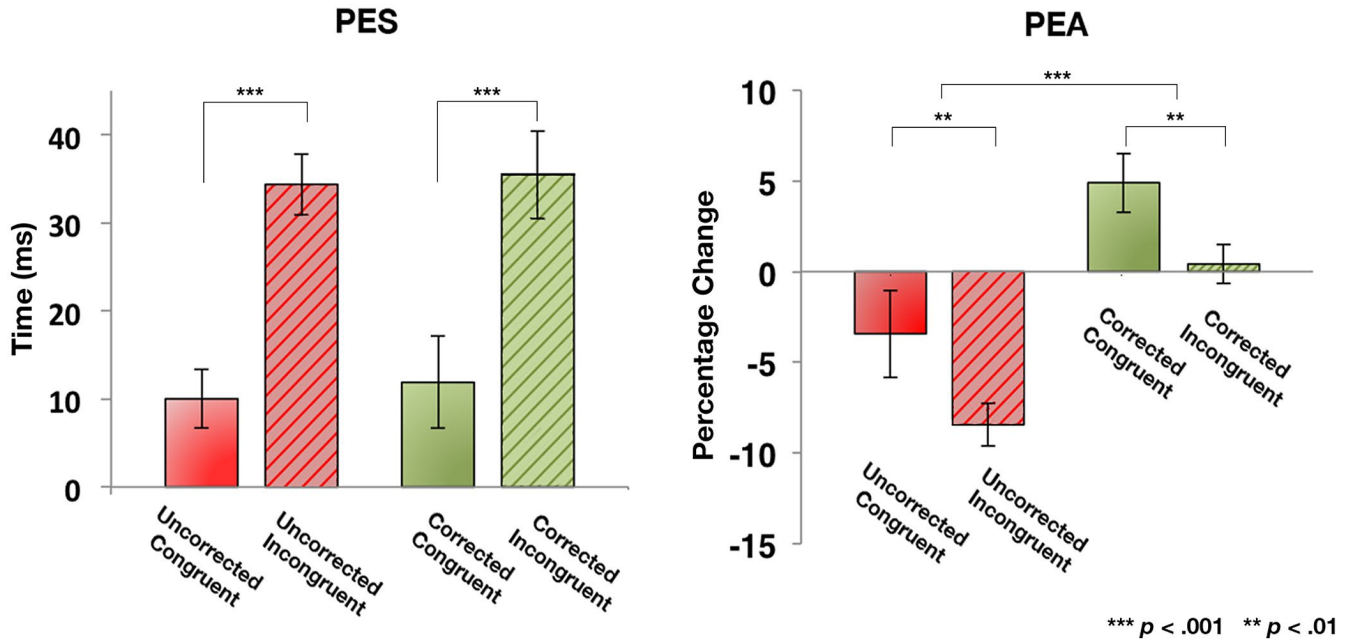


FIGURE 3 Post-error behavior. Displays post-error slowing (PES) and post-error accuracy (PEA) as a function of congruency for both uncorrected and corrected errors. The error bars depict the standard error. This analysis reports data for a subset of participants used to investigate fully executed error corrections

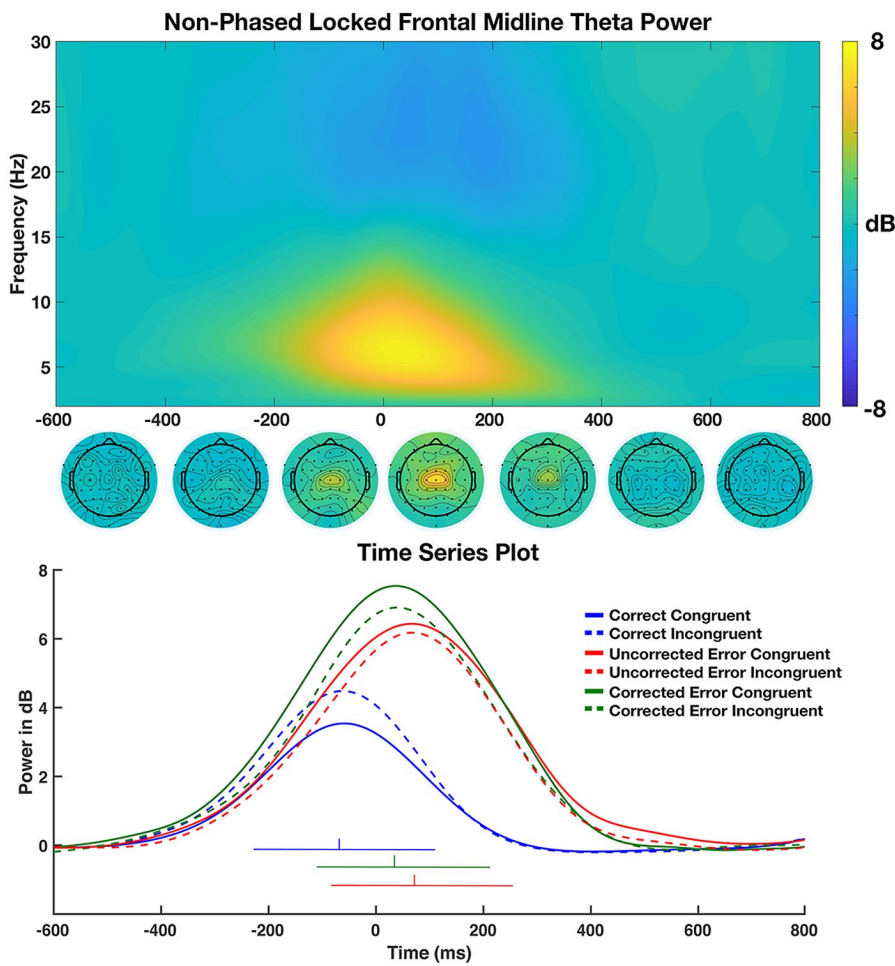


FIGURE 4 Theta power. Displays induced theta power (at electrode Cz) and corresponding topographic time series, which illustrate theta power over time in 200 ms intervals. The time-series plot was constructed by collapsing across wavelets corresponding to frequencies between 4 and 7 Hz. The colored bars represent the respective analysis windows per condition, which are centered at the peak of the waveform (collapsing across congruency). This analysis reports data for a subset of participants used to investigate fully executed error corrections

$SE = 2.984$), but failed to reveal a main effect of corrections ($F(1,18) = .117, p = .736, \eta^2 = .006$), or a congruency by corrections interaction ($F(1,18) = .014, p = .908, \eta^2 = .001$). Analyses of post-error accuracy revealed a main effect of congruency ($F(1,18) = 9.719, p = .006, \eta^2 = .351$), in which post-congruent trials ($M = .739\%$; $SE = 1.681\%$) were more accurate than post-incongruent trials ($M = -4.008\%$; $SE = .825\%$), as well as a main effect of corrections ($F(1,18) = 33.085, p < .001, \eta^2 = .648$), in which corrected errors ($M = 2.664\%$; $SE = 1.076\%$) were more accurate than uncorrected errors ($M = -5.933\%$; $SE = 1.519\%$). There was not a congruency by corrections interaction ($F(1,18) = .040, p = .844, \eta^2 = .002$).

3.1.2 | Electrophysiology

For theta power (Figure 4), a 2 (Accuracy: Correct, Error) by 2 (Congruency: Congruent, Incongruent) repeated measures ANOVA, revealed a main effect of accuracy ($F(1,18) = 168.464, p < .001, \eta^2 = .903$), in which errors ($M = 5.460$; $SE = .346$) were larger than corrects ($M = 3.212$; $SE = .307$), but failed to reveal a main effect of congruency ($F(1,18) = 1.490, p = .238, \eta^2 = .076$). However, there was an accuracy by congruency interaction ($F(1,18) = 70.164, p < .001, \eta^2 = .796$). Paired samples t tests revealed that incongruent trials ($M = 3.580$; $SE = .312$) were larger than congruent trials ($M = 2.844$; $SE = .307$) for corrects ($p < .001, d = -.542$), but congruent trials ($M = 5.740$; $SE = .376$) were larger than incongruent trials ($M = 5.179$; $SE = .327$) for errors ($p < .001, d = .340$). In addition to this analysis, limiting the included trials to only errors, an additional 2 (Congruency: Congruent, Incongruent) by 2 (Corrections: Corrected, Uncorrected) repeated measures ANOVA, revealed a main effect of congruency ($F(1,18) = 19.653, p < .001, \eta^2 = .522$),

in which congruent errors ($M = 5.762$; $SE = .377$) were larger than incongruent errors ($M = 5.191$; $SE = .327$), as well as a main effect of corrections ($F(1,18) = 14.995, p = .001, \eta^2 = .454$), in which corrected errors ($M = 5.894$; $SE = .364$) were larger than uncorrected errors ($M = 5.060$; $SE = .362$). There was not a significant congruency by corrections interaction ($F(1,18) = .983, p = .335, \eta^2 = .052$).

For the ERN (Figure 5), a 2 (Accuracy: Correct, Error) by 2 (Congruency: Congruent, Incongruent) repeated measures ANOVA, revealed a main effect of accuracy ($F(1,18) = 78.263, p < .001, \eta^2 = .813$), in which errors ($M = -16.605$; $SE = 2.468$) were larger (more negative) than corrects ($M = 5.374$; $SE = 2.217$), but failed to reveal a main effect of congruency ($F(1,18) = 1.785, p = .198, \eta^2 = .090$). However, there was an accuracy by congruency interaction ($F(1,18) = 13.705, p = .002, \eta^2 = .432$). Paired samples t tests revealed that congruent trials ($M = 3.945$; $SE = 2.212$) were larger (more negative) than incongruent trials ($M = 6.804$; $SE = 2.250$) for corrects ($p < .001, d = -.291$), but incongruent trials ($M = -19.264$; $SE = 2.810$) were larger (more negative) than congruent trials ($M = -13.946$; $SE = 2.496$) for errors ($p = .015, d = .455$). In addition to this analysis, limiting the included trials to only errors, an additional 2 (Congruency: Congruent, Incongruent) by 2 (Corrections: Corrected, Uncorrected) repeated measures ANOVA, revealed a main effect of congruency ($F(1,18) = 7.271, p = .015, \eta^2 = .288$), in which incongruent errors ($M = -19.264$; $SE = 2.810$) were larger (more negative) than congruent errors ($M = -13.926$; $SE = 2.496$), as well as a main effect of corrections ($F(1,18) = 40.880, p < .001, \eta^2 = .694$), in which corrected errors ($M = -22.857$; $SE = 3.091$) were larger (more negative) than uncorrected errors ($M = -10.353$; $SE = 2.131$). There was not a significant congruency by corrections interaction ($F(1,18) = 3.477, p = .079, \eta^2 = .162$).

For beta power (Figures 6 and 7), a 2 (Laterality: Ipsilateral, Contralateral) by 2 (Accuracy: Correct, Error)

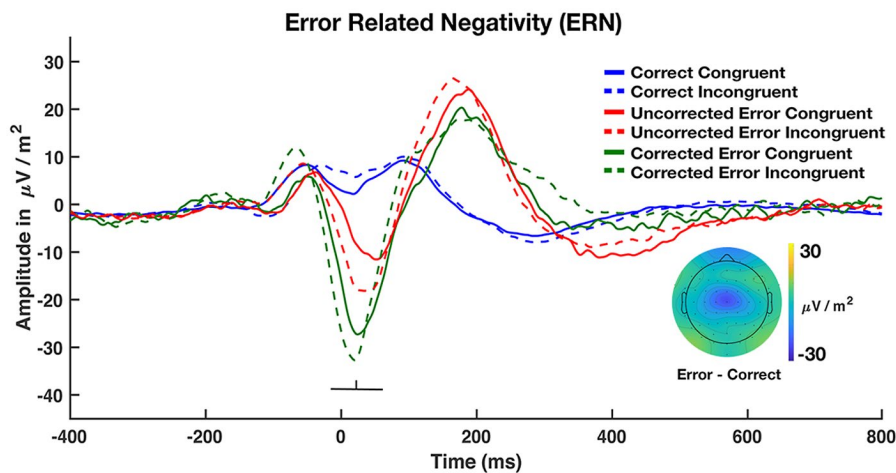


FIGURE 5 Error-related negativity (ERN). Displays the ERN (at electrode Cz) and corresponding difference (error–correct) topographic plot. The black bar represents the analysis window for all conditions, which was centered at the peak of the grand average waveform collapsing across all conditions. This analysis reports data for a subset of participants used to investigate fully executed error corrections

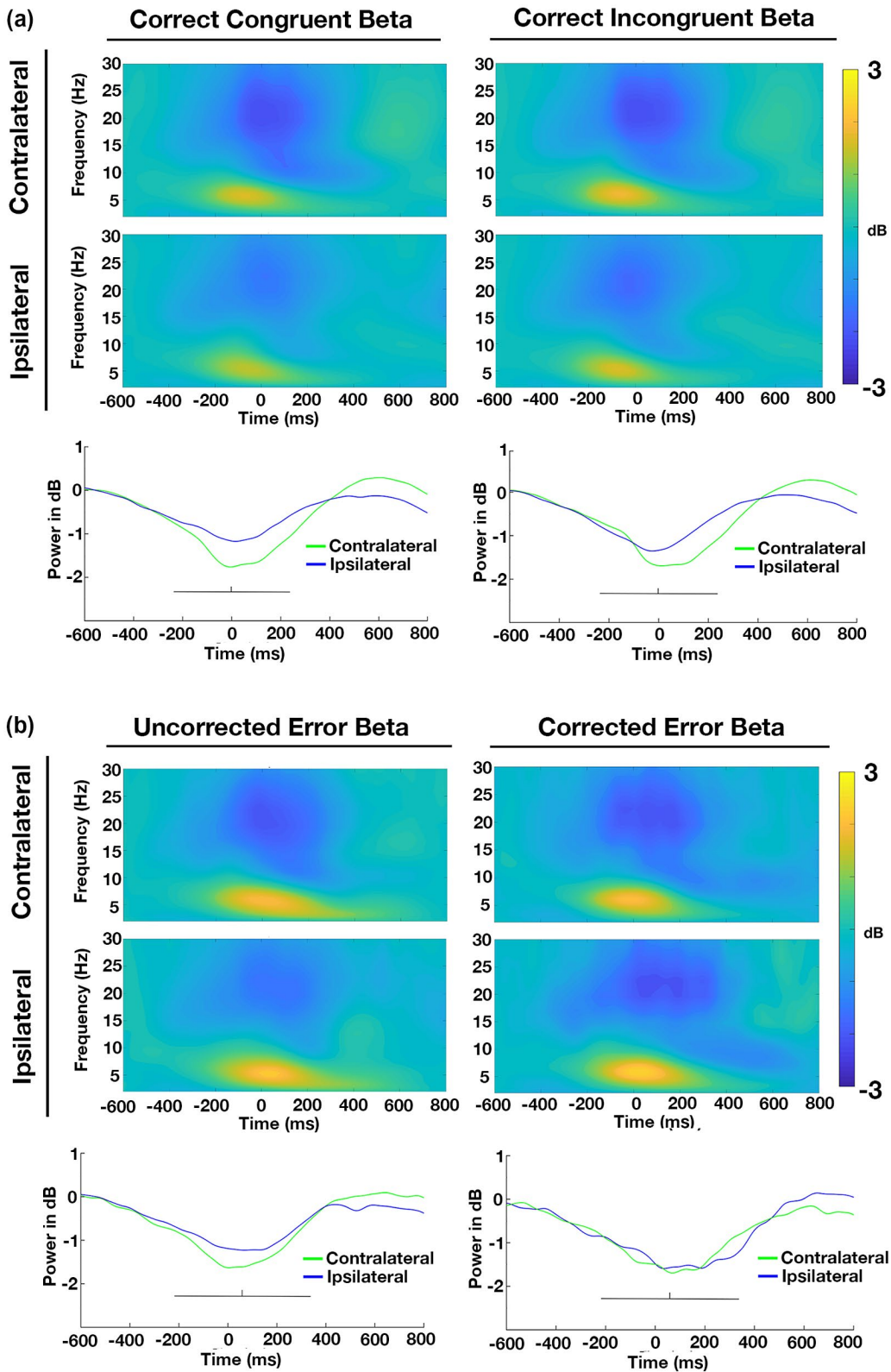
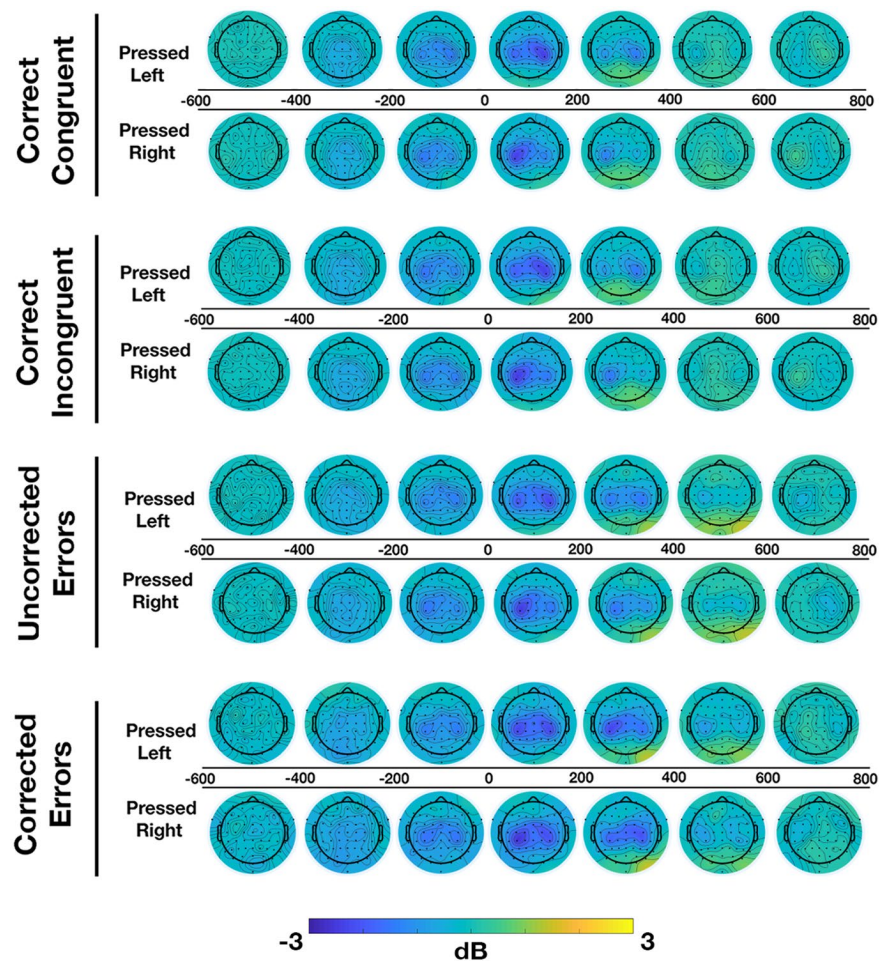


FIGURE 6 Beta power. Displays beta power for (a) correct trials as a function of congruency and (b) error trials as a function of corrections, collapsing across left (C3, C5, CP3, CP5) and right (C4, C6, CP4, CP6) electrode clusters to obtain ipsilateral and contralateral activity relative to initial response side. The time-series plot was constructed by collapsing across wavelets corresponding to frequencies between 13 and 25 Hz. The black bars represent the analysis windows, which are centered at the peak of the waveform (collapsing across congruency, corrections, and laterality). This analysis reports data for a subset of participants used to investigate fully executed error corrections

FIGURE 7 Topographical time series for beta power. Displays topographic plots illustrating beta power over time (as a function of whether the left or right button was pressed) in 200 ms intervals. This is presented for all conditions corresponding to Figure 6: correct congruent trials, correct incongruent trials, uncorrected errors, and corrected errors. This analysis reports data for a subset of participants used to investigate fully executed error corrections



by 2 (Congruency: Congruent, Incongruent) repeated measures ANOVA, revealed a main effect of laterality ($F(1,18) = 19.296, p < .001, \eta^2 = .517$), in which contralateral ($M = -1.388; SE = .197$) were more suppressed than ipsilateral ($M = -1.152; SE = .162$), as well as a main effect of accuracy ($F(1,18) = 7.927, p = .011, \eta^2 = .306$), in which errors ($M = -1.363; SE = .194$) were more suppressed than corrects ($M = -1.176; SE = .167$). There was a laterality by accuracy interaction ($F(1,18) = 12.474, p = .002, \eta^2 = .409$). Paired samples t tests revealed that error trials ($M = -1.314; SE = .184$) were more suppressed than correct trials ($M = -.990; SE = .148$) for ipsilateral ($p = .001, d = .402$), but were not significantly different (correct: $M = -1.363, SE = .193$; error: $M = -1.412, SE = .207$) for contralateral ($p = .514$). Finally, there was a laterality by accuracy by congruency interaction ($F(1,18) = 17.541, p = .001, \eta^2 = .494$). For corrects, contralateral (Congruent: $M = -1.391, SE = .196$; Incongruent: $M = -1.335, SE = .190$) was more suppressed than ipsilateral (Congruent: $M = -.936, SE = .141$; Incongruent: $M = -1.044, SE = .158$), irrespective of congruency. For errors, contralateral ($M = -1.407, SE = .215$) was more suppressed than ipsilateral ($M = -1.204, SE = .167$) for incongruent trials, but was not different (Contralateral:

$M = -1.418, SE = .208$; Ipsilateral: $M = -1.425, SE = .212$) for congruent trials. All other effects failed to reach significance (all $p > .14$). In addition to this analysis, limiting the included trials to only errors, a 2 (Laterality: Ipsilateral, Contralateral) by 2 (Congruency: Congruent, Incongruent) by 2 (Corrections: Corrected, Uncorrected) repeated measures ANOVA, revealed a main effect of corrections ($F(1,18) = 20.094, p < .001, \eta^2 = .527$), in which corrected errors ($M = -1.540; SE = .222$) were more suppressed than uncorrected errors ($M = -1.187; SE = .171$). There was also a laterality by congruency interaction ($F(1,18) = 7.646, p = .013, \eta^2 = .298$). Paired samples t tests revealed that congruent trials ($M = -1.425; SE = .212$) were more suppressed than incongruent ($M = -1.204; SE = .167$) for ipsilateral ($p = .039, d = -.242$), but were not significant different (congruent trials: $M = -1.418, SE = .208$; incongruent trials: $M = -1.407, SE = .215$) for contralateral ($p = .904$). Critically, there was also a laterality by corrections interaction ($F(1,18) = 7.652, p = .013, \eta^2 = .298$). Paired samples t tests revealed that contralateral ($M = -1.299; SE = .195$) was more suppressed than ipsilateral ($M = -1.075; SE = .152$) for uncorrected errors ($p = .007, d = .239$), but was not a significantly different (ipsilateral: $M = -1.553, SE = .225$;

contralateral: $M = -1.526$, $SE = .224$) for corrected errors ($p = .663$). All other effects and interactions failed to reach significance (all $p > .6$).

3.2 | Single-trial analyses

Since we are interested in the role of subthreshold error corrections (instead of fully executed corrections) on post-error compensations, all single-trial analyses (Figures 8 and 9) were conducted using uncorrected error trials, which permitted us to include all 50 participants in the analyses.

For the model of “theta, congruency, and next trial congruency predicting PES,” there was an effect of theta, $t(12,430) = -2.203$, estimate = $-.021$, $SE = .009$, $p = .028$, in which greater theta power predicted post-error speeding. All other interactions involving theta failed to reach significance (all $p > .2$). For the model of “theta, congruency, and next trial congruency predicting PEA,” there was an effect of theta, ($z = 3.291$, estimate = $.070$, $SE = .021$, $p < .001$), in which greater theta power predicted increases in PEA. All other interactions involving theta failed to reach significance (all $p > .2$).

For the model of “ERN, congruency, and next trial congruency predicting PES,” there was an effect of ERN, $t(13,250) = -3.866$, estimate = $-.035$, $SE = .009$, $p < .001$,

in which a larger ERN predicted post-error slowing. All other interactions involving the ERN failed to reach significance (all $p > .2$). For the model of “ERN, congruency, and next trial congruency predicting PEA,” there were no significant effects or interactions involving the ERN (all $p > .3$).

For the model of “ipsilateral beta, contralateral beta, congruency, and next trial congruency predicting PES,” there was an effect of ipsilateral beta, $t(13,820) = 3.317$, estimate = $.034$, $SE = .010$, $p < .001$, in which greater ipsilateral beta suppression predicted post-error speeding. All other interactions involving ipsilateral or contralateral beta failed to reach significance (all $p > .3$). For the model of “ipsilateral beta, contralateral beta, congruency, and next trial congruency predicting PEA,” there was an effect of ipsilateral beta, ($z = -2.289$, estimate = $-.053$, $SE = .023$, $p = .022$), in which greater ipsilateral beta suppression predicted *increased* PEA. There was also an effect of contralateral beta, ($z = 2.369$, estimate = $.056$, $SE = .024$, $p = .018$), in which greater contralateral beta suppression predicted *decreased* PEA. Finally, there was a contralateral beta by current trial congruency by next trial congruency interaction, ($z = -2.220$, estimate = $-.207$, $SE = .093$, $p = .026$), in which, on a congruent trial, greater contralateral beta suppression predicted increases in post-error accuracy if the next trial was congruent, but decreases in post-error accuracy if the next trial was an incongruent. However, on an incongruent trial, greater contralateral beta suppression predicted decreases in post-error accuracy irrespective of next trial congruency. In addition to the models predicting PES, we conducted an additional model that directly predicts next trial RT (without the post-error RT minus error RT computation). For the model of “ipsilateral beta, contralateral beta, congruency, and next trial congruency predicting next trial RT,” there was an effect of ipsilateral beta, $t(13,790) = 2.162$, estimate = $.022$, $SE = .010$, $p = .031$, in which greater ipsilateral beta suppression predicted post-error speeding. All other interactions involving either ipsilateral or contralateral beta failed to reach significance.

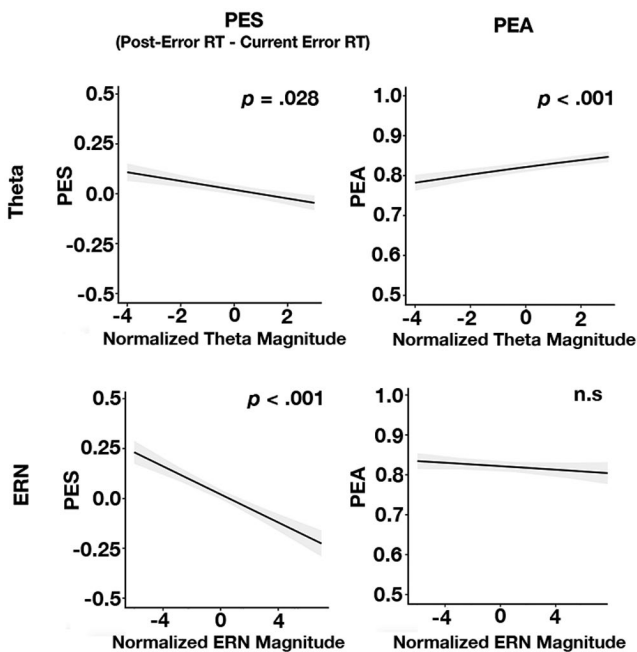


FIGURE 8 Single-trial models for theta power and ERN magnitude. Displays plots for single-trial models for theta power magnitude and ERN magnitude predicting post-error slowing/speeding (PES; Next Trial RT–Current Trial RT), and post-error accuracy. Models were limited such that the current trial must be an uncorrected error trial. Shaded regions depict the standard error. This analysis reports data for all 50 participants

4 | DISCUSSION

While many studies have investigated how error processing dynamics influence post-error compensations such as PES, studies often fail to consider the propensity for participants to immediately correct for their errors prior to the onset of the subsequent trial. We argue that the tendency to immediately correct errors as they occur is a critical step in the cascade of processing that links error commission and post-error behavior. The present study utilized lateralized beta power, which has been shown to index motor preparation (Fischer et al., 2018), to examine subthreshold error corrections. We observed that, even when a correction is not made, greater corrective activity predicts post-error speeding and enhanced

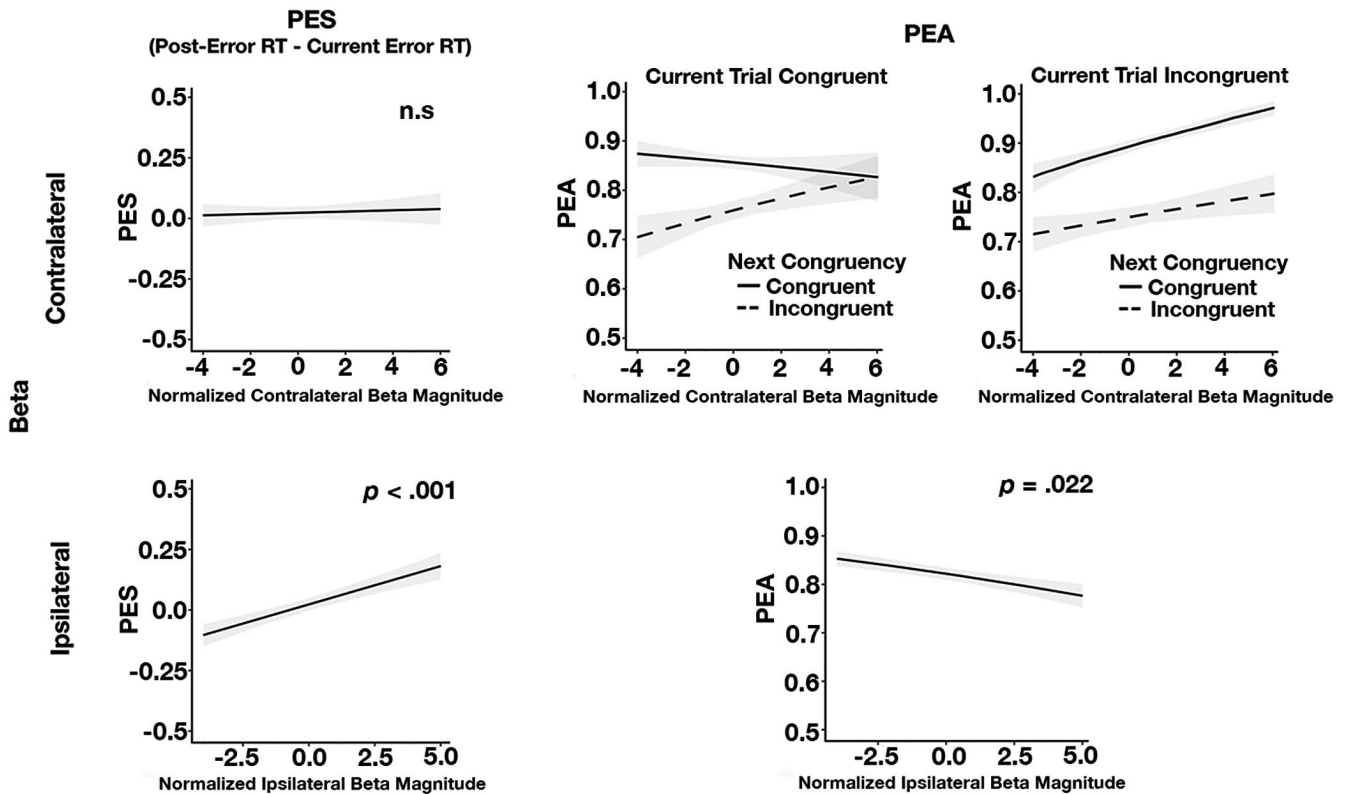


FIGURE 9 Single-trial models for beta power. Displays plots for single-trial models for beta power magnitude predicting post-error slowing/ speeding (PES; Next Trial RT–Current Trial RT), and post-error accuracy. Models were limited such that the current trial must be an uncorrected error trial. Shaded regions depict the standard error. This analysis reports data for all 50 participants

post-error accuracy at the single-trial level. This provides support for the notion that immediate corrective activity is adaptive and suggests that subthreshold error corrections should be taken into account to fully understand error-monitoring processes.

4.1 | Subthreshold error corrections predict post-error compensations

In order to validate lateralized beta power as an index of subthreshold error corrections (i.e., ipsilateral beta suppression), we first investigated trials in which an overt correction was made. While we found that there was greater suppression in both hemispheres for corrected errors, the laterality by corrections interaction and follow-up analyses indicated that there was a difference between ipsilateral and contralateral beta suppression for uncorrected, but not corrected, errors. Although this analysis demonstrates that lateralized beta suppression can serve as an index for overt error corrections, beta power has also been shown to be sensitive to the intention to perform an action in the absence of an overt response (Pfurtscheller et al., 1997; Tzagarakis et al., 2010). These data are in line with the notion that lateralized beta suppression can be employed as an index of motor preparation, as

opposed to simply capturing overt motor responses per se. Accordingly, we examined lateralized beta suppression at the single-trial level to investigate subthreshold motor activation for error corrections that take place on uncorrected error trials. More specifically, while contralateral beta suppression indexed the magnitude of activation for the initial erroneous response, ipsilateral beta suppression indexed the magnitude of activation for the corrective response. When predicting post-error compensations, contralateral beta suppression (activation for the initial response) failed to predict post-error slowing or speeding, but predicted decreased post-error accuracy (i.e., stronger activation for the erroneous response on trial N predicted worse performance on trial $N + 1$). However, ipsilateral beta suppression (activation for the corrective response) predicted both post-error speeding and increased post-error accuracy (i.e., activation for the corrective response on trial N predicted better performance on trial $N + 1$). These data are in line with the notion that initial error commissions can lead to further performance decrements, whereas the magnitude of a subthreshold error correction leads to performance recovery.

Recently, Crump and Logan (2013) demonstrated that while post-error slowing is often observed following uncorrected error trials, post-error *speeding* can sometimes be observed following corrected error trials. Consequently, they

proposed an alternative interpretation of post-error slowing: during experimental paradigms in which participants must respond on successive trials, but are instructed not to correct their errors, post-error slowing occurs as the result of inhibiting the implicit urge to correct one's errors. In contrast, when participants are allowed to correct their errors, post-error speeding can be observed (Crump & Logan, 2013). In agreement with these prior findings, the present study found that greater activation for subthreshold error corrections (greater ipsilateral beta suppression) predicted post-error speeding. Although it may seem surprising that post-error *increases* in motor activity are related to post-error *speeding*, this is in agreement with previous research demonstrating that post-error *reductions* in motor activity were related to greater post-error *slowing* (Danielmeier et al., 2011; King et al., 2010). We also note that for the present study, participants were instructed to weigh the speed and accuracy of their response equally and were not provided any instruction regarding whether they should (or should not) correct their errors. A recent study by Damaso et al., (2020) illustrated that when errors are committed during trials in which accuracy is prioritized over speed, performance on that trial would benefit from spending more time evaluating the incoming information and would consequently lead to post-error slowing. However, during trials in which accuracy and response time are equally prioritized, performance on that trial would *not* benefit from additional information and would consequently lead to post-error speeding.

Although not a primary focus of the current investigation, it is important to note that the present study provides a direct test of the core assumptions for conflict monitoring theory (Botvinick et al., 2001; Yeung et al., 2004), which states that the ERN reflects competing co-activations of motor programs for both response options—the initial erroneous response and the corrective response, which is based on further stimulus processing. For the present study, when investigating corrected error trials, the magnitude difference of beta suppression between hemispheres was not significantly different, suggesting that participants experience the most conflict on corrected error trials. This interpretation is also supported by the pattern of results observed for theta and the ERN, which were greatest in magnitude for corrected compared to uncorrected errors (also see Kalfaoğlu et al., 2018). This pattern of results matches the predictions of conflict monitoring theory, which predicts that the ERN should be largest for error trials that are followed by fully executed corrections (Botvinick et al., 2001; Yeung et al., 2004). However, only theta completely aligns with the predictions of conflict monitoring theory when congruency is taken into account, with theta power being greater on congruent trials as compared to incongruent trials and the reverse being true for the ERN (see below for further discussion of the dissociation between theta and the ERN). When investigating activation of the corrective

response on uncorrected error trials, however, as the magnitude of ipsilateral beta suppression (the subthreshold error correction) increases, the greater the conflict between each response option. Similar to full corrections, this suggests that greater magnitude activation for subthreshold error corrections are associated with greater magnitudes of response conflict. What we cannot determine from the data however is the mechanism by which conflict improves task performance: Does the propensity to make a correction resolve ongoing error-processing faster (Gehring et al., 2011), or does the propensity to make a correction recruit additional control, which is carried over to the subsequent trial (Cavanagh & Shackman, 2015; Ridderinkhof et al., 2004)? Evidence for resolving error-processing faster is derived from the finding that theta peaks earlier for corrected compared to uncorrected errors (also see Navarro-Cebrian et al., 2016), which suggests that conflict has been addressed earlier in time. Evidence for recruiting additional control is derived from the finding that greater conflict (as indexed by both greater ipsilateral beta suppression and greater induced theta power) predicts post-error speeding and greater post-error accuracy, which suggests that participants are exerting greater cognitive control on post-high conflict trials. Although this would appear to be similar to the Gratton Effect (Gratton et al., 1992), in the present scenario, conflict associated with the subthreshold error correction, not conflict associated with stimulus congruency, modulates accuracy, and response time on the following trial. Regardless of the interpretation, the present findings provide additional evidence that conflict plays an adaptive role during decision-making processes.

4.2 | Dissociation of post-error response time and post-error accuracy

A number of studies have shown that greater post-error slowing can be associated with either increased post-error accuracy (adaptive; Beatty et al., 2020; Botvinick et al., 2001; King et al., 2010; Maier et al., 2011; Marco-Pallarés et al., 2008; Steinhäuser & Andersen, 2019) or decreased post-error accuracy (maladaptive; Beatty et al., 2018; Buzzell et al., 2017; Jentsch & Dudschig, 2009; Notebaert et al., 2009; Steinhäuser et al., 2018; Ullsperger & Danielmeier, 2016; Van der Borgh et al., 2016). In terms of averaged-level post-error behavior, we found that the extent of post-error slowing for corrected compared to uncorrected errors did not differ. Interestingly, when investigating post-error accuracy, there were relative *decreases* in accuracy following *uncorrected* errors, but relative *increases* in accuracy following *corrected* errors. This suggests that post-error slowing and post-error accuracy can be dissociated when accounting for whether a person corrected for their response. Taken together with the linear mixed-effects models, the present data collectively

suggest that both fully executed corrections and subthreshold error corrections benefit subsequent trial performance. Although previous work has shown a relationship between corrected and uncorrected errors and the magnitude of post-error response time (Kalfaoğlu et al., 2018), to our knowledge, the present study is the first to illustrate a relationship between response corrections, subthreshold error corrections, and post-error accuracy. Furthermore, this dissociation of post-error slowing and post-error accuracy is in agreement with previous work (Carp & Compton, 2009; Danielmeier & Ullsperger, 2011), which suggests that post-error slowing and post-error increases in accuracy can be dissociated as a function of variable response-stimulus interval durations.

4.3 | Dissociation between theta power and the ERN

Investigating fully executed and subthreshold error corrections has also enabled us to expand on previous findings that suggest that theta power and the ERN are dissociable processes (Beatty et al., 2020). In concordance with previous research investigating theta power as a function of response corrections, we observed greater theta power (Kieffaber et al., 2016), as well as an earlier peak latency for theta power (Navarro-Cebrian et al., 2016), on corrected compared to uncorrected errors. However, when investigating the ERN as a function of corrections, although the ERN has been found to be larger for corrected compared to uncorrected errors (Gehring et al., 1993; Kalfaoğlu et al., 2018; Rodríguez-Fornells et al., 2002) it was not significantly different in its latency. Perhaps the temporal dynamics of conflict for the ERN are obscured because of potential influence from overlapping components in the EEG signal. Moreover, this suggests that although the ERN can reliably index conflict magnitude, the lack of interference from overlapping neural processes enables theta to reliably index both the magnitude and temporal dynamics of conflict.

Consistent with our previous findings (Beatty et al., 2020), the averaged-level analysis indicated that, while theta power was larger for congruent compared to incongruent errors, the ERN was larger for incongruent compared to congruent errors. This replication provides additional evidence that the ERN and theta respond in a diametrically opposed manner to response conflict, which suggests that they are dissociable neural processes. The differential response of theta and the ERN as a function of conflict suggests that these indices are differentially sensitive to error types (van Driel et al., 2012; Maier et al., 2008). While theta more closely aligns with the congruency dynamics outlined in conflict monitoring theory (Botvinick et al., 2001; Yeung et al., 2004), the ERN, as suggested in our previous report (Beatty et al., 2020), is perhaps

selectively sensitive to an affective aspect of action-monitoring (Dignath et al., 2020) associated with incongruent errors. Critically, we emphasize that although there were differences in component sensitivity as a function of congruency, the rate of corrections for congruent and incongruent errors was equivalent. Furthermore, both current and next trial congruency did not play a role when predicting post-error behavior.

Finally, when investigating how theta and the ERN predict next trial behavior at the single-trial level, we constructed linear mixed-effects models that predicted post-error slowing and post-error accuracy. We observed differences in component sensitivity when predicting next trial behavior: although theta predicted post-error speeding and increased post-error accuracy, the ERN predicted post-error slowing, but failed to predict post-error accuracy. This finding provides additional evidence that induced theta power and the ERN are not the same neurocognitive process captured in different domains.

5 | CONCLUSION

In conclusion, we demonstrate that fully executed and subthreshold error corrections can be indexed by greater beta suppression ipsilateral to the initial response. Furthermore, we demonstrate that both of these phenomena, which are linked to response conflict, lead to improved performance following an erroneous response. Our findings suggest that when investigating the relationship between error processing and post-error behavior, it is important to take into consideration the magnitude of subthreshold error corrections. By investigating subthreshold error corrections, we were able to differentiate and explore the unique characteristics of important variables related to error processing such as post-error slowing/speeding and post-error accuracy, as well as differentiate other neural indices of conflict and cognitive control such as induced theta power and the ERN.

AUTHOR CONTRIBUTIONS

Paul J Beatty: Conceptualization; Data curation; Formal analysis; Funding acquisition; Investigation; Methodology; Project administration; Validation; Visualization; Writing-original draft; Writing-review & editing. **George A Buzzell:** Conceptualization; Validation; Writing-review & editing. **Daniel M Roberts:** Formal analysis; Methodology; Validation; Writing-review & editing. **Yelyzaveta Voloshyna:** Data curation. **Craig G McDonald:** Conceptualization; Funding acquisition; Investigation; Methodology; Project administration; Supervision; Validation; Visualization; Writing-review & editing.

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the Supporting Information section.

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