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Speeded response errors and the error-related negativity modulate early sensory processing☆

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ABSTRACT

Empirical research demonstrates that when the time following error commission is constrained, subsequent sensory processing can be impaired (Buzzell et al., 2017). This reduction in sensory processing is presumably due to a bottleneck for cognitive resources produced by an overlap between error processing and subsequent stimulus processing. This finding suggests that the system dedicated to improving task performance can actually sometimes be the source of performance failures. Although this finding established that data-limited errors lead to a reduction in sensory processing at short response stimulus intervals (RSIs), it remains unclear if the relationship between error processing and subsequent sensory processing can be modulated by speeded-response errors. In the present study, event-related potentials and behavioral measures were recorded while participants performed a modified version of a Simon task, in which RSI duration was varied. We found that sensory processing, indexed by the P1 component, was reduced following errors at short (200–533 ms), but not long (866–1200 ms), RSIs. Moreover, the magnitude of error processing differentially influenced subsequent sensory processing as a function of RSI. However, whereas prior work demonstrated that the error positivity (Pe) modulated sensory processing on the subsequent trial, only the error-related negativity (ERN) did so within the Simon task. This suggests that although both data-limited errors and speeded-response errors can impact subsequent sensory processing, different stages of error processing appear to mediate this phenomenon.

The performance monitoring system is a network of brain regions, including the medial frontal cortex (MFC; Ridderinkhof et al., 2004), that (1) determines when an error has been committed and (2) provides a signal to instantiate cognitive control to reduce the probability of committing additional errors (Shenhav et al., 2013; Ullsperger et al., 2014a,b). As a result of these processes, various types of post-error compensatory behavior, such as increases in response time following an error (post-error slowing; PES) and modulation of response accuracy (post-error accuracy; PEA), are often observed (Laming, 1979; Rabbitt, 1979). Although these adjustments have come to be expected during tasks that require performance monitoring and the instantiation of cognitive control, adaptive and maladaptive theories of post-error adjustments differ with respect to how they interpret post-error slowing. According to the *adaptive* perspective, these adjustments are suggested to reflect a more cautious response strategy (greater PES) with the goal to improve task performance (increased PEA; Botvinick et al., 2001; King et al., 2010; Maier et al., 2011). According to the *maladaptive* perspective, these adjustments are thought of as a disruption from the task set that delays subsequent trial processing (greater PES) and leads to a deterioration of performance (decreased PEA; Buzzell et al., 2017; Jentzsch and Dudschig, 2009; Notebaert et al., 2009; Ullsperger and Danielmeier, 2016; Van der Borght, Schevernels, Burle and Notebaert, 2016). One explanation for the post-error decrease in task performance is that the error itself captures the participant's attention (orienting theory; Notebaert et al., 2009), distracting the participant and impeding performance on the subsequent trial. A related explanation is that deficits in processing occur as a result of limitations in cognitive resources when error processing and subsequent stimulus processing overlap in time (bottleneck theory; Jentzsch and Dudschig, 2009).

To further investigate this phenomenon, we previously utilized EEG to demonstrate that as the magnitude of error processing increases, as indexed by the error positivity (Pe; Nieuwenhuis, Ridderinkhof, Blom, Band and Kok, 2001; Steinhauser and Yeung, 2010), the magnitude of sensory processing on the following (post-error) trial (as indexed by the

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lateral occipital P1) decreases (Buzzell et al., 2017). Critically, this negative relationship between the Pe and subsequent P1 was present at short (200–533 ms), but not long (866–1200 ms) RSI durations, which suggests the presence of an attentional bottleneck when error processing overlaps with subsequent stimulus processing. This finding provided electrophysiological evidence as to how the performance monitoring system, which is generally believed to produce task-positive behavioral adaptations, can sometimes lead to distraction and additional errors.

Given that we previously utilized an attentionally demanding color discrimination task, it remains unclear whether diminished task-relevant post-error sensory processing would also be observed in tasks in which response conflict, as opposed to stimulus ambiguity, was the primary determinant of error commission. Errors produced by stimulus ambiguity can be considered "data limited errors," which can result in delayed activation for both response options because of difficulty in discriminating the imperative stimuli; both choices seem equally likely to be correct until enough evidence has been accumulated. In contrast, errors resulting from response conflict can be considered "speeded response errors" in which the participant responds prior to fully evaluating stimulus information. Conflict arises when a comparison is made between an efference copy of the initial, incorrect response and motor programming of the correct response (determined from continued stimulus processing). The primary difference for these error-types is the temporal dynamics of how conflict arises in the two contexts. Although conflict for data-limited errors builds throughout the decision process itself, conflict for speeded-response errors arises from a post-response comparison. While our previous study investigated the relationship between error and sensory processing with regard to conflict that builds throughout the decision process itself, by utilizing a task that produces speeded-response errors, such as a Simon task (Simon, 1969), it is possible to specifically investigate that relationship with regard to conflict that takes place during this post-response comparison.

Recent work has suggested that surprising events (such as errors) initiate a cascade of neural events that lead to adjustments in processing and compensatory behavior in an effort to maintain performance despite a disruption of the task set (Wessel, 2017; Wessel and Aron, 2017). More specifically, Wessel (2017) has provided a theoretical framework suggesting that when an error occurs, it produces an automatic inhibition of both motor and cognitive activity, as well as a rapid attentional orienting to the error. These reflexive processes are then followed by a more deliberative error processing stage that can yield adaptive changes to behavior. We propose that the *type* of error committed determines which part of this error processing cascade influences subsequent cognitive processes. During speeded response errors, given the comparatively rapid and easily discerned comparison of the initial, incorrect response and the correct response mapping, it is possible that early, reflexive error processing (as indexed by the ERN) could have a greater influence on subsequent cognitive processes. By contrast, for data-limited errors, given the relatively slow activation of response options elicited by ambiguous stimuli, it is possible that a more deliberative process (as indexed by the Pe) could have a greater influence on subsequent cognitive processes.

The present study utilized a Simon task, while manipulating RSI duration in order to investigate if task-relevant, error-induced reductions in sensory processing can be generalized to contexts other than those in which task performance is determined by stimulus discriminability. Similar to our previous study, we analyzed the response-locked fronto-central error-related negativity (ERN) and parietal error positivity (Pe) as indices of error processing, as well as the stimulus-locked lateral occipital P1 as an index of sensory processing on the following trial. We also employed the use of a Laplacian transform (current source density; CSD) of the EEG data in order to reduce volume conduction and allow for the analysis of these temporally overlapping, but spatial distinct, neural components (Kayser and Tenke, 2006). There are three hypotheses of error activity and its impact on subsequent trial processing. First, if the error itself prompts an increase in attention to the task set, it is possible that larger error-related ERPs would be predictive of an *enhancement* in the P1 on the following trial. Second, since speeded-response errors are more salient than data limited errors, there could be a general increase in the magnitude of error processing, which would place increased demand on capacity-limited cognitive resources (e.g. create a bottleneck) or capture more attention (error-orienting). In this scenario, it is possible that increases in error-related ERPs would be predictive of a *reduction* in the P1 on the following trial, as has been previously observed for data-limited errors (Buzzell et al., 2017). Third, it is also possible that since speeded response errors are more salient, error monitoring, although enhanced, would be more efficient, resulting in a reduced impact on subsequent sensory processing. This latter hypothesis would be associated with a reduced or absent effect on the subsequent trial P1.

1. Method

1.1. Participants

Twenty-four participants between 18 and 45 years of age (mean age = 24.75; 16 female) participated in this study in exchange for course credit at George Mason University. A total of three participants were removed (one participant was removed due to below chance accuracy, one participant was removed because they routinely corrected their responses, and one participant was removed due to experimental error), leaving a total of twenty-one participants (mean age = 23.62; 13 female) to be incorporated into all analyses. All participants were right-handed, had normal (or corrected to normal) vision, had no known neurological deficits, and were not taking any medications that affect the nervous system. All participants provided written informed consent after having been explained the experimental procedures. All procedures were approved by the George Mason University Office of Research Integrity and Assurance.

1.2. Experiment design

In order to attain a sufficient number of speeded response errors, participants completed a Simon task (see Fig. 1). All stimuli were generated via custom MATLAB (MathWorks, Natick, MA) scripts and Psychtoolbox (Brainard, 1997; Kleiner et al., 2007; Pelli, 1997) functions and were presented on the Cambridge Research Systems Display++ LCD monitor (Cambridge Research Systems Ltd). On each trial, 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°, located 4.25° 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°, was presented for 200 ms in the center of one of the two boxes. Red and green stimuli (as well as the side of the screen they were presented on) were equiprobable and perceived luminance of the stimuli was equated using the sRGB gamma function.

To familiarize themselves with the task, participants first completed an easier version of the task, in which they were provided with a 2 s response deadline as well as trial-by-trial feedback of their performance (no trial-level accuracy feedback was presented during the actual experiment). Following practice of an easy version of the task, participants completed an additional practice that was more difficult and identical to the experimental version of the task. Participants were instructed to respond as quickly and accurately as possible by pressing either the "2" key (using their left index finger) or the "8" key (using their right index finger) located on the top row of the computer keyboard to indicate what color was presented. These response mappings were counterbalanced across participants. During the main task, if the participant's response exceeded a 500 ms response window, the words "Too Slow" were presented on the screen and the trial, as well as the following trial, were removed from all analyses. Participants were advised not to correct their responses, given the short RSI between some trials. Following the response, participants were provided with a randomly selected RSI between 200 ms and 1200 ms in duration. In order to combat

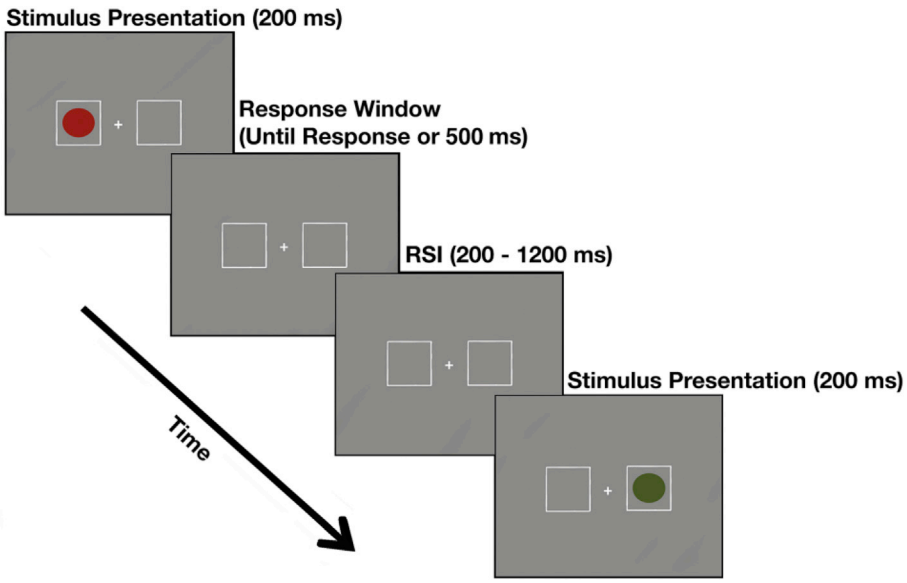

Fig. 1. Experimental Paradigm. Relative stimulus contrasts have been increased for presentation purposes.

fatigue, between each block, participants were required to rest for at least 30 s prior to beginning the next block. During this period, they were also informed of their block accuracy. The main experiment consisted of 3520 trials (22 blocks of 160 trials). The last two blocks, however, were dedicated to passive viewing of the stimuli and are not discussed here.

The behavioral analyses, EEG data processing, and single trial analyses are largely similar to those presented in Buzzell et al. (2017). For completeness, they are described below.

1.3. Behavioral analysis

If the participant responded within 150 ms of stimulus onset, responded after the 500 ms response deadline, corrected for their response, or did not provide a response, this trial (as well as the following trial) were removed from all analyses. Overall accuracy and response times for correct, error, congruent, and incongruent trials were calculated and statistically compared using a 2 [Accuracy (Correct, Error)] by 2 [Congruency (Congruent, Incongruent)] repeated-measures ANOVA. PES was calculated as a percentage change between (1) the response time for correct trials following an error and (2) the response time for correct trials following a correct response. Likewise, PEA was calculated as a percentage change between (1) the accuracy for trials following an error and (2) the accuracy for trials following a correct response. Both PES and PEA were calculated as a percentage change in order to capture *relative* changes in compensatory behavior while still accounting for potential differences in RT distributions across participants (Buzzell et al., 2017). PES and PEA were calculated independently for short (200–533 ms) and long (866–1200 ms) RSIs. The range for short and long RSIs reflect the lower and higher tertiles of all possible RSI durations. The data were separated into these bins to increase signal-to-noise ratio and the likelihood of detecting an effect of RSI (Buzzell et al., 2017; Jentzsch and Dudschig, 2009). Potential differences in PES and PEA for short and long RSI trials were statistically compared using repeated-measures *t* tests.

1.4. EEG data acquisition and processing

EEG data were collected using a Brain Vision ActiChamp 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. The data were recorded at a sampling rate of 1000 Hz, which utilized an online 0.1–250 Hz bandpass filter. In concordance with impedance recommendations for high-impedance active EEG systems, impedance for all electrodes was maintained below 25 kΩ throughout the recording session.

Processing of EEG data was conducted using the EEGLAB (Delorme and Makeig, 2004) toolbox for the MATLAB programming environment (MathWorks, Natick, MA). Data were detrended, low-pass filtered at 30 Hz (using a Butterworth filter from the ERPLAB plugin; Lopez-Calderon and Luck, 2014) and down-sampled to 500 Hz. On a copy of the original dataset, the data were high pass filtered at 1 Hz, segmented into a series of consecutive 1000-ms epochs, and subjected to automated amplitude rejection threshold of ±500 μV and a spectral rejection threshold of 50 dB within the 20–40 Hz band using the pop_rejspec function (to remove EMG-like activity; Delorme and Makeig, 2004). If more than 20% of epochs were marked for rejection in a given channel, that channel was removed from all copies of the dataset. The data were then decomposed using independent component analysis (ICA; Winkler et al., 2015). The ICA component weights that were generated for the 1 Hz high-pass filtered dataset were then copied onto the original 0.1 Hz high-pass dataset. All further analyses were performed on the 0.1 Hz high-pass dataset. After rejecting independent components corresponding to blinks and saccades, the data were epoched from −200 to 800 ms for all stimulus and response markers. The data were then subjected to a more strict automated rejection procedure using a voltage rejection threshold of ±100 μV and a spectral rejection threshold of 50 dB within the 20–40 Hz band using the pop_rejspec function. Similar to earlier in the processing stream, if more than 20% of epochs were marked for rejection for a given channel, that channel was removed from the dataset. To ensure that the response and following trial stimulus pairs were matched, if one of the two trials were rejected during processing, the corresponding trial was also rejected. All rejected channels were interpolated (using a spherical spline interpolation) and all epochs were baseline corrected from −200 ms to 0 ms. Lastly, to separate cortical sources and reduce the impact of volume conduction, we conducted a Laplacian (current source density; CSD) transformation of the data (Kayser and Tenke, 2006). Reducing the impact of volume conduction is important following short RSI durations, since it is possible to observe a temporal overlap between error-related and subsequent stimulus-related ERPs. The use of a Laplacian transformation allowed for the removal of

volume conduction and the separate analysis of error and stimulus-related ERPs.

1.5. ERP analyses

Since we are interested in speeded-response errors that are believed to produce response conflict (incongruent trial errors) - not errors presumably due to lapses in attention (congruent trial errors) - and that there is no reason to believe that error processing would differentially modulate subsequent sensory processing depending on subsequent trial accuracy, all analyses were conducted utilizing trial pairs in which correct/error incongruent trials were followed by either correct or error trials. For consistency with our previous study however, analyses were also performed while limiting the subsequent trial to correct trials; these additional analyses are reported in the supplemental material. The average numbers of trials incorporated into the matched response and following trial stimulus grand average waveforms were as follows: "Correct Incongruent followed by Correct at Short" (M = 271.952; SD = 41.033), "Error Incongruent followed by Correct at Short" (M = 51.857; SD = 18.339), "Correct Incongruent followed by Correct at Long" (M = 267.286; SD = 45.372), "Error Incongruent followed by Correct at Long" (M = 58.238; SD = 21.196). Although there were not enough congruent errors to provide a reliable signal for stimulus locked P1 - "Correct Congruent followed by Correct at Short" (M = 301.524; SD = 49.305), "Error Congruent followed by Correct at Short" (M = 31.429; SD = 11.599), "Correct Congruent followed by Correct and Long" (M = 295.429; SD = 48.046), and "Error Congruent followed by Correct and Long" (M = 34.762; SD = 14.007), there were enough trials to provide a reliable signal for the response-locked ERN and Pe as a function of congruency (see supplemental materials). To clarify, the fact that incongruent errors are described as speeded-response errors, does not indicate that congruent errors are data-limited errors. Electrodes selected for all analyses were chosen based on visual inspection of where each component was maximal after collapsing across conditions. Statistical analyses of the ERN (maximal at electrode Cz) and Pe (maximal at electrode POz) were conducted using trial accuracy (correct, error) paired-sample t-tests with a predefined time window of 40 ms for the ERN (16 ms–56 ms) and 100 ms for the Pe (278 ms–378 ms). Time windows for the response-locked ERPs were centered on the respective peaks of the grand-average difference waveform (error minus correct). Given that the color stimuli were presented in different hemifields from trial-to-trial, there was a latency difference for the P1 component depending on stimulus presentation hemifield and the analysis electrode (PO7 or PO8). For instance, analyzing the contralateral electrode relative to stimulus presentation yielded an earlier peak latency of the P1 compared to using the ipsilateral electrode. To compensate for this latency shift, two distinct analysis windows were computed (Di Russo, Martínez, Sereno, Pitzalis and Hillyard, 2002). Prior to statistical analysis, the mean amplitude values for the contralateral windows and ipsilateral windows at each electrode were each averaged together to create general contralateral and ipsilateral mean amplitudes. Statistical analysis of the P1 was conducted using a 2 × 2 × 2 [Laterality (Contra, Ipsi) by Previous Accuracy (Correct, Error) by RSI (Short, Long)] ANOVA, with a predefined time window of 40 ms (Ipsilateral: 122 ms–162 ms; Contralateral: 90 ms–130 ms), centered on the respective peaks. Analysis window durations for all components were based on window durations of our previous paper (Buzzell et al., 2017) as well as consideration from prior work investigating these components (ERN and Pe - Steinhauser and Yeung, 2010; Ipsilateral and Contralateral P1s - Di Russo et al., 2002).

1.6. Single trial analyses

Similar to Buzzell et al. (2017), to assess if error processing (as indexed by the ERN/Pe) was predictive of changes in post-error sensory processing (as indexed by the subsequent trial P1), as well as changes in behavioral indices of post-error compensation (PES, PEA) on a single-trial level, we performed generalized linear mixed-effects analyses 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). The only difference in the approach used here, as compared to our previous report (Buzzell et al., 2017), is that RSI was treated as a continuous variable as opposed to a categorical variable. Prior to carrying out each analysis, models were constructed such that the continuous variables (P1 magnitude, RSI, ERN magnitude, Pe magnitude, response time) were centered and scaled to have a mean of 0 and SD of 1 across the data set. These variables were fit using linear mixed-effects analysis using the lmer function, with restricted maximum likelihood estimation. The categorical variable (accuracy) was examined using sum contrasts and fit using generalized linear mixed-effects models using the glmer function with logit link with maximum likelihood estimation. Within-subject 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. Each mixed-effects model was defined by the following formula:

$$Y = X\beta + Zu + \varepsilon \quad (1)$$

Where Y represents the response variable, X represents the fixed effect design matrix, β represents the fixed effect coefficients, Z represents the random effect design matrix, u represents the random effect coefficients, and ε indicates the error term.

Using the syntax of the R package lme4, each mixed-effects model was formed via:

$$\text{dependent\_var} \sim 1 + (\text{fixed\_effect\_1} * \text{fixed\_effect\_2}) + (1 \mid \text{Participant}) \quad (2)$$

This syntax indicates a model with a fixed effect for overall model intercept (the initial '1'), fixed effects for all independent variables of interest and their interactions (here listed with just two independent variables for clarity), and a random effect of variation in intercept per participant ('1 | Participant').

The main analysis explored the effect of error-related ERP component magnitude (either ERN or Pe) and RSI (as a continuous variable) on subsequent trial sensory-related ERP component magnitude (P1), with laterality (ipsilateral or contralateral stimulus presentation) as a factor. The models predicting single trial modulation of post-error behavior [next trial response time (PES) and next trial accuracy (PEA)] were run as separate models. Similar to the group-level analysis, all models and analyses were limited to trial pairs in which the current trial was an incongruent error and the following trial could be a correct or error trial. For consistency with our previous study, analyses were also performed while limiting the subsequent trial to correct trials (see supplemental material).

2. Results

2.1. Behavior

Overall accuracy throughout the task was 83.4% (congruent trials: 87.7%, incongruent trials: 79.0%). Investigating the traditional behavioral effects for a Simon task, a 2 (Accuracy [Correct, Error]) by 2 (Congruency [Congruent, Incongruent]) repeated measures ANOVA, revealed a main effect of accuracy ($F(1,20) = 65.642$, $p < .001$, $\eta_p^2 = 0.766$), in which errors (M = 344.111; SE = 5.184) were faster than corrects (M = 364.735; SE = 3.283), and a main effect of congruency ($F(1,20) = 16.301$, $p = .001$, $\eta_p^2 = 0.449$), in which incongruent trials (M = 352.566; SE = 4.337) were faster than congruent trials (M = 356.280; SE = 4.337). Additionally, there was an accuracy by congruency interaction ($F(1,20) = 274.280$, $p < .001$, $\eta_p^2 = 0.932$). Paired samples t-tests revealed that errors (M = 329.121; SE = 4.792) were faster than corrects (M = 376.011; SE = 3.568) for incongruent trials

($p < .001$, $d = 2.21$), but were not significantly different (Corrects: $M = 353.459$, $SE = 3.182$; Errors: $M = (359.121$, $SE = 4.792)$ for congruent trials ($p = .101$). In addition, congruent trials ($M = 353.459$; $SE = 3.182$) were faster than incongruent trials ($M = 376.011$; $SE = 3.568$) for corrects ($p < .001$, $d = -1.415$), but incongruent trials ($M = 329.121$; $SE = 4.792$) were faster than congruent trials ($M = 359.100$; $SE = 5.732$) for errors ($p < .001$, $d = 1.105$).

With regard to effects on post-error behavior following incongruent trials (Fig. 2), a paired-sample *t*-test revealed an effect on PES ($t(20) = 2.555$, $p = .019$, $d = 0.538$), in which slowing following short RSIs ($M = 1.542\%$; $SD = 5.559\%$) was greater than long RSIs ($M = -1.080\%$; $SD = 2.772\%$). In addition, a paired-sample *t*-test revealed an effect on PEA ($t(20) = -6.295$, $p < .001$, $d = -0.924$), in which accuracy following short RSIs ($M = -17.483\%$; $SD = 12.044\%$) was lower than long RSIs ($M = -6.841\%$; $SD = 10.702\%$). (See supplemental material for analyses investigating effects on post-error behavior following *congruent* trials; Fig. S1).

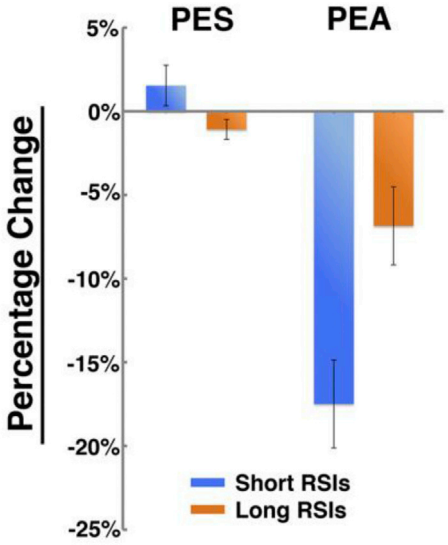

Fig. 2. Post-error behavior. PES and PEA following short and long RSI durations for incongruent trials. PES and PEA were calculated as a percentage change between post-error behavior and post-correct behavior. Error bars signify the SEM.

2.2. ERP components

For the current trial response-locked ERN (Fig. 3A), a paired-sample *t*-test revealed an effect of accuracy ($t(20) = 6.476$, $p < .001$, $d = 2.23$) in which errors ($M = -23.615$; $SE = 2.293$) were larger (more negative) than corrects ($M = 4.929$; $SE = 3.253$). For the Pe (Fig. 3B), a paired-sample *t*-test revealed an effect of accuracy ($t(20) = -8.445$, $p < .001$, $d = -1.231$), in which errors ($M = 7.295$; $SE = 1.787$) were larger (more positive) than corrects ($M = -5.572$; $SE = 2.415$). This pattern of results is consistent with previous literature on these components (Steinhauser and Yeung, 2010).

An analysis was also conducted to investigate the amplitudes of the ERN and Pe for congruent and incongruent errors (see supplemental material; Fig. S2). Although there is evidence from flanker tasks in which congruent errors produce a larger ERN than incongruent errors (Scheffers and Coles, 2000; Yeung et al., 2004), the present study, which utilized a Simon task, observed that incongruent errors produce a larger ERN than congruent errors. This finding may be attributable to task differences - in a flanker task, errors are likely influenced by variation in feature-based attentional selectivity, whereas in the Simon task, spatial attention can be expected to play a larger role. It is possible that these two aspects of executive attention differentially influence congruency-dependent error processing.

For the following trial stimulus-locked P1 (Fig. 4), a $2 \times 2 \times 2$ (Laterality by RSI by Previous Accuracy) repeated measures ANOVA revealed a main effect of laterality ($F(1,20) = 5.314$, $p = .032$, $\eta_p^2 = 0.210$), in which ipsilateral P1s ($M = 18.261$; $SE = 2.263$) were larger than contralateral P1s ($M = 13.812$; $SE = 2.342$), and a main effect of accuracy

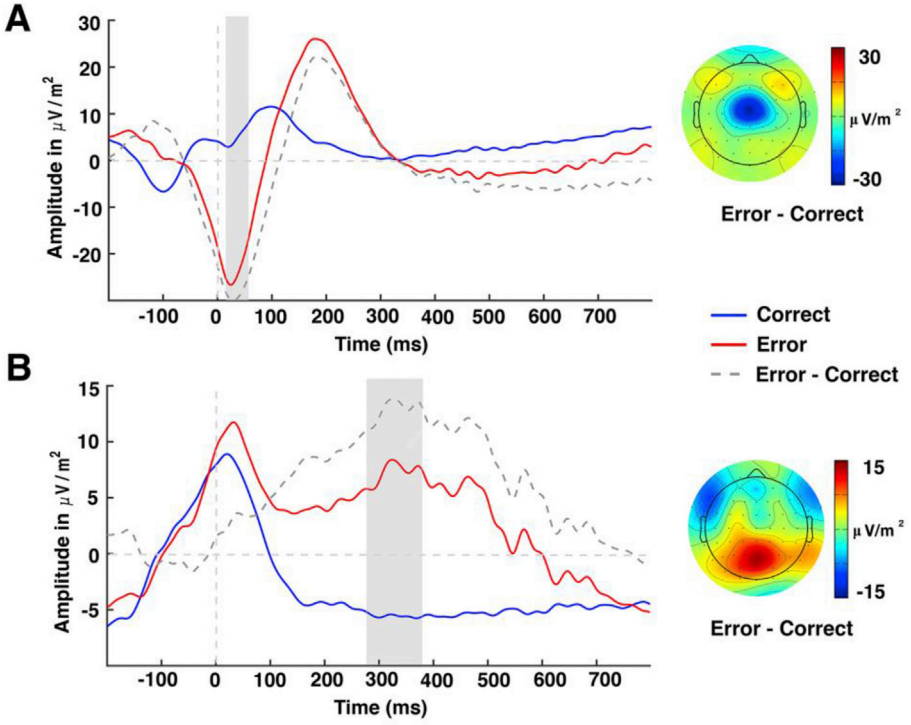

Fig. 3. Response-locked ERP activity. ERPs and topographic plots for (***A***) the ERN (at electrode Cz) and (***B***) the Pe (at electrode POz). The analysis window for each component is designated by the shaded box. The topographic plots signify the amplitude of the error - correct difference wave throughout the analysis window.

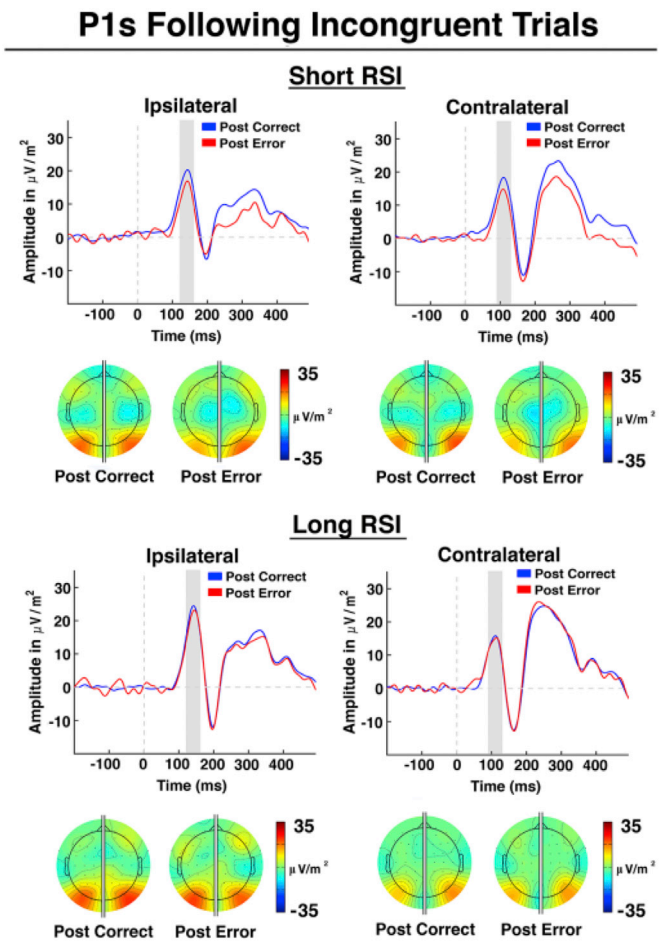

Fig. 4. Changes in Subsequent Trial P1 as a Function of Previous Accuracy and RSI Duration. P1 ERP components and topographic plots for the both ipsilateral and contralateral P1s following incongruent trials, for short and long RSIs. The analysis windows for the ipsilateral and contralateral P1 components are designated by the shaded boxes. Topographic plots are bisected given that the data for each hemisphere are comprised of responses to ipsilaterally or contralaterally presented stimuli.

($F(1,20) = 11.292$, $p = .003$, $\eta_p^2 = 0.361$), in which post-correct P1s ($M = 17.106$; $SE = 2.069$) were larger than post-error P1s ($M = 14.966$; $SE = 2.160$). Critically, there was also an accuracy by RSI interaction ($F(1,20) = 7.297$, $p = .014$, $\eta_p^2 = 0.267$), in which post-correct P1s ($M = 16.915$; $SE = 2.178$) were larger than post-error P1s ($M = 13.239$; $SE = 2.375$) at short RSIs ($p = .001$, $d = 0.345$), but were not significantly different (post-corrects: $M = 17.297$, $SE = 2.159$; post-errors: $M = 16.693$, $SE = 2.145$) at long RSIs ($p = .395$). Lastly, there was a laterality by RSI interaction ($F(1,20) = 25.630$, $p < .001$, $\eta_p^2 = 0.562$), in which ipsilateral P1s ($M = 20.556$; $SE = 2.355$) were larger than contralateral P1s ($M = 13.435$; $SE = 2.291$) ($p = .001$, $d = 0.669$) at long RSIs, but did not differ (ipsilateral: $M = 15.966$, $SE = 2.430$; contralateral: $M = 14.189$, $SE = 2.495$) at short RSIs ($p = .410$). No other main effects or interactions reached significance (all $p > .05$). The findings were comparable when limiting the subsequent trial to correct trials (see supplemental material; Fig. S3).

Although it may seem counterintuitive that ipsilateral P1s were larger than contralateral P1s, we note that the majority of studies that report larger contralateral P1s use lateralized stimulus presentation to investigate the component using spatial cuing paradigms (Hillyard and Anllo-Vento, 1998). When the P1 is investigated *without* the use of spatial cues, similar patterns of larger ipsilateral P1s have been observed (Di Russo et al., 2002).

We conducted a control analysis (Fig. 5) comparing current

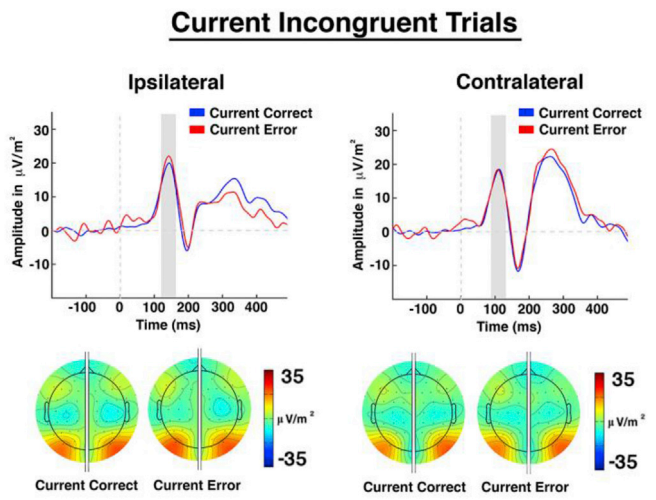

Fig. 5. Current Trial P1 Control Analysis. Current trial P1 ERP components and topographic plots at short RSIs for both ipsilateral and contralateral P1s. The analysis windows for the ipsilateral and contralateral P1 components are designated by the shaded boxes. Topographic plots are bisected given that the data for each hemisphere are comprised of responses to ipsilaterally or contralaterally presented stimuli.

incongruent trial correct and error P1s at short RSIs, in which a 2 [Laterality (Ipsilateral, Contralateral)] by 2 [Accuracy (Correct, Error)] repeated measures ANOVA failed to reveal any significant differences (all $p > .214$). This indicates that the relationship between the current trial ERN and subsequent trial P1s are not carry over effects from lapses in stimulus processing on the previous trial and suggest that there are not any effects of fatigue on stimulus processing.

2.3. Single trial analysis

The model of ERN and RSI on next trial P1 revealed an effect of RSI (estimate=0.051, SE=0.010, df=9218, t=5.054, $p < .001$), in which long RSIs produced larger P1s relative to short RSIs, and an effect of laterality (estimate=0.121, SE=0.020, df=9216, t=6.010, $p < .001$), in which ipsilateral P1s were larger than contralateral P1s. Critically, there was also a significant interaction in which ERN amplitude predicted P1 amplitude as a function of RSI (estimate=−0.022, SE=0.010, df=9219, t=−2.175, p=.030; Fig. 6). Although the simple slopes of the ERN at one standard deviation *above* the mean (corresponding to long RSIs) failed to yield a significant difference from zero ($p = .562$), analysis of the simple slopes of the ERN at one standard deviation *below* the mean (corresponding to short RSIs) was indeed significant ($p = .014$). Therefore, the nature of this interaction was such that a larger ERN led to reductions in the P1 at short, but not long, RSIs. The model of Pe and RSI on next trial P1 revealed an effect of RSI (estimate=0.050, SE=0.010, df=9218, t=4.996, $p < .001$), in which longer RSIs were associated with larger P1s, and an effect of laterality (estimate=0.121, SE=0.020, df=9216, t=6.022, $p < .001$), in which ipsilateral P1s were larger than contralateral P1s. The findings were comparable when limiting the subsequent trial to correct trials, with the exception that the simple slopes were not significantly different from zero (see supplemental material; Fig. S4).

It is possible that the observed effects (that favor the ERN rather than the Pe) were because of differences in variance for each component. In order to rule this out, we calculated the standard deviations for the ERN and Pe (across trials) and conducted a paired samples t-test to investigate if they were significantly different. We failed to observe significant a difference between the ERN and Pe ($p > .05$), which suggests that the present findings are not due to task-dependent differences in variance for each component.

The model of ERN and RSI on next trial RT revealed an effect of RSI

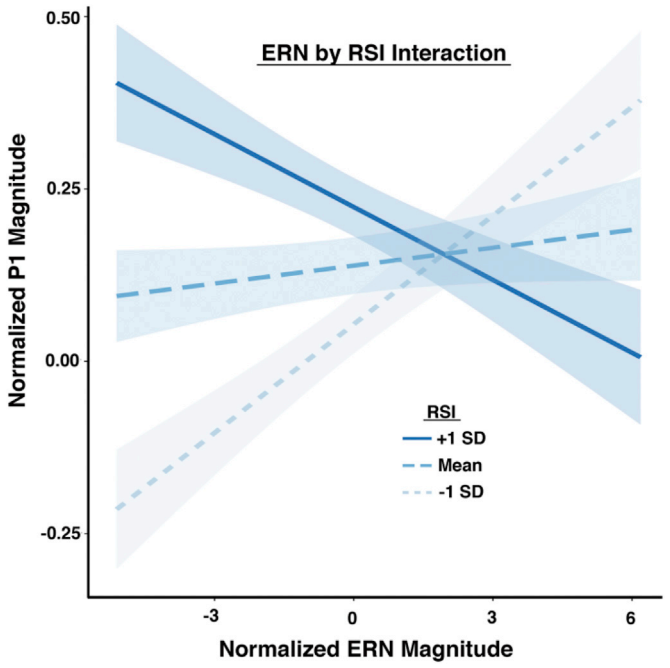

Fig. 6. Single Trial Relationship between ERN (on Incongruent Trials) and RSI on Subsequent Trial P1. Predicted values for the post-error P1, relative to ERN magnitude and RSI duration (as continuous variables). The influence of ERN magnitude on subsequent trial P1 differs at short (−1 SD) compared to long (+1SD) RSI durations.

(estimate=−0.158, SE=0.014, df=4600, t=−11.085, p < .001), in which next trial RT was faster for trials with longer RSIs. The model of ERN and RSI on next trial accuracy revealed an effect of RSI (estimate=0.143, SE=0.035 z=4.072, p < .001), in which next trial accuracy was higher for trials with longer RSIs. The model of Pe and RSI on next trial RT revealed an effect of RSI (estimate=−0.158, SE=0.014, df=4600, t=−11.095, p < .001), in which faster next trial RTs were associated with longer RSIs. The model of Pe and RSI on the likelihood of next trial accuracy revealed an effect of RSI (estimate=0.144, SE=0.035, z=4.109, p < .001), in which next trial accuracy was higher only for trials in which RSIs were longer. These findings were comparable when limiting the subsequent trial to correct trials (see supplemental material).

3. Discussion

The present study demonstrates that error-induced reductions in task relevant sensory processing, as they relate to data limited errors (Buzzell et al., 2017), are generalizable to speeded response errors. In concordance with previous findings, we found that errors were associated with reduced sensory processing (as indexed by the P1) on the following trial at short (200–533 ms), but not long (866–1200 ms) RSIs, which suggests a bottleneck for cognitive resources (Jentzsch and Dudschig, 2009) when error processing and subsequent stimulus processing overlap. Moreover, at the single-trial level, we found that the P1 was differentially affected by the ERN as a function of RSI, which provides additional evidence for a relationship between error processing and subsequent stimulus processing. This pattern of findings is largely consistent with recent theoretical views on the complex neural dynamics that follow commission of an error (Ullsperger and Danielmeier, 2016; Wessel, 2017). Additionally, whereas our prior work demonstrated that the Pe was predictive of P1 amplitude on the following trial, in the present study, we found that only the ERN was predictive of subsequent sensory processing. Collectively, the findings suggest that although data-limited errors and speeded-response errors both impact subsequent sensory processing, they do so via different stages of the error-processing cascade.

The observed behavioral results are consistent with previous studies investigating the influence of RSI on post-error compensations, such that there were *relative* increases in PES and decreases in PEA at short compared to long RSIs (Buzzell et al., 2017; Houtman and Notebaert, 2013; Jentzsch and Dudschig, 2009; Van der Borght et al., 2016). In addition, the observed electrophysiological findings support previous work that demonstrates reductions in sensory component amplitudes following errors at short compared to long RSIs (Buzzell et al., 2017; Van der Borght et al., 2016). The present findings, as well as the findings from our previous study, suggest that the performance monitoring system requires time for optimal functionality, in line with recent theoretical views (Ullsperger and Danielmeier, 2016; Wessel, 2017). The primary novel finding of the present study however, is that the magnitude of processing for speeded response errors (as indexed by the ERN, not the Pe) predicts modulation of subsequent sensory processing (as indexed by the P1). This relationship was illustrated by the single-trial modulation of post-error P1 magnitude as a function of ERN magnitude and RSI duration. This finding supports the idea that, for speeded response errors, post-error cognition is impacted by an early stage of the error-processing cascade responsible for automatic suppression of ongoing activity (Wessel, 2017). This stands in contrast to our previous finding that, for data-limited errors, post-error cognition is impacted by a later, deliberative stage of error processing. These results collectively suggest that within the context of speeded response errors, the neural processing indexed by the ERN—and not the later and more deliberative processing indexed by the Pe—predicts modulation of sensory processing on post-error trials.

An additional question with regard to interpreting the relationship between the ERN and next-trial sensory processing is that if there is indeed a bottleneck for cognitive resources at short RSIs, then one might expect that sensory processing may also impact error processing. It is possible, for example, that error processing may be diminished as a result of cognitive resources being devoted to subsequent sensory processing when the two processes overlap in time (see supplemental material). Analysis of the ERN and Pe revealed that the ERN was not affected as a function of subsequent trial RSI. However, the Pe was smaller at short, as compared to long RSIs. Critically, this deficit for the Pe was present only on error trials. These results suggest that the Pe (which is thought to index the more deliberative, resource intensive processing of an error) is impacted as a result of a bottleneck for cognitive resources. It is important to clarify however, that although the Pe was modulated as a function of next trial RSI, the main finding of the present study is that the ERN, not the Pe, predicts modulation of the P1 on the following trial. Thus, the impact of RSI on the Pe appears to be orthogonal to the influence of the ERN on post-error sensory processing.

Although we observed an interaction between ERN amplitude and post-error behavior as a function of RSI at the group level, we did not observe a comparable relationship at the single-trial level. We note that it is possible that separable antagonistic processes immediately follow an error. For instance, Purcell and Kiani (2016) proposed that PES is associated with two different processes: (1) modulation of the decision boundary when selecting a response and (2) decrements in sensory evidence accumulation. In other words, PES reflects an *adaptive* increase of the decision boundary (initiating motor inhibition) as well as *maladaptive* reductions in sensitivity to incoming evidence (sensory suppression). In a follow-up commentary by Ullsperger and Danielmeier (2016), they note that a potential reason why the literature observes a wide range of results for PES is because the adaptive and maladaptive mechanisms counteract each other to a different extent depending on the type of task. Perhaps in this case, the adaptive motor inhibition and maladaptive sensory suppression cancelled each other out. This idea is supported by research demonstrating that, depending on the circumstances of an error (perceptual/motor, aware/not aware), there are disparities as to how the system will compensate (Navarro-Cebrian et al., 2013). In addition, an emerging view is that errors (Ullsperger and Danielmeier, 2016; Wessel, 2017) or other unexpected events (Wessel and Aron, 2017) cause a short-lived global inhibition of the cognitive system, but that with ample

time between trials, more deliberative processing can yield improvements on the next trial; the current results are consistent with this hypothesis.

In order to clarify the role of the ERN and Pe as predictors of subsequent reductions in sensory processing as a function of speeded response errors and data limited errors respectively, we contrasted the components from the present study with the components observed during our previous study (Buzzell et al., 2017), which were elicited within the context of stimulus ambiguity. Critically, for the ERN, we found that the Simon task employed here yielded a larger ERN effect size (d = 2.23), compared to the ERN in the perceptual decision-making task (d = 0.51) employed in our previous study. Moreover, within the current Simon task, a larger effect size was observed for the ERN (d = 2.23), compared to the Pe (d = 1.231). In direct contrast, the perceptual decision-making task of our previous study yielded a larger effect size for the Pe (d = 0.66), compared to the ERN (d = 0.51). Please note that here, the method for calculating Cohen's *d* differs from the approach employed by Buzzell et al. (2017). For the current report, we leveraged the method suggested by Dunlap et al., (1996) when calculating the ERN and Pe effect sizes for both studies, as simulations demonstrate that this approach yields less distortion in the estimation of Cohen's *d* (Dunlap et al., 1996). We also note that peak latency of the Pe for speeded-response errors in the Simon task is nearly 100 ms earlier in comparison to the Pe observed for data-limited errors in the perceptual decision-making task (Buzzell et al., 2017). Similar to how stimulus-locked P3 latency indexes stimulus evaluation time (Kutas et al., 1977), perhaps the latency shift in the Pe (Ullsperger et al., 2014a,b) implies a shorter duration of more deliberative aspects of error processing for speeded response errors.

The present study (as well as our previous study; Buzzell et al., 2017) observed error-dependent modulation of the P1, while other similar work investigating this phenomenon (Van der Borght et al., 2016) observed effects for the N1. It is important to note that recent work provides compelling evidence that diminishment of the P1 indexes active attentional suppression (Moher et al., 2014; Slagter et al., 2016). In addition, previous work from our lab has demonstrated that the N1 can be enhanced by top down attention, and that this component is diminished when preceded by attentional lapses (Fedota et al., 2012; Roberts et al., 2014). We propose that error-related diminishment of P1 likely resulted from active suppression of task relevant stimuli, whereas the diminishment of N1 observed by Van der Borght et al. (2016) likely reflects a lapse in attention to task irrelevant stimuli when transitioning to an orthogonal task.

One limitation of the present study is that, in order to attain enough errors at short RSIs (200–533 ms) that would not be contaminated with corrections from the previous trial, participants were instructed not to correct for their responses and any trials in which a response was provided prior to 150 ms were discarded from all analyses. An unavoidable consequence of instructing participants to not correct for their errors is that it could have introduced additional cognitive load. Indeed, participants might be more likely to spontaneously correct their responses in a response conflict task. As they were not permitted to correct their responses, greater suppression of ongoing activity might have been required than what would normally have been the case during a Simon task without such an instruction. Future research may wish to investigate the role of response corrections as it relates to cognitive load and post-error reductions in sensory processing. Another potential explanation for these results that we are not able to rule out is that underlying phase and amplitude shifts of induced activity (such as post-error theta or post-stimulus alpha) could drive such results. Future research may wish to investigate the impact of rhythmic brain activity as a function of manipulating the degree of overlap between error processing and subsequent stimulus processing.

The present findings demonstrate that error processing in the context of speeded response errors can modulate task-relevant sensory processing on the following trial. More specifically, the magnitude of error processing, as indexed by the ERN, predicts a reduction of the magnitude of task-relevant sensory processing, as indexed by the P1, on subsequent trials. These results suggest that error-induced reductions in task relevant sensory processing can be generalized beyond tasks where sensory inputs are ambiguous and that error commission can lead to failures of the performance monitoring system. Together, the present results and the results from our previous study, allow for a more mechanistic understanding of how error processing leads to distraction, and how different stages of error processing can drive such distraction depending on the context in which the error was committed.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.neuroimage.2018.08.009.

References

Bates, D., Mächler, M., Bolker, B., Walker, S., 2014. Fitting Linear Mixed-effects Models Using Lme4. ArXiv:1406.5823 [Stat]. Retrieved from. http://arxiv.org/abs/1406.5823.

Botvinick, M.M., Braver, T.S., Barch, D.M., Carter, C.S., Cohen, J.D., 2001. Conflict monitoring and cognitive control. Psychol. Rev. 108 (3), 624–652.

Brainard, D.H., 1997. The psychophysics toolbox. Spatial Vis. 10 (4), 433–436.

Buzzell, G.A., Beatty, P.J., Paquette, N.A., Roberts, D.M., McDonald, C.G., 2017. Error-induced blindness: error detection leads to impaired sensory processing and lower accuracy at short response–stimulus intervals. J. Neurosci. 37 (11), 2895–2903. https://doi.org/10.1523/JNEUROSCI.1202-16.2017.

Delorme, A., Makeig, S., 2004. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. J. Neurosci. Meth. 134, 9–21.

Di Russo, F., Martínez, A., Sereno, M.I., Pitzalis, S., Hillyard, S.A., 2002. Cortical sources of the early components of the visual evoked potential. Hum. Brain Mapp. 15 (2), 95–111. https://doi.org/10.1002/hbm.10010.

Dunlap, W.P., Cortina, J.M., Vaslow, J.B., Burke, M.J., 1996. Meta-analysis of experiments with matched groups or repeated measures designs. Psychol. Meth. 170–177.

Fedota, J.R., McDonald, C.G., Roberts, D.M., Parasuraman, R., 2012. Contextual task difficulty modulates stimulus discrimination: electrophysiological evidence for interaction between sensory and executive processes. Psychophysiology 49 (10), 1384–1393. https://doi.org/10.1111/j.1469-8986.2012.01455.x.

Hillyard, S.A., Anllo-Vento, L., 1998. Event-related brain potentials in the study of visual selective attention. Proc. Natl. Acad. Sci. Unit. States Am. 95 (3), 781–787. https://doi.org/10.1073/pnas.95.3.781.

Houtman, F., Notebaert, W., 2013. Blinded by an error. Cognition 128 (2), 228–236. https://doi.org/10.1016/j.cognition.2013.04.003.

Jentzsch, I., Dudschig, C., 2009. Why do we slow down after an error? Mechanisms underlying the effects of posterror slowing. Q. J. Exp. Psychol. 62 (2), 209–218. https://doi.org/10.1080/17470210802240655.

Kayser, J., Tenke, C.E., 2006. Principal components analysis of Laplacian waveforms as a generic method for identifying ERP generator patterns: II. Adequacy of low-density estimates. Clin. Neurophysiol. 117 (2), 369–380. https://doi.org/10.1016/j.clinph.2005.08.033.

King, J.A., Korb, F.M., Cramon, D. Y. von, Ullsperger, M., 2010. Post-error behavioral adjustments are facilitated by activation and suppression of task-relevant and task-irrelevant information processing. J. Neurosci. 30 (38), 12759–12769. https://doi.org/10.1523/JNEUROSCI.3274-10.2010.

Kleiner, M., Brainard, D.H., Pelli, D., Ingling, A., Murray, R., Broussard, C., 2007. What's new in Psychtoolbox-3. Perception 36, 1–16. https://doi.org/10.1068/v070821.

Kutas, M., McCarthy, G., Donchin, E., 1977. Augmenting mental chronometry: the P300 as a measure of stimulus evaluation time. Science 197 (4305), 792–795. https://doi.org/10.1126/science.887923.

Kuznetsova, A., Brockhoff, P.B., Christensen, R.H.B., 2016. Package "lmerTest." R Package Version, 2. Retrieved from. http://cran.uib.no/web/packages/lmerTest/lmerTest.pdf.

Laming, D., 1979. Choice reaction performance following an error. Acta Psychol. 43 (3), 199–224. https://doi.org/10.1016/0001-6918(79)90026-X.

Lopez-Calderon, J., Luck, S.J., 2014. ERPLAB: an open-source toolbox for the analysis of event-related potentials. Front. Hum. Neurosci. 8. https://doi.org/10.3389/fnhum.2014.00213.

Maier, M.E., Yeung, N., Steinhauser, M., 2011. Error-related brain activity and adjustments of selective attention following errors. Neuroimage 56 (4), 2339–2347. https://doi.org/10.1016/j.neuroimage.2011.03.083.

Moher, J., Lakshmanan, B.M., Egeth, H.E., Ewen, J.B., 2014. Inhibition drives early feature-based attention. Psychol. Sci. 25 (2), 315–324. https://doi.org/10.1177/0956797613511257.

Navarro-Cebrian, A., Knight, R.T., Kayser, A.S., 2013. Error-monitoring and post-error compensations: dissociation between perceptual failures and motor errors with and without awareness. J. Neurosci.: The Official Journal of the Society for Neuroscience 33 (30), 12375–12383. https://doi.org/10.1523/JNEUROSCI.0447-13.2013.

Nieuwenhuis, S., Ridderinkhof, K.R., Blom, J., Band, G.P.H., Kok, A., 2001. Error-related brain potentials are differentially related to awareness of response errors: evidence from an antisaccade task. Psychophysiology 38 (5), 752–760. https://doi.org/10.1111/1469-8986.3850752.

Notebaert, W., Houtman, F., Opstal, F.V., Gevers, W., Fias, W., Verguts, T., 2009. Post-error slowing: an orienting account. Cognition 111 (2), 275–279. https://doi.org/10.1016/j.cognition.2009.02.002.

Pelli, D.G., 1997. The VideoToolbox software for visual psychophysics: transforming numbers into movies. Spatial Vis. 10 (4), 437–442.

Purcell, B.A., Kiani, R., 2016. Neural mechanisms of post-error adjustments of decision policy in parietal cortex. Neuron 89 (3), 658–671. https://doi.org/10.1016/j.neuron.2015.12.027.

R Core Team, 2016. R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing, Vienna, Austria. URL. http://www.R-project.org/.

Rabbitt, 1979. How old and young subjects monitor and control responses for accuracy and speed. Br. J. Psychol. 70 (2), 305–311. https://doi.org/10.1111/j.2044-8295.1979.tb01687.x.

Ridderinkhof, K.R., Ullsperger, M., Crone, E.A., Nieuwenhuis, S., 2004. The role of the medial frontal cortex in cognitive control. Science 306 (5695), 443–447. https://doi.org/10.1126/science.1100301.

Roberts, D.M., Fedota, J.R., Buzzell, G.A., Parasuraman, R., McDonald, C.G., 2014. Prestimulus oscillations in the alpha band of the EEG are modulated by the difficulty of feature discrimination and predict activation of a sensory discrimination process. J. Cognit. Neurosci. 26 (8), 1615–1628. https://doi.org/10.1162/jocn_a_00569.

Scheffers, M.K., Coles, M.G., 2000. Performance monitoring in a confusing world: error-related brain activity, judgments of response accuracy, and types of errors. J. Exp. Psychol. Human Perception and Performance 26 (1), 141–151.

Shenhav, A., Botvinick, M.M., Cohen, J.D., 2013. The expected value of control: an integrative theory of anterior cingulate cortex function. Neuron 79 (2), 217–240. https://doi.org/10.1016/j.neuron.2013.07.007.

Simon, J.R., 1969. Reactions toward the source of stimulation. J. Exp. Psychol. 81 (1), 174–176.

Slagter, H.A., Prinssen, S., Reteig, L.C., Mazaheri, A., 2016. Facilitation and inhibition in attention: functional dissociation of pre-stimulus alpha activity, P1, and N1 components. Neuroimage 125, 25–35. https://doi.org/10.1016/j.neuroimage.2015.09.058.

Steinhauser, M., Yeung, N., 2010. Decision processes in human performance monitoring. J. Neurosci.: The Official Journal of the Society for Neuroscience 30 (46), 15643–15653. https://doi.org/10.1523/JNEUROSCI.1899-10.2010.

Ullsperger, M., Danielmeier, C., 2016. Reducing speed and sight: how adaptive is post-error slowing? Neuron 89 (3), 430–432. https://doi.org/10.1016/j.neuron.2016.01.035.

Ullsperger, M., Danielmeier, C., Jocham, G., 2014a. Neurophysiology of performance monitoring and adaptive behavior. Physiol. Rev. 94 (1), 35–79. https://doi.org/10.1152/physrev.00041.2012.

Ullsperger, M., Fischer, A.G., Nigbur, R., Endrass, T., 2014b. Neural mechanisms and temporal dynamics of performance monitoring. Trends Cognit. Sci. 18 (5), 259–267. https://doi.org/10.1016/j.tics.2014.02.009.

Van der Borght, L., Schevernels, H., Burle, B., Notebaert, W., 2016. Errors disrupt subsequent early attentional processes. PLoS One 11 (4) e0151843. https://doi.org/10.1371/journal.pone.0151843.

Wessel, J.R., 2017. An adaptive orienting theory of error processing. Psychophysiology n/a-n/a. https://doi.org/10.1111/psyp.13041.

Wessel, J.R., Aron, A.R., 2017. On the globality of motor suppression: unexpected events and their influence on behavior and cognition. Neuron 93 (2), 259–280. https://doi.org/10.1016/j.neuron.2016.12.013.

Winkler, I., Debener, S., Müller, K.R., Tangermann, M., 2015. On the influence of high-pass filtering on ICA-based artifact reduction in EEG-ERP. In: 2015 37th Annual International Conference of the IEEE Engineering in Medicine and Biology Society (EMBC), pp. 4101–4105. https://doi.org/10.1109/EMBC.2015.7319296.

Yeung, N., Botvinick, M.M., Cohen, J.D., 2004. The neural basis of error detection: conflict monitoring and the error-related negativity. Psychol. Rev. 111 (4), 931–959. https://doi.org/10.1037/0033-295X.111.4.931.