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Testing the Bottleneck Account for Post-Error Slowing Beyond the Post-Error Response

Dmitri Lavro[1✉], Mattan S. Ben Shachar[1], Christopher W.N. Saville[2], Christoph Klein[3,4,5], and Andrea Berger[1]

[1] Department of Psychology and Zlotowski Center for Neuroscience, Ben-Gurion University of the Negev, Israel

[2]School of Psychology, Bangor University, United Kingdom

[3]Department of Child and Adolescent Psychiatry, Psychotherapy, and Psychosomatics, Medical Faculty, University of Freiburg, Germany

[4]Department of Child and Adolescent Psychiatry, Medical Faculty, University of Cologne, Germany

[5]Department of Psychiatry, National and Kapodistrian University of Athens, Eginition Hospital, Greece

Author Note

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Correspondence should be sent to: Dmitri Lavro, Department of Psychology and Zlotowski Center for Neuroscience, Ben-Gurion University of the Negev, Beer Sheva, Israel 84105-01. E-mail: lavro@post.bgu.ac.il

Word count: 6358

Abstract

The bottleneck account for post-error slowing assumes that cognitive resources are depleted after errors and thus the processing of subsequent events is delayed. To test this, we used a novel speeded-choice task and recorded behavioral measures and ERP (event-related potential) components on five trials following either an erroneous or correct response. We found that participants were slower and less accurate immediately after making an error and that this reduction of performance decayed on the following trials. Moreover, post-correct versus post-error differences in both the visual N1 and the P3 component were found. However, the difference in the P3 component rapidly diminished over time, whereas the differences in the N1 component were still evident in the fourth trial following the erroneous response. The results lay further support to the bottleneck account for post-error slowing and show a combination of early attentional and higher-order processing changes that occur after erroneous responses.

Keywords: error; error-related negativity; ERN; post-error accuracy; post-error adjustments; post-error performance; error monitoring; error detection; post-error slowing; PES; error processing; bottleneck account; N1; P3; N100; P300; attention; cognitive control.
Abstract Word Count: 149

Testing the Bottleneck Account for Post-Error Slowing Beyond the Post-Error Response

An integral part of human behavior is making mistakes, which can sometimes lead to unwanted or even devastating consequences. Therefore, the ability to detect and monitor our errors is crucial for survival and success. In speeded response time (RT) tasks, the presence of response monitoring can be inferred from the post-error slowing (PES) effect (Laming, 1979; Rabbitt, 1966). PES reflects the increase in RT in trials following the commission of an error. However, in spite of the robustness of the PES effect across numerous studies and contexts, the reason for and the interpretation of the effect continues to be debated (Danielmeier & Ullsperger, 2011; Schroder & Moser, 2014).

Two of the most frequently discussed explanations for the PES effect are the cognitive control account and the orienting account. Arguably, they represent the extremes of functional and non-functional accounts for PES. On this axis, cognitive control operations and top-down behavioral adjustments after errors are linked to functional explanations, whereas non-strategic, early attentional interference after errors is usually linked to a non-functional explanation for PES. The cognitive control account (Botvinick, Braver, Barch, Carter, & Cohen, 2001) argues that PES is the outcome of control up-regulation after errors and therefore relies on the prediction that, after making an error, participants will respond more slowly but more accurately. The somewhat opposing view for the functionality of the PES effect is the orienting account (Notebaert et al., 2009), which does not relate PES to a control mechanism, but rather to a shift in attention created by an unusual event (e.g., the error). Perhaps the most obvious way to determine which explanation is correct is to look at post-error response accuracy. However,

many studies show inconsistent results regarding post-error accuracy. Some studies show more accurate responses after incorrect responses (compared to correct responses), supporting a strategic change in speed-accuracy trade-off (Chiu & Deldin, 2007; Desmet et al., 2012; Ridderinkhof, van den Wildenberg, Wijnen, & Burle, 2004; Saunders & Jentzsch, 2012; Van der Borght, Desmet, & Notebaert, 2015), whereas other studies show that performance is *less* accurate after the commission of an error (Castellar, Kühn, Fias, & Notebaert, 2010; Danielmeier & Ullsperger, 2011; Hajcak, McDonald, & Simons, 2003; Notebaert et al., 2009), which is consistent with the non-functional explanation for PES.

Another explanation for PES that has gained support in recent years is the bottleneck account for the PES effect. This explanation focuses on the depletion of limited cognitive resources after errors, resulting in a bottleneck in the processing of subsequent events (Buzzell, Beatty, Paquette, Roberts, & McDonald, 2017; Dudschig & Jentzsch, 2009; Hochman & Meiran, 2005; Houtman & Notebaert, 2013; Jentzsch & Dudschig, 2009; Lavro & Berger, 2015). The studies that have investigated the bottleneck account using behavioral measures suggest that the processing bottleneck has an impact mainly on central cognitive stages of processing. Specifically, these studies suggest that PES is the outcome of depleted central resources in the response-selection phase of the trial following an error (Dudschig & Jentzsch, 2009; Hochman & Meiran, 2005; Jentzsch & Dudschig, 2009; Lavro & Berger, 2015). However, recent studies with the event-related potential (ERP) technique in visual discrimination tasks suggest that the bottleneck created by the error signal mainly influences early attentional processes, in a pattern that also fits the orienting account (Buzzell et al., 2017; Van der Borght, Schevernels, Burle, & Notebaert, 2016).

ERP measures have been widely used in error-monitoring research. Perhaps the most well-known ERP component associated with error detection is error-related negativity (ERN), which peaks at frontocentral sites and can be seen roughly 50 ms to 100 ms after an error has been made (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The source of the error-related activity is believed to be the anterior cingulate cortex, a brain region that is highly involved in performance monitoring and cognitive control (Botvinick et al., 2001; Botvinick, Cohen, & Carter, 2004; Gehring & Fencsik, 2001; Kerns et al., 2004; Kiehl, Liddle, & Hopfinger, 2000; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003; Mathalon, Whitfield, & Ford, 2003; Rushworth, Walton, Kennerley, & Bannerman, 2004). It is therefore surprising that ERN and PES do not always correlate, which may suggest that they represent overlapping but different aspects of the error-processing mechanism, such as the trigger for adjustment and the actual post-error adjustment (Danielmeier & Ullsperger, 2011; Dudschig & Jentzsch, 2009; Schroder & Moser, 2014).

Compared to the wide range of studies that have investigated ERN, less effort has been devoted to the post-error ERP investigation (e.g., Buzzell et al., 2017; Van der Borght et al., 2016). To our knowledge, no study has looked beyond the first trial after the error, which could potentially reveal important insights on the time course of the error-related bottleneck effect, which is critical for this account.

Therefore, in this study, we examined behavioral and ERP measures on the first trial (i.e., trial N+1) and second trial (i.e., trial N+2) following an error in a speeded-choice task. Similarly to previous studies that investigated post-error effects, we were interested in the RT and accuracy of post-error trials (Dudschig & Jentzsch, 2009; Hochman & Meiran, 2005; Jentzsch & Dudschig, 2009). In line with the bottleneck account for PES, we hypothesized that the responses

would be slower and less accurate after errors compared to post-correct responses and that both effects would be attenuated on trial N+2, showing that the bottleneck caused by the error diminishes over time. Although this prediction differentiates between the cognitive control account for PES and the other mentioned accounts, it does not distinguish between the bottleneck account and the orienting account for PES because both – from a different perspective – predict the effect of slower and less accurate responses after errors, which can be attenuated with time. Therefore, to differentiate between the bottleneck and the orienting accounts, we also used the ERP technique. In contrast to behavioral measures that reflect the output of many cognitive processes, the ERP measures provided us with a continuous measure of processing before and after the response and allowed us to focus on different stages of processing that could be affected by the error (Luck, 2005).

Specifically, we focused on the ERP components that differentiate early attentional versus higher-order central processing (Buzzell et al., 2017; Van der Borght et al., 2016). Namely, the N1 component was selected as an electrophysiological marker for an early discrimination process (Vogel & Luck, 2000) and the P3 component as a representative of the central processes of decision and memory updating (Kok, 2001; Polich, 2007). We expected that the pattern of behavioral results would also be reflected in the ERP components. As outlined above, different views exist regarding the exact stage in which the post-error bottleneck occurs. Accordingly, a difference between post-correct and post-error amplitude in the visual N1 component would support the orienting account and the mentioned ERP studies, demonstrating that errors affect early attentional processes (Buzzell et al., 2017; Van der Borght et al., 2016). On the other hand, post-correct and post-error amplitude differences in the P3 component would support the mentioned behavioral studies of the bottleneck effect, showing that central cognitive

stages of processing are affected by errors (Hochman & Meiran, 2005; Jentzsch & Dudschig, 2009; Lavro & Berger, 2015). Furthermore, if both or any of the ERP components were related to the post-error performance – consistent with the general logic of the bottleneck account for the PES – we would expect that the differences in amplitude between post-correct and post-error trials would be attenuated on trial N+2 when compared to trial N+1.

Method

Participants

Thirty undergraduate students at Ben-Gurion University of the Negev participated in the experiment. Ethical approval was obtained from the Ethics Committee at Ben-Gurion University of the Negev. Sample size was chosen to be comparable to previous studies that reported the behavioral post-error bottleneck effects (Dudschig & Jentzsch, 2009; Hochman & Meiran, 2005; Houtman & Notebaert, 2013; Jentzsch & Dudschig, 2009; Lavro & Berger, 2015) and, based on the existing literature, to provide an adequate signal-to-noise ratio (SNR) for visual N1 (Vogel & Luck, 2000), P3 (Kok, 2001) and ERN (Steele et al., 2016) components. All participants reported to be right-handed, healthy, with no history of neurological illness, and had normal or corrected-to-normal vision. Participants gave informed consent and participated in the study as partial fulfillment of course requirements. Data from 27 of the 30 participants (22 females and 15 males) were retained for analysis, and data for the other three participants were discarded due to poor data quality or insufficient artifact-free error trials (i.e., less than six; Steele et al., 2016) for signal averaging. The mean age of the remaining sample was 25 years (SD = 3.42).

Apparatus and Stimuli

A 17″ LCD computer monitor was used to present visual stimuli. Green arrows on a black background were presented to the participants in the middle of the screen; the width of the

arrow did not exceed the visual angle of 1º, assuming a viewing distance of 60 cm. Responses were recorded by a four-key response box that was connected to a serial port. The box was placed on a desk in front of the participant; all participants responded with the index finger of each hand. The experiment was programmed with E-prime 2.0 software (Schneider, Eschman, & Zuccolotto, 2002).

Procedure

Participants performed a new task that combined congruent and incongruent stimulus-response conditions with a one-back condition. In this task – which we named the *congruency one-back task* (COBT) – participants saw arrows pointing either left or right and were asked to respond with one hand if each arrow matched the previous arrow in the sequence and with the other hand if it did not. Trials could thus be a repeat requiring a response congruent with the direction of the arrow, a repeat requiring an incongruent response, a non-repeat requiring a congruent response, or a non-repeat requiring an incongruent response. The hand used to indicate the repeat versus non-repeat decision was counterbalanced across participants. Stimuli were presented for 83 ms, followed by an intertrial interval (ITI) of 1617 + 100/150/200 ms jitter, to eliminate stimulus anticipation ERP components (Tecce, 1972). All trial types were equiprobable and presented in pseudorandom order (Figure 1). Each experimental session started with 10 practice trials to ensure that the participants understood the task. The participants could choose to continue to practice until they were sure that they understood task instructions. The rest of the experiment contained four experimental blocks, each with 225 trials, resulting in a total of 900 experimental trials.

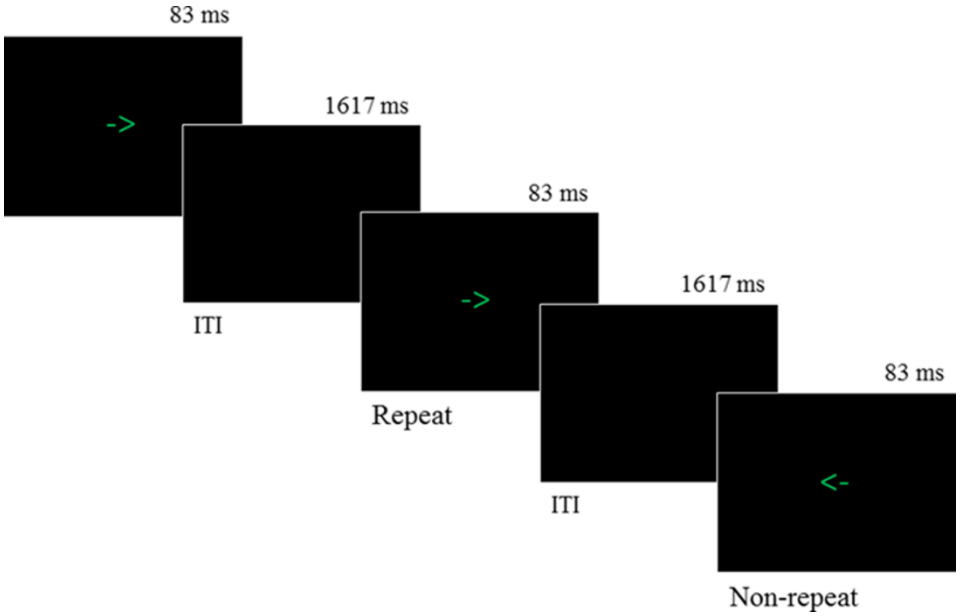

Figure 1. Trial design for the COBT. The five panels from top-left to bottom-right show an example of a few standard trials.

Behavioral Analysis

Responses faster than 100 ms (0.1% of trials) were excluded from data analysis. On the remaining data, accuracy (proportion of trials responded to correctly) and mean reaction time (MRT) for correct trials were computed. For trial N+1, PES was calculated as $RT_{\text{trial N:error}}$ - $RT_{\text{trial N:correct}}$. For trial N+2, PES was calculated as $RT_{\text{trial N:error}}$ - $RT_{\text{trial N:correct}}$ when trial N+1 was correct (to eliminate a confound of the accuracy for trial N+1 on trial N+2). This logic was implemented in the ERP and the follow-up analyses. For our main hypothesis, separate repeated measures of analysis of variance (ANOVA) were run for accuracy and MRT, with trial (trial N+1 and trial N+2) and correctness of trial N (correct and error) as independent within-subject variables. According to the bottleneck hypothesis, the post-error effects should diminish from trial N+1 to trial N+2; therefore, only for this specific case, a one-tailed *t*-test was used to test the under-additive interaction between trial and the accuracy on the previous trial on all analyses.

Then, planned *t*-test comparisons were performed as follow-up analyses on significant interaction effects.

Electroencephalogram (EEG) Recording

The EEG was recorded from 128 scalp sites using the HydroCel Geodesic Sensor Net and system (Tucker, 1993). Electrode impedances were kept below 40 kΩ, an acceptable level for this system (Ferree, Luu, Russell, & Tucker, 2001). During EEG acquisition, all channels were referenced to the VREF (Cz) channel, and a 100 Hz hardware low-pass filter was used. Signals were collected at 250 samples per second and digitized with a 24-bit A/D converter.

EEG data from trials included in the behavioral analysis were analyzed using Matlab (Mathworks, Inc.), EEGLAB (Delorme & Makeig, 2004), and the EPP ToolBox (Ben Shachar, 2017). A 0.1-30 finite impulse response (FIR) Hz digital band-pass filter was applied. Then the filtered datasets were re-referenced to the average reference. For the ERN analysis, segments of 200 ms prior to response presentation and 600 ms post response were used, with a -200 to -100 baseline. For the post-error analyses, segments of 200 ms prior to stimulus presentation and 600 ms post stimulus were used, with a -200 to 0 baseline. The resulting segments were submitted to an epoch rejection procedure in which epochs with eye blinks and movements were removed. Specifically, outlier values of ±150 μV on frontal eye channels and epochs that deviated from the mean by +/-50 dB in the 0-2 Hz frequency window were removed. The average number of trials remaining per participant and condition was 305 (559 when trial N was correct versus 50 when trial N was incorrect) and trial (319 on trial N+1 versus 290 on trial N+2). In remaining segments, bad channels with joint probability or a spectrum that deviated more than five standard deviations from the epoch mean were spherically interpolated for each condition and participant. The average percentage of channels interpolated for every recording session was less than 4% of

electrodes. The artifact-free trials were then averaged for each subject. Finally, a grand average (average of all participants for each experimental condition) was created. Visual inspection of grand average ERP waveforms and topographical maps, guided by previous ERP literature, led to selection of the following ERP components: For the ERN component, a group of central channels were selected, in the time window of 0-150 ms after response onset. This group was located around Cz of the 10-20 international standard system (6, 13, 112, 30, 7, 106, 105, 37, 31, Cz, 80, 87, 55, 54, 59; Figure 2). For the visual N1 effect, a bilateral group of occipital channels was selected in the time window of 150-250 ms after stimulus presentation. This group was located in the O1 and O2 area of the 10-20 mapping (70, 69, 83, 89; Figure 2), closely overlapping with groups of electrodes from previous ERP investigations of visual processing (Vogel & Luck, 2000). For the P3 effect, a group of central channels around the Pz of the 10-20 mapping (54, 55, 79, 78, 77, 72, 67, 61, 80, 87, 86, 85, 77, 62, 60, 53, 37, 31; Figure 2) was selected in the 300-500 ms time window, closely overlapping with groups of electrodes from previous ERP investigations of memory updating and central processing (Kok, 2001; Polich, 2007).

An average amplitude measure was calculated across the chosen electrodes for each component. The ERN component was analyzed using repeated measures ANOVA, with the correctness of trial N as an independent within-subject variable. For our main hypothesis, N1 and P3 components were analyzed using separate repeated measures ANOVA, with trial and the correctness of trial N as independent within-subject variables.

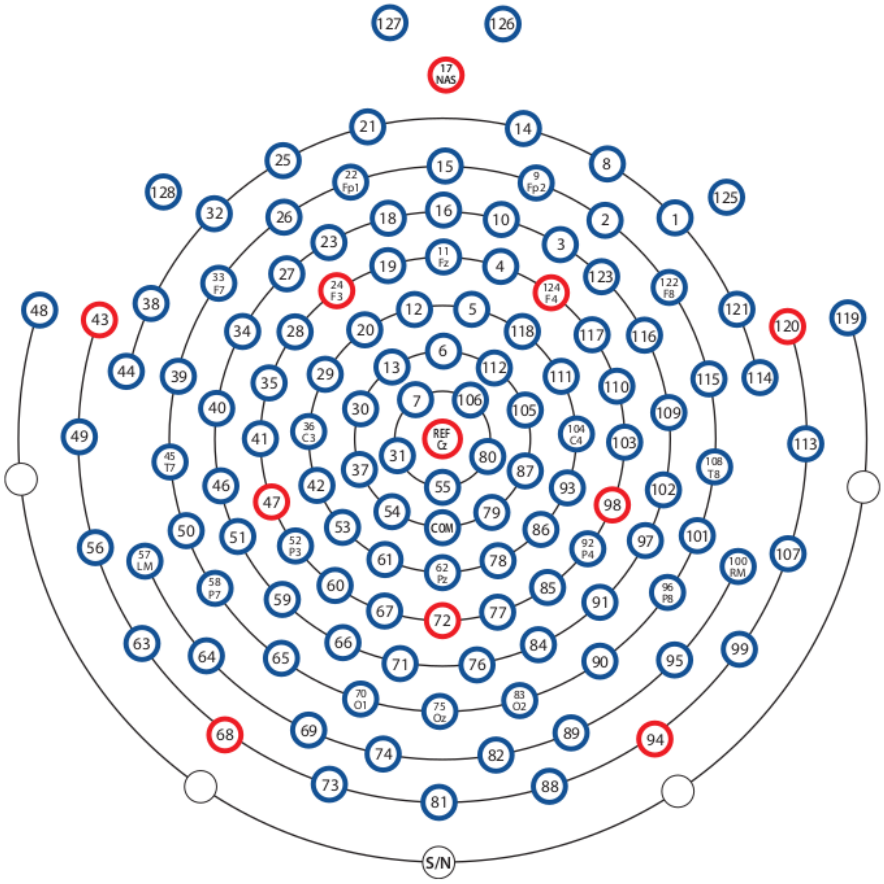

Figure 2. Schematic layout of the 128-electrode EGI HydroCel Geodesic Sensor Net.

Results

Behavioral Analysis

Trial N

The ANOVA on MRT revealed a significant main effect for repetition ($F_{1, 26} = 91.11$, $p < .001$, $\eta^2_p = .778$), with slower MRTs on non-repeat trials compared to repeat trials. The main effect for congruency was also significant ($F_{1, 26} = 13.54$, $p = .001$, $\eta^2_p = .342$), indicating that MRTs on incongruent trials were slower than on congruent trials. The interaction between repetition and congruency did not reach statistical significance ($F_{1, 26} = 3.84$, $p = .064$, $\eta^2_p = .129$;

top left panel in Figure 3). The ANOVA on accuracy revealed a significant main effect for congruency ($F_{1, 26} = 40.34$, $p < .001$, $\eta^2_p = .608$), with lower accuracy on incongruent trials compared to congruent trials. However, the main effect for repetition ($F_{1, 26} = 2.482$, $p = .127$, $\eta^2_p = .087$) and the interaction between repetition and congruency ($F_{1, 26} < 1$) were not significant (middle left panel in Figure 3). As shown on the top right panel of Figure 3, MRT on correct and erroneous trials did not differ significantly ($M = 12.9$, $SE = 9.61$, $t_{26} = 1.34$, $p = .192$, two-tailed, $d = 0.258$).

Trials N+1 and N+2

The ANOVA on MRT revealed a significant main effect for the correctness of trial N ($F_{1, 26} = 41.61$, $p < .001$, $\eta^2_p = .615$), with slower MRTs when there was an erroneous response on trial N compared to when there was a correct response on trial N, showing the PES effect. The main effect for trial was also significant ($F_{1, 26} = 24.56$, $p < .001$, $\eta^2_p = .486$), indicating that MRTs on trial N+1 were slower than on trial N+2. In line with our hypothesis, PES was larger on N+1 ($M = 72.9$, $SE = 10.86$, $t_{26} = 6.72$, $p < .001$, two-tailed, $d = 1.293$) compared to N+2 ($M = 23.7$, $SE = 7.40$, $t_{26} = 3.20$, $p = .004$, two-tailed, $d = 0.616$), which was supported by a significant under-additive interaction between trial and the correctness of trial N ($M = -49.26$, $SE = 11.00$, $t_{26} = -4.479$, $p < .001$, one-tailed, $d = -0.862$; bottom left panel in Figure 3).

The ANOVA on accuracy revealed a significant main effect for trial ($F_{1, 26} = 4.35$, $p = .047$, $\eta^2_p = .143$), indicating that accuracy on trial N+1 was lower than trial N+2. The main effect of the correctness of trial N did not reach statistical significance ($F_{1, 26} = 3.33$, $p = .079$, $\eta^2_p = .114$). However, in line with our hypothesis, an accuracy drop after an error on trial N was evident on trial N+1 ($M = 0.032$, $SE = 0.015$, $t_{26} = 2.141$, $p = .042$, two-tailed, $d = 0.412$) but not on trial N+2 ($t_{26} < 1$), which was supported by a significant under-additive interaction between

trial and the correctness of trial N (M = -0.027, SE = 0.014, t_{26} = -1.86, p = .037, one-tailed, d = -0.357; bottom right panel in Figure 3).

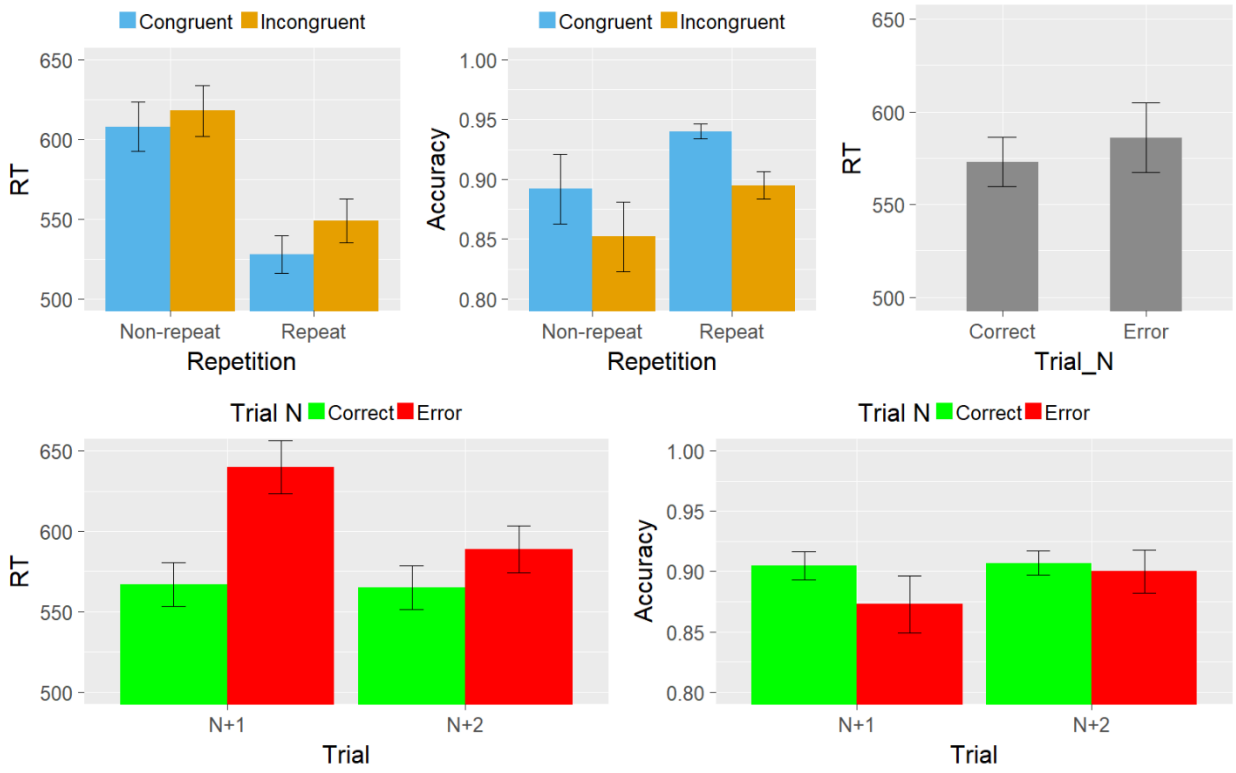

Figure 3. MRT (top left) and accuracy (top middle) on trial N as a function of repetition and congruency. MRT for correct and erroneous responses on trial N (top right). MRT for trial N+1 and trial N+2 as a function of the correctness of trial N (bottom left). Accuracy for trial N+1 and trial N+2 as a function of the correctness of trial N (bottom right). Vertical error bars represent standard errors.

ERP Analysis

N1 and P3 in Trial N

Although the ANOVA on N1 peak amplitude showed a significant main effect for congruency on trial N ($F_{1, 26}$ = 1.27, p = .044, η^2_p = .147), which indicated a more negative amplitude for congruent compared to incongruent trials, the significant interaction between

repetition and congruency ($F_{1, 26}$ = 37.74, p < .001, η^2_p = .592) revealed a more complex pattern of the effect. Namely, a more negative amplitude for congruent compared to incongruent trials was evident on the repeat condition (M = -0.838, SE = 0.167, t_{26} = -5.03, p < .001, two-tailed, d = -0.968), but a marginal effect in the opposite direction was evident on the non-repeat condition (M = 0.301, SE = 0.147, t_{26} = 2.05, p = .051, two-tailed, d = 0.394). The main effect of repetition ($F_{1, 26}$ = 1.27, p = .269, η^2_p = .047) was not significant (top left panel in Figure 4).

The ANOVA on P3 mean amplitude revealed that both effects of repetition ($F_{1, 26}$ = 7.12, p = .013, η^2_p = .215) and congruency ($F_{1, 26}$ = 8.66, p = .007, η^2_p = .250) were significant, with lower amplitude on repeat trials compared to non-repeat trials and lower amplitude on congruent trials compared to incongruent trials (top right panel in Figure 4). The interaction between repetition and congruency did not reach statistical significance ($F_{1, 26}$ = 3.65, p = .067, η^2_p = .123).

ERN in Trial N

As shown in Figure 4 (bottom panel), mean amplitudes on erroneous responses were significantly more negative compared to correct responses (M = 1.10, SE = 0.238, t_{26} = 4.62, p < .001, two-tailed, d = -0.890), demonstrating the typical ERN effect (Falkenstein et al., 2000; Gehring et al., 1993).

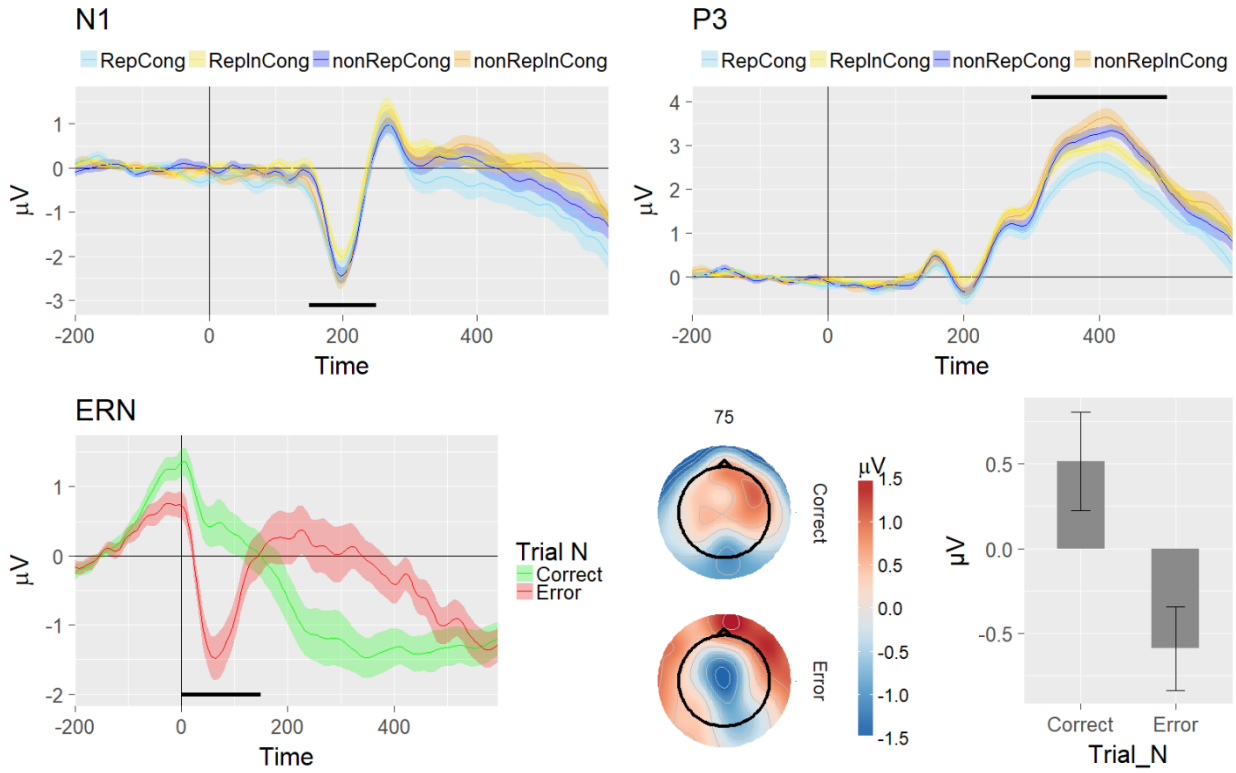

Figure 4. N1 component: Stimulus-locked waveforms of occipital electrodes as a function of repetition and congruency on trial N, with the horizontal black line representing the selected time window for analysis (top left). P3 component: Stimulus-locked waveforms of central electrodes as a function of repetition and congruency on trial N, with the horizontal black line representing the selected time window for analysis (top right). Full condition names for top plots are: RepCong = repeat congruent; RepInCong = repeat incongruent; nonRepCong = non-repeat congruent; nonRepInCong = non-repeat incongruent. ERN component (bottom): Response-locked waveforms of central electrodes for correct and erroneous response, with the horizontal black line representing the selected time window for analysis (bottom left). Topographic maps for the correct and incorrect response 75 ms after response onset (bottom middle). Mean amplitudes for correct and incorrect conditions (bottom right).

N1 and P3 in Trials N+1 and N+2

The ANOVA on N1 peak amplitude revealed a significant main effect for the correctness of trial N ($F_{1, 26} = 10.926$, $p = .003$, $\eta^2_p = .296$), indicating a more negative amplitude after an

erroneous response on trial N compared to after a correct response on trial N. The main effect of trial ($F_{1, 26}$ = 1.39, p = .250, η^2_p = .051) and the under-additive interaction between trial and the correctness of trial N, (t_{26} < 1) were not significant (Figure 5).

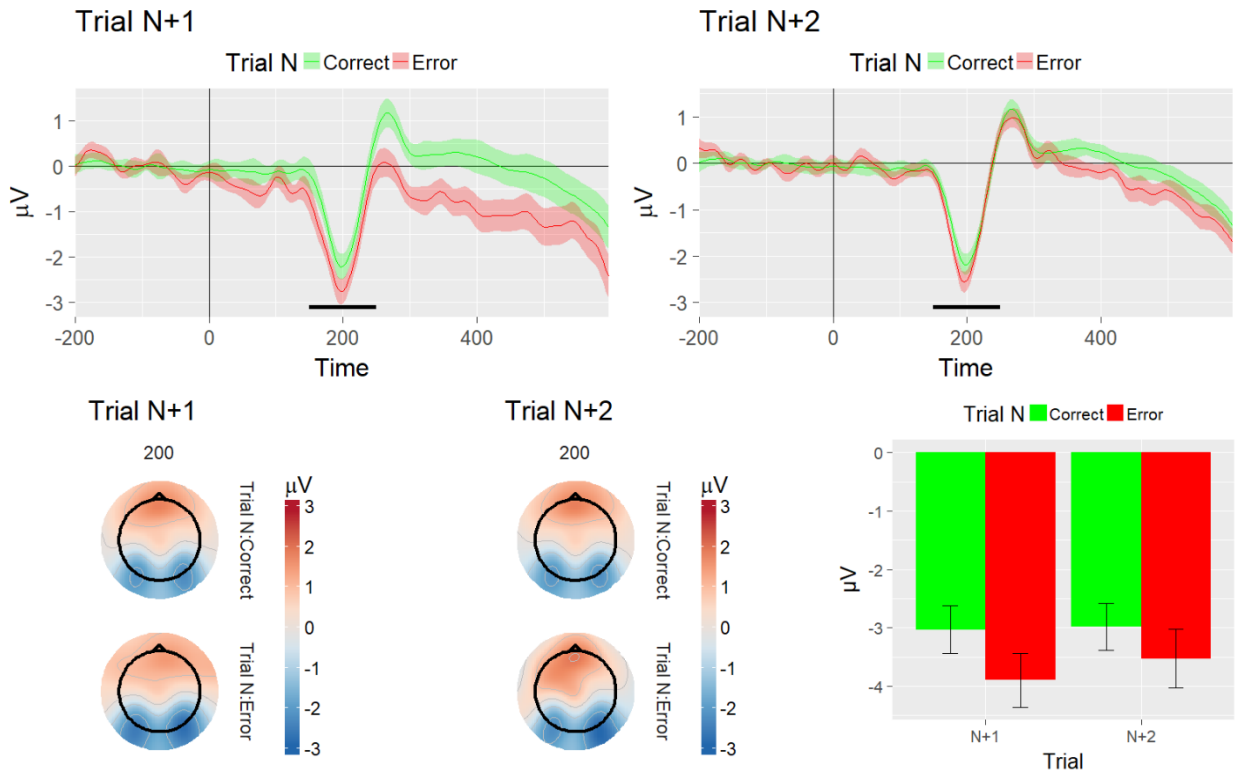

Figure 5. N1 component: Stimulus-locked waveforms of occipital electrodes as a function of the correctness of trial N, on trial N+1 and trial N+2, with the horizontal black line representing the selected time window for analysis (top). Topographic maps for trial N+1 (bottom left) and trial N+2 (bottom middle) 200 ms after stimulus onset. Peak amplitudes as a function of the correctness of trial N, on trial N+1 and trial N+2 (bottom right).

The ANOVA on P3 mean amplitude revealed a significant main effect for the correctness of trial N ($F_{1, 26}$ = 8.39, p = .008, η^2_p = .244), indicating a lower amplitude after an erroneous response on trial N compared to after a correct response on trial N. The main effect of trial ($F_{1, 26}$ = 7.57, p = .011, η^2_p = .226) was also significant, with lower amplitudes on trial N+1 compared

to trial N+2. In line with our hypothesis, the P3 differences were evident on trial N+1 (M = 0.692, SE = 0.203, t_{26} = 3.399, p = .002, two-tailed, d = 0.654) but not on trial N+2 (t_{26} < 1), which was supported by a significant under-additive interaction between trial and the correctness of trial N (M = -0.579, SE = 0.222, t_{26} = -2.61, p = .007, one-tailed, d = -0.501; Figure 6).

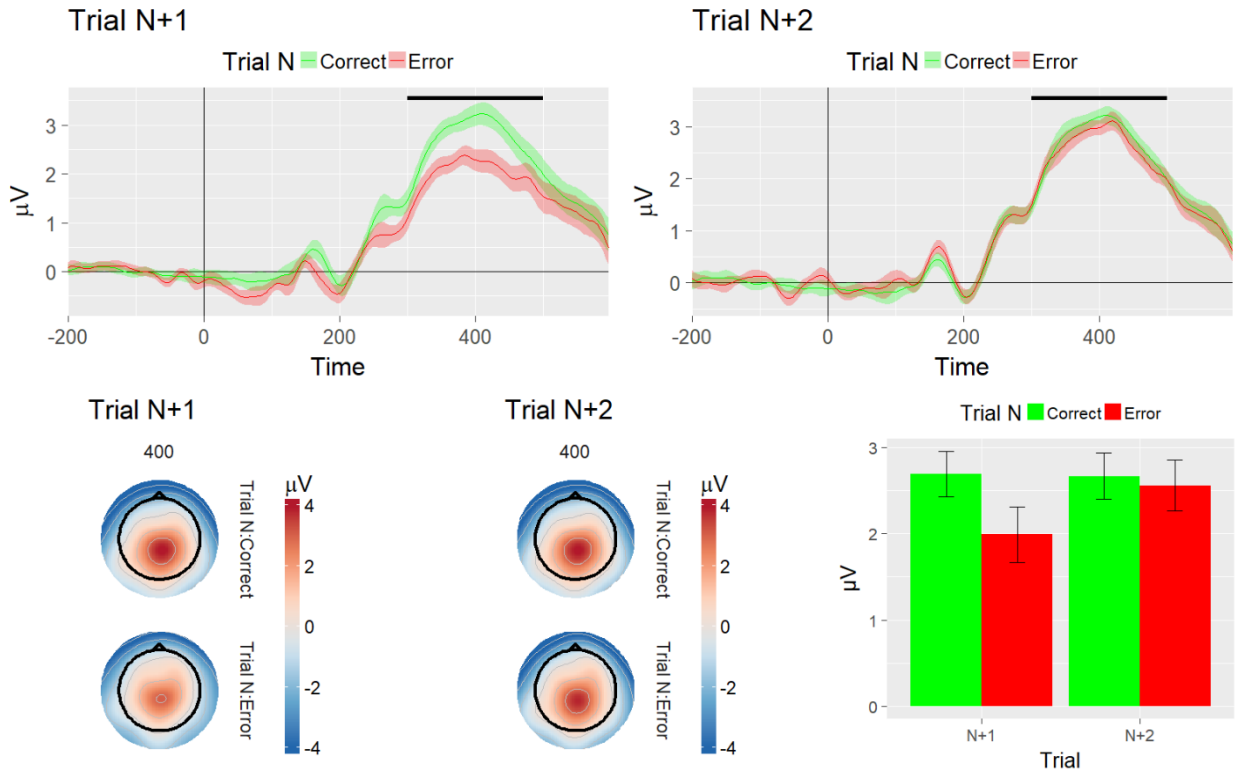

Figure 6. P3 component: Stimulus-locked waveforms of central electrodes as a function of the correctness of trial N, on trial N+1 and trial N+2, with the horizontal black line representing the selected time window for analysis (top). Topographic maps for trial N+1 (bottom left) and trial N+2 (bottom middle) 400 ms after stimulus onset. Peak amplitudes as a function of the correctness of trial N, on trial N+1 and trial N+2 (bottom right).

As in most cognitive tasks, our participants had more correct responses than erroneous responses (right panel in Figure 3), meaning that the SNR for the post-correct conditions might have been higher compared to the post-error conditions, which could have driven the effects on

N1 and P3 amplitudes. To rule out a general confound with SNR and correctness of trial N, we compared the N1 and P3 amplitudes on the post-correct conditions with a subset of post-correct trials. The subset of post-correct trials was matched to post-error trials by choosing post-correct trials that preceded an error (cf. Dutilh et al., 2012). Specifically, the N1 and P3 components were submitted to repeated measures ANOVA with component (N1 and P3), trial (N+1 and N+2), and number of trials (all post-correct and subset post-correct) as independent within-subject variables. The main effect of number of trials ($F_{1, 26} = 2.24$, $p = .147$, $\eta^2_p = .079$) and all interactions between number of trials and other conditions were not significant ($Fs_{1, 26} < 1.41$), weakening the claim for an SNR confound in our data.

Follow-up Analyses

Because the effect of the correctness of trial N on the PES and the N1 component persisted after trial N+1, we further analyzed the post-correct versus post-error sequence in the trials following the post-error N+2, to shed light on the time course of this phenomenon. We visually examined the topographic maps, ERP waveforms, and the behavioral descriptive data. While doing so, we strived to inspect, on average, no fewer than 30 trials per condition for each participant. Consequently, this allowed us to go as far as N+5 from the correct or erroneous response on trial N. Although the predictions of the follow-up analyses were clear in terms of the direction of the post-error effects, namely, that the effects of N1 and PES would diminish with time, the tests that we performed were all two-tailed tests because of the exploratory nature of the analyses. Furthermore, to reduce the number of tests and minimize alpha accumulation, significant effects on N+3, N+4, and N+5 trials were compared to the previous trials in the sequence, with a single interaction contrast comparing the current trial to all previous trials. We also tested for SNR confound in the follow-up ERP analysis, and as before, the main effect of

number of trials and the interactions between number of trials and other conditions were not significant (*Fs* < 1).

Behavioral Analysis

As in our main analysis, we examined MRT and accuracy as a function of the correctness of trial N, on trial N+3, trial N+4, and trial N+5.

As shown in Figure 7, the effect on MRT diminished with time on the post-correct versus post-error sequence, continuing the trend observed on trials N+1 and N+2. On trial N+3, MRTs were slower when there was an erroneous response on trial N compared to when there was a correct response on trial N (*M* = -15.900, *SE* = 6.37, t_{26} = -2.49, *p* = .019, two-tailed, *d* = -0.480), showing a prolonged PES effect, which was also smaller compared to previous trials in the sequence, as confirmed by a significant interaction contrast comparing the effect of correctness of trial N on trial N+3 with previous trials (*M* = -64.797, *SE* = 16.81, t_{104} = -3.84, *p* < .001, two-tailed, *d* = -0.708). On trial N+4, MRTs were only marginally slower when there was an erroneous response on trial N compared to when there was a correct response on trial N (*M* = -10.841, *SE* = 5.30, t_{26} = -2.04, *p* = .051, two-tailed, *d* = -0.393), which was also supported by a significant interaction contrast comparing the effect of correctness of trial N on trial N+4 with previous trials (*M* = -79.974, *SE* = 23.78, t_{104} = -3.36, *p* = .001, two-tailed, *d* = -0.840). On trial N+5, however, the effect of correctness of trial N on PES disappeared (t_{26} < 1), which was supported as well by a significant interaction contrast comparing the effect of correctness of trial N on trial N+5 with previous trials (*M* = -120.649, *SE* = 30.69, t_{104} = -3.93, *p* < .001, two-tailed, *d* = - 0.787).

The null effect of the correctness of trial N on accuracy was observed on trials N+3, N+4, and N+5 (ts_{26} < 1), continuing the pattern observed on trials N+1 and N+2.

ERP Analysis

As in our main analysis, we examined the N1 and P3 components as a function of the correctness of trial N on trial N+3, trial N+4, and trial N+5.

As shown in Figure 7, the effect on N1 corresponded with the behavioral effect on MRT. On trial N+3, the N1 peak amplitude was more negative when there was an erroneous response on trial N compared to when there was a correct response on trial N (M = -0.806, SE = 0.18, t_{26} = -4.39, $p < .001$, two-tailed, d = -0.844), showing a prolonged N1 effect that was similar in size to previous trials in the sequence ($t_{104} < 1$). On trial N+4, the same pattern was observed, where N1 peak amplitude was more negative when there was an erroneous response on trial N compared to when there was a correct response on trial N (M = -0.600, SE = 0.21, t_{26} = -2.85, p = .008, two-tailed, d = -0.549), which was also comparable to the N1 effect observed on previous trials ($t_{104} < 1$). On trial N+5, however, the effect of correctness of trial N on the N1 component disappeared ($t_{26} < 1$), which was supported as well by a significant interaction contrast comparing the effect of correctness of trial N on trial N+5 with previous trials (M = -2.912, SE = 1.05, t_{104} = -2.78, p = .006, two-tailed, d = - 0.416).

The effect on P3 mirrored the behavioral effect on accuracy, as no effect was found for the correctness of trial N on accuracy on trials N+3, N+4, and N+5 ($ts_{26} < 1$), continuing the pattern observed on trials N+1 and N+2.

Figure 7. Follow-up analyses: Stimulus-locked waveforms of occipital electrodes as a function of the correctness of trial N on trial N+3, N+4, and trial N+5, with the horizontal black line representing the selected time window for N1 component analysis (top). Peak amplitudes as a function of the correctness of trial N on trial N+3, N+4, and trial N+5 (bottom left). MRT for trial N+3, N+4, and trial N+5 as a function of the correctness of trial N (bottom right).

Discussion

We set out to examine the behavioral and ERP measures on the first and second trials following the commission of an error in a speeded-choice task. Our hypothesis was that the responses would be slower and less accurate after commission of errors compared to post-correct responses and that both effects would be attenuated on trial N+2. Consistent with our hypotheses, we found that participants were slower and less accurate immediately after making an error and that this reduction of performance decayed over time. Moreover, the behavioral

post-error effects were reflected in differences in the visual N1 component as well as in the P3 component. Interestingly, only the difference in the P3 component diminished over time, contrary to our expectations, whereas the differences in the N1 component were still evident in the second trial following the erroneous response.

As expected, we observed the PES and post-error accuracy decrease pattern in our experiment. If we focus solely on the behavioral data, this pattern can be laid as support for the orienting account (Notebaert et al., 2009) as well as the bottleneck account for PES (Hochman & Meiran, 2005; Jentzsch & Dudschig, 2009). In other words, the PES that was still evident on the second trial after the error corresponds well with the limited-resources explanation for PES and may also hold for the explanation that relies on a prolonged orienting response that carries over across trials. Moreover, the reduced post-error effect on trial N+2 compared to trial N+1 supports the logic of the bottleneck account, namely that there are more cognitive resources and thus better performance as time from the error increases. However, it can also be argued that the orienting response is a transient phenomenon that also decays over time. Nevertheless, the diminished accuracy drop on trial N+2 closely replicates the previously reported pattern of post-error bottleneck effect on long ITIs (Jentzsch & Dudschig, 2009).

Although the main question of the present study deals with post-error brain-behavior effects, it was important for us to confirm that the ERN observed after errors was expressed in a typical form in our data. Indeed, the pattern of the results is similar to the ERN found in numerous previous studies (Dudschig & Jentzsch, 2009; Falkenstein et al., 2000; Ganushchak & Schiller, 2006; Gehring & Fencsik, 2001; Gehring et al., 1993; Hajcak et al., 2003; Hajcak, Moser, Yeung, & Simons, 2005; Luu et al., 2003; Mathalon et al., 2003; Perri, Berchicci, Lucci, Spinelli, & Di Russo, 2015; Rigoni, Pourtois, & Brass, 2015; Steele et al., 2016; Van der Borght

et al., 2016). Importantly, we used a constant-stimulus presentation, ensuring that there would be no differences in the perceptual level of encoding between the correct and incorrect responses. This was done with the risk of having slight ITI differences, depending on correct and erroneous RTs. However, the results showed that no differences existed between correct and erroneous responses, thus eliminating the risk for an ITI confound.

As mentioned, previous results showed that error detection can lead to impairment in attention-related processes in short and long ITIs, confirming that error processing demands attentional resources, thus supporting the bottleneck account (Buzzell et al., 2017; Van der Borght et al., 2016). By comparing the first trial after the correct or erroneous response with the second trial after the correct and erroneous response, our data demonstrate an even longer lasting effect of the post-error bottleneck. Although the mentioned studies relied on the attenuation of amplitude to represent impairment in attentional processes[1], in the context of the N1 component, higher amplitude can account for reorienting of attention. In support, previous ERP studies in the visual domain have shown an enhanced N1 component for conditions that require redirection of attention (Boksem, Meijman, & Lorist, 2005; Heinze, Luck, Mangun, & Hillyard, 1990; Luck, Heinze, Mangun, & Hillyard, 1990; Yamaguchi, Tsuchiya, & Kobayashi, 1995). This mechanism of reorienting of attention that might take place on post-error trials corresponds well with the orienting explanation for PES, which states that errors shift attention away from the current task (Houtman & Notebaert, 2013; Notebaert et al., 2009). Notably, our follow-up analyses showed that the post-error effect on the N1 component was coupled with the PES effect. Namely, they both persisted until approximately trial N+4 and disappeared on trial N+5. Taken

[1] Although not tested directly in Buzzell et al., 2017, the descriptive data provided show a trend of increased post-error N1 compared to post-correct N1.

together, this might suggest that at least part of the PES effect can account for the time it takes to shift attention back from the committed error toward the ongoing task.

Moreover, based on the P3 component, our results revealed that higher-order processing, such as memory updating, was affected following an error. In support, previous ERP studies that have investigated the P3 component in the *n*-back paradigm drew a link between P3 amplitude and working memory load. Specifically, the findings showed that higher working memory demands were reflected by decreased P3 amplitudes (Gevins et al., 1996; McEvoy, Smith, & Gevins, 1998; Watter, Geffen, & Geffen, 2001). As additional evidence for this link, our data on trial N showed that P3 amplitude was the largest on repeat congruent trials when working memory demands were low and smallest on non-repeat incongruent trials when working memory demands were high. In regard to the post-error trials, Watter and colleagues (2001) have proposed that the *n*-back paradigm can be conceptualized as a subtype of a dual-task paradigm, with distinct components of a working memory updating subtask and a matching subtask. According to the proposed conceptualization, a decreased P3 amplitude represents a reallocation of processing capacity away from the matching subtask to the working memory updating subtask, which may become more difficult to execute with error monitoring operations competing for central resources. This change of strategy and resource allocations might also be triggered by the error and represent the main locus of the post-error bottleneck effects (Lavro & Berger, 2015; Lavro, Levin, Klein, & Berger, 2018).

A considerable overlap exists between the processes that determine RT and those that produce the P3 (Kutas, McCarthy, & Donchin, 1977). This is also shown in our results: The P3 attenuation was coupled with slower RTs on post-error trials compared to post-correct trials on N+1 trials. Correspondingly, Van der Borght and colleagues (2016) found the same pattern for

P3 in their data. However, our study also showed that the P3 difference between post-correct and post-error trials disappeared on trial N+2. Interestingly, the disappearance of P3 differences was also coupled with the disappearance of the accuracy difference on trial N+2, which may link post-error accuracy changes with higher-order processing (Danielmeier & Ullsperger, 2011). Moreover, as the follow-up analyses showed, this pattern persisted to N+5 in the trial sequence, strengthening the link between post-error accuracy and higher-order processing.

Although the attenuation of the P3 differences between post-correct and post-error amplitudes was expected, we did not expect the N1 component to show the same difference between the post-correct and post-error amplitude on the first and second trial after the correct or erroneous response. Only the P3 component was diminished from trial N+1 to trial N+2, which corresponds to the behavioral studies that focus on the central bottleneck account (Hochman & Meiran, 2005; Jentzsch & Dudschig, 2009; Lavro & Berger, 2015) as opposed to recent ERP findings that support the attentional bottleneck (Buzzell et al., 2017; Van der Borght et al., 2016). Importantly, however, Van der Borght and colleagues (2016) also reported that the N1 differences between the post-correct and post-error trials were found, irrespective of ITI. Nevertheless, it should be noted that our results do not reflect a standard, central bottleneck model that predicts a processing delay of central processes. In contrast to our findings that focused on P3 amplitude, it has been shown that processing delay of central processes is represented by a P3 latency postponement rather than P3 amplitude attenuation (Dell'Acqua, Jolicoeur, Vespignani, & Toffanin, 2005), further supporting the claim that the current results represent a more widespread bottleneck effect resulting from a strategy change following an

error as opposed to merely being a central processing delay[2]. Together, these results might suggest that the commission of errors triggers a combination of attentional reorientation that persists over time as well as a high-level resource reallocation, which can be dissociated by the N1 and P3 components, respectively.

A possible limitation of our study should be addressed. As outlined before, to succeed in the COBT, the participants were not only required to accurately perceive and process the current trial but also had to remember the stimulus that appeared on the previous trial. In the context of error-monitoring investigation, this setting has a clear advantage of being relatively challenging to participants, producing errors that can be studied later. Also, the COBT incorporates low and high levels of processing that can be associated with well-known ERP components. However, it can be argued that the working-memory element of the COBT structurally binds each trial to the next one, limiting the results presented here to this specific setting. Nevertheless, as the results show, the PES and the ERN – well-established markers of error-monitoring – were evident in their standard and typical form, which replicated previous findings. Moreover, in a broader context, it should be noted that modifications of existing one-dimensional tasks to more complex tasks prove to be useful in error-monitoring research (e.g., Chiu & Deldin, 2007; Hoffmann & Falkenstein, 2011; Saunders & Jentzsch, 2012; Takács, Kóbor, Honbolygó, & Csépe, 2015; Ullsperger & von Cramon, 2004). Yet, we cannot fully reject the argument for inadequate external validity in our study; therefore, future research is needed to confirm our observations in a wider scope of different task settings.

[2] The null effect of P3 latency was confirmed by a set of *t*-tests comparing P3 50% area latency (Luck, 2005) on post-correct and post-error trials. These tests did not show any significant differences of latency on N+1, N+2, N+3, N+4, and N+5 trials ($ts_{26} < 1$).

To conclude, our results provide additional support for the bottleneck account for PES (Jentzsch & Dudschig, 2009) and shed light on the processes that take place during the attentional-central bottleneck phase. Our findings suggest that both low- and high-order processes are triggered by the commission of an error. This is consistent with claims that the best explanation for PES can be achieved with a combination of early attentional and higher-order processing changes that occur after erroneous responses (Purcell & Kiani, 2016; Ullsperger & Danielmeier, 2016). Future research will show whether the N1 and P3 components are, indeed, related directly to these processes.

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