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Outcome expectancy and not accuracy determines posterror slowing: ERP support

Elena Núñez Castellar
Ghent University, Ghent, Belgium

Simone Kühn
Ghent University, Ghent, Belgium,
and Max Planck Institute for Human Cognitive and Brain Sciences, Leipzig, Germany

and

Wim Fias and Wim Notebaert
Ghent University, Ghent, Belgium

A considerable number of studies have recently used event-related potentials (ERPs) to investigate the mechanisms underlying error processing. Nevertheless, how these mechanisms are associated with behavioral adjustments following errors remains unclear. In the present study, we investigated how posterror slowing is linked to outcome expectations and error feedback. We used an adaptive four-choice reaction time task to manipulate outcome expectancy. Behaviorally, the results show posterror slowing when errors are unexpected and postcorrect slowing when correct responses are unexpected, indicating that outcome expectancy is crucial for posterror slowing. ERP analyses revealed that the error-related negativity and the feedback-related negativity were not correlated with the behavioral reaction time pattern, whereas the P3 was. The results support the hypothesis that posterror slowing is caused by attentional orienting to unexpected events.

Mechanisms that support cognitive control induce behavioral adaptations based on performance of earlier trials in order to optimize task performance. Errors are thought to play an important role in this by signaling the necessity to implement online adjustments. On the behavioral level, it has consistently been found that participants' response speed decreases after an error. According to conflict monitoring theory (CMT; Botvinick, Braver, Barch, Carter, & Cohen, 2001), errors result in a strategic adaptation (i.e., an increase of the response threshold) in order to reduce error likelihood on the following trial. Consequently, CMT predicts increased reaction times (RTs) and increased accuracy following errors. However, although posterror slowing in combination with increased accuracy has been reported occasionally (Laming, 1968; Marco-Pallares, Camara, Münte, & Rodríguez-Fornells, 2008; Rabbitt, 1966), other studies have found posterror slowing in combination with decreased accuracy (Hajcak, McDonald, & Simons, 2003; Hajcak & Simons, 2008; Laming, 1979; Rabbitt & Rodgers, 1977).

Other lines of research raise additional doubt about this cognitive explanation for posterror slowing. For instance, recent psychopharmacological studies have shown that deficient error detection is not related to modulations in the size of the slowing (Riba, Rodríguez-Fornells, Morte, Münte, & Barbanoj, 2005; Riba, Rodríguez-Fornells, Münte, & Barbanoj, 2005; Rodríguez-Fornells, Kurzbuch, & Münte, 2002). Moreover, studies with schizophrenic and Parkinson's patients show deficient error detection in the frontal brain, whereas posterror slowing is not affected (e.g., Mathalon et al., 2002). Along the same lines, neurological studies have demonstrated that damage in brain areas that are thought to play an essential role in cognitive control and conflict monitoring (i.e., lateral prefrontal cortex and dorsal anterior cingulate cortex) does not affect posterror slowing (Gehring & Knight, 2000; Modirrousta & Fellows, 2008).

Taken together, these findings argue against the widely accepted idea that posterror slowing is a compensatory control mechanism geared toward improving performance in subsequent trials (Gehring & Fencsik, 2001). Interestingly, Notebaert et al. (2009) recently reported evidence in favor of an alternative account for posterror slowing, which suggests that slowing after errors might be caused by attentional orienting to unexpected events (orienting account). In a first experiment, an adaptive four-color-choice RT task was used to manipulate the expectancy of error and correct responses. Participants performed the task in three accuracy conditions: a 75%-correct condition (correct expectancy), a 55%-correct condition (control), and a 35%-correct condition (error expectancy); color

E. Núñez Castellar, elenapatricia.nunezcastellar@ugent.be

intensity was adjusted in order to reach these accuracy levels. Behaviorally, the results showed post*error* slowing in the 75%-correct condition, but, remarkably, in the 35%-correct condition, post*correct* slowing was found. Likewise, in a second experiment, in which expected or unexpected tones followed the response (unrelated to participants' performance), slowing was observed after unexpected irrelevant tones.

In the present study, we investigate the neurophysiological correlates of posterror and postcorrect slowing. The procedure developed by Notebaert et al. (2009) allows us to test whether posterror slowing is related to event-related potential (ERP) components traditionally associated with error processing, such as error-related negativity (ERN) and feedback-related negativity (FRN), or with components usually associated with attention and expectancy (P3).

Previous ERP studies have identified a component associated with errors in the response-locked signal. This ERN is a negative deflection maximal at frontocentral sites and peaking approximately 50 msec after the onset of the incorrect response (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). The most influential theories suggest that the ERN is generated when conflict between two or more responses occurs (Botvinick et al., 2001) or when consequences of an action are worse than expected (reinforcement learning theory; Holroyd & Coles, 2002). Brown and Braver (2005) integrated these perspectives, postulating that the ERN is a training signal modulated by error likelihood rather than response conflict. This hypothesis, however, is still debated (Nieuwenhuis, Schweizer, Mars, Botvinick, & Hajcak, 2007). Furthermore, some recent studies have also shown an ERN-like component for correct responses (Falkenstein et al., 2000; Luu, Collins, & Tucker, 2000; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). Although the functional significance of this correct-related negativity (CRN) remains unclear, it has been suggested that the CRN reflects response-monitoring processes (Vidal et al., 2000).

More important for the present purposes is the functional significance of the ERN in relation to posterror slowing. Although some studies have shown a correlation between the ERN and slowing (Debener et al., 2005; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Marco-Pallares et al., 2008; West & Travers, 2008; see also, for recent work in the time-frequency domain, Cavanagh, Cohen, & Allen, 2009), at least an equal number of studies reported no such relationship. Mathalon et al. (2002), for instance, observed posterror slowing in the absence of an ERN in a group of schizophrenic patients (for similar data in Parkinson's patients, see Stemmer, Segalowitz, Dywan, Panisset, & Melmed, 2007), and a considerable number of studies have indicated that ERN is not associated with slowing after errors (Beste, Willemssen, Saft, & Falkenstein, 2009; Carp & Compton, 2009; Endrass, Reuter, & Kathmann, 2007; Hajcak et al., 2003; Nieuwenhuis, Ridderinkhof, Blow, Band, & Kok, 2001).

Additionally, feedback-locked ERP studies have identified two components associated with feedback processing: the feedback-related negativity (FRN) and the P3 (or P300; Frank, Woroch, & Curran, 2005; Holroyd & Krigolson, 2007; Miltner, Braun, & Coles, 1997; Sato et al., 2005; Yeung & Sanfey, 2004). The FRN is a negative deflection, maximal at frontocentral recording sites, occurring approximately 250 msec after negative feedback (Miltner et al., 1997). The interpretation of the FRN is assumed to be very similar to the ERN: Both are thought to reflect error signals that indicate a violation of a "high-level" goal, and have been assumed to be relevant for the adaptive modification of behavior (Krigolson & Holroyd, 2006).

The P3 is a slow-wave component peaking in the period of 200–600 msec after feedback onset with a positive polarity (Yeung, Holroyd, & Cohen, 2005; Yeung & Sanfey, 2004). Evidence from gambling and probability learning tasks suggests that changes in the P3 amplitude are associated with attentional processes related to reward: either the processing of reward (reward/nonreward; Hajcak, Moser, Holroyd, & Simons, 2007) or the processing of reward magnitude, in particular (small/large; Sato et al., 2005; Yeung & Sanfey, 2004). In general, the events that give rise to a P3 can vary widely (from salient, novel, or rare stimuli, to the absence of expected stimuli) but have in common that they are motivationally significant (Ridderinkhof, Ramautar, & Wijnen, 2009). The P3 is thought to reflect attentional processes as indexed by two subcomponents: the "P3a," sensitive to novel events that involuntarily capture attention (e.g., Friedman, Cycowicz, & Gaeta, 2001) and the "P3b," sensitive to the amount of attentional resources allocated to a stimulus (e.g., Polich, 2007). Importantly, although both components may represent distinct neural processes, several studies suggest that both reflect the output of a widely distributed neural network (Polich, 2007), which seems to be crucial not only for the allocation of attentional resources but also for context memory updating.

Considering that previous studies have demonstrated that the P3 reflects detection of violations of expectations regarding gains/losses (Hajcak, Holroyd, Moser, & Simons, 2005; Hajcak et al., 2007), and in particular that the P3a subcomponent has been interpreted as an index of orienting responses (see, e.g., Friedman et al., 2001), the present study aimed to explore the relationship between the P3 and posterror slowing. Specifically, we tested the main prediction of the orienting account (Notebaert et al., 2009) that orienting toward unexpected error feedback causes posterror slowing, just as orienting toward unexpected correct feedback causes postcorrect slowing. If slowing indeed occurs on the basis of the preceding orienting response, the amplitude of the P3 on trial n, and not the amplitude of the ERN or FRN, should predict the slowing on trial $n+1$. To this end, we used an adaptive four-choice RT task (see Notebaert et al., 2009). Participants performed the task in two conditions: 75%-correct responses (expectancy for correct) and 35%-correct responses (expectancy for error). In Notebaert et al., behavioral results showed post*error* slowing in the 75%-correct condition and post*correct* slowing in the 35%-correct condition.

METHOD

Participants

Fifteen participants (age range, 18–24 years; mean, 20.6 years; 11 female, 4 male) took part in the experiment. Each gave informed consent, had the approval of the local ethical committee, and participated in accord with the Declaration of Helsinki. All participants had normal or corrected-to-normal vision and were neurologically and psychiatrically healthy. Participants were paid €15 per hour and received, on average, €37 for their participation.

Procedure

The participants were seated in a comfortable armchair in a light-dimmed and sound-attenuated room. An adaptive four-choice RT task was used to manipulate error rates, closely following Notebaert et al. (2009). Stimuli were 0.4º × 0.4º colored squares presented centrally on a white background. The brightness of the colors was adjusted in order to keep every participant's performance at a prespecified level (35% or 75% accuracy). Colors are described according to the HSV color model with three parameters: hue (0–360), saturation (0–100), and value (0–100). The four colors that were used in the practice trials were red (20, 100, 80), yellow (60, 100, 80), green (120, 100, 80), and blue (240, 100, 80). Participants responded to each of the four colors with one of the four buttons on an E-Prime response box, using their left and right middle and index fingers. Four different color-to-button mappings were used, and participants were randomly assigned to one of these mappings.

Each trial started with a central fixation cross (500 msec) before target stimulus onset. The stimulus was presented for a maximum of 500 msec or until a response button was pressed. The response was immediately followed by a feedback signal ("J" for correct and "F" for incorrect, corresponding to the Dutch words *juist* and *fout*). Four different intertrial intervals were randomly intermixed (150 msec, 250 msec, 500 msec, and 750 msec).

We used immediate feedback following the response in order to avoid expectancies (and expectancy violations) for the timing of feedback onset (which would have occurred with variable response–feedback intervals).

In a first practice block, 30 trials were presented without a response deadline. In a second block of 60 practice trials, a response deadline of 1,000 msec was introduced together with a feedback signal: "T," for "too slow" (*te laat*, in Dutch). This practice block was followed by two experimental blocks of 400 trials, corresponding to the 35%- and 75%-accuracy rate manipulation. The order in which the blocks were presented was counterbalanced: Half of the participants performed the blocks in a 35%–75% order and the other half in a 75%–35% order. Within each block, participants received a short break after 200 trials. On every trial, the program calculated the accuracy of the last 20 trials and adjusted the color value by 1 value point when accuracy deviated from the specified level (75% or 35%). The color value increased when accuracy was too low and decreased when accuracy was too high. With constant hue and saturation levels, adjusting the color value affects the brightness of the stimuli (see Notebaert et al., 2009).

Electrophysiological Recordings

Brainwaves were measured with 31 Ag/AgCl electrodes mounted in an elastic electrode cap (EasyCap, Herrsching-Breitbrunn, Germany) according to a modified 10–20 setting (with the electrodes Fp1, Fpz, Fp2, F7, F3, Fz, F4, F8, FT7, FC3, FCz, FC4, FT8, T7, C3, Cz, C4, T8, TP7, CP3, CPz, CP4, TP8, P7, P3, Pz, P4, P8, O1, Oz, O2). The EEG was recorded continuously with a sampling rate of 512 Hz (REFA-64 amplifier, TMS International, The Netherlands), and EEG signals were referenced to the mean of all electrodes. Impedances of the electrodes were kept below 4 KΩ. The electrooculogram (EOG) was recorded with bipolar montage. The vertical EOG was measured with two electrodes placed above and below the left eye; the horizontal EOG was measured with two electrodes placed on the left and right cantus. The EEG was rereferenced offline to the average signal of the electrodes placed on the left and right mastoid and digitized at 512 Hz. The continuous EEG was filtered offline with a band-pass filter of 0.1–30 Hz and a 50-Hz notch filter.

Data Analysis

EEG data were analyzed using EEProbe 3.1 (ANT, Inc., Enschede, The Netherlands). ERPs were time-locked to the onset of the response/feedback (as in Luu, Shane, Pratt, & Tucker, 2009) with a time window from −200 to 1,000 msec. Epochs containing artifacts, such as blinks, were rejected from further analysis if the standard deviation of any scalp electrode exceeded 20 μV within a sliding window of 200 msec. Since the accuracy rates were manipulated and the conditions of interest had different numbers of trials, we selected a random sample of trials for the analyses on the basis of the condition with the fewest trials. Using this procedure, we ensured that all conditions had an equal number of trials (58, on average, with a minimum of 30).

The ERN analysis was performed at the midline electrode FCz, where the ERN was maximal, using peak-to-peak quantification (Falkenstein et al., 2000). ERN amplitude was measured as the difference of the negative peak in a time window 0–120 msec relative to buttonpress onset and the preceding positive peak time in the window −100 to 0 msec (see, e.g., Eppinger, Kray, Mock, & Mecklinger, 2008; Ullsperger & von Cramon, 2006; Yeung & Sanfey, 2004).

The FRN was quantified at FCz, where it was maximal. The FRN was defined as the negative deflection maximal over (fronto)central scalp locations after performance feedback, and was measured as the difference between the maximum value between 250 and 400 msec following feedback onset and the most negative point between this maximum and 450 msec (see also Hajcak, Moser, Holroyd, & Simons, 2006). If there was no negative deflection (e.g., if two data points were the same), the FRN was scored as 0. Additionally, in order to rule out the possibility that the FRN variations were the result of differences in the P3 component, we filtered low frequencies using a band-pass filter at 3–30 Hz. The P3 has a relatively low frequency (~3 Hz) (Polich & Hoffman, 1997a, 1997b) and it has been shown that the upper-band EEG frequencies do not substantially contribute to P3 values (Intriligator & Polich, 1995; Polich & Hoffman, 1997a, 1997b). Therefore, we predicted that the pattern of results for the FRN should not change after filtering.

To measure the amplitude of the feedback-locked P3, a peak-to-peak analysis was performed (note that the effects with a mean amplitude analysis were qualitatively the same). The amplitude of the P3 component was quantified at FCz, where the amplitude of this component was maximal (see Figure 3 potential maps), and measured as the difference between the first maximum positive deflection in a time window 250–450 msec relative to buttonpress onset and the preceding negative peak in the time window 200–350 msec. In order to filter out the FRN, we filtered high frequencies, applying a 3-Hz low-pass filter to discard the possibility that the differences in the P3 might be primarily tied to variations in the FRN component (see also Bernat, Hall, Steffen, & Patrick, 2007).

The peak amplitude data for the ERN, FRN, and the P3 components were subjected to a 2 × 2 repeated measures ANOVA with the factors accuracy on the current trial (correct vs. error) and accuracy condition (35% correct vs. 75% correct). We applied the Greenhouse–Geisser correction when there was more than one degree of freedom in the numerator. The corrected p values are reported. We employed post hoc paired t tests.

RESULTS

Behavioral Data

We applied the same procedure described by Notebaert et al. (2009) for the data analysis: We excluded trials that occurred before a stable accuracy level was reached (on average, 35 trials [4.3%] were excluded in

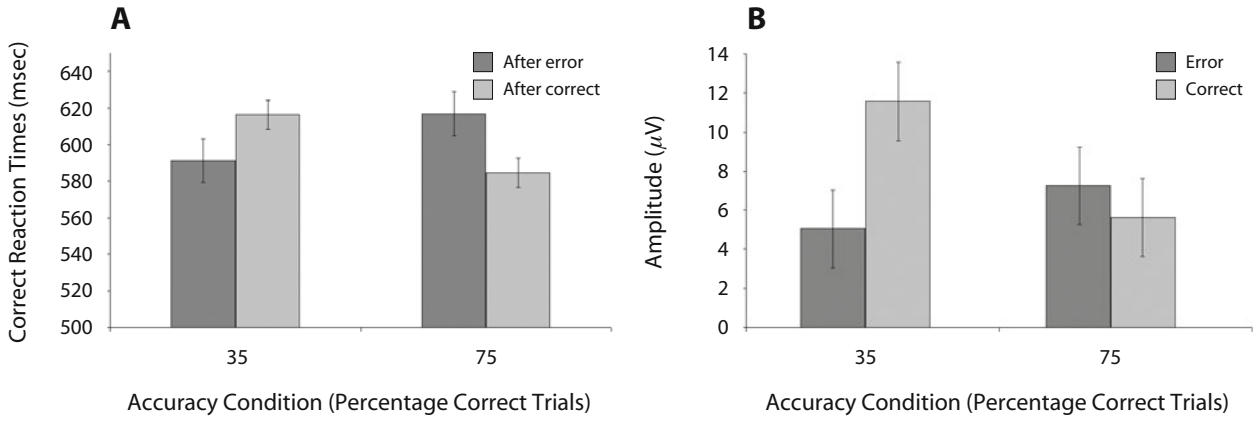

Figure 1. (A) Mean reaction times for correct trials after errors and correct responses as a function of accuracy condition. (B) P3 amplitude analyses revealed a striking similarity with the behavioral data: 35% condition, increased P3 amplitude for correct responses; 75% condition, increased amplitude for error responses.

the 35%-accuracy condition and 60 trials [7.6%] were excluded in the 75%-accuracy condition). Moreover, trials with RTs faster than 200 msec or slower than 1,000 msec (response deadline) and trials that were preceded by these trials were excluded. In total, 23.6% of the trials were excluded. The adaptation procedure worked as expected for the 75%- and 35%-correct conditions, with accuracies of 75.4% and 37.4%, respectively. The order in which the conditions were administered did not yield significant effects; therefore, we omitted this factor in subsequent analyses.

A 2 (accuracy in previous trial: correct vs. error) × 2 (accuracy condition: 35% correct vs. 75% correct) repeated measures ANOVA on correct RTs revealed no main effect of condition [$F(1,14) = 0.07$, $p = .79$] or of previous trial accuracy [$F(1,14) = 0.26$, $p = .61$]. But the interaction between accuracy condition and accuracy of the previous trial was significant [$F(1,14) = 30.04$, $p < .001$]. Replicating the study of Notebaert and colleagues (2009), we observed posterror slowing in the 75%-correct condition [$t(14) = 6.24$, $p < .001$]. Crucially, in the 35%-correct condition, we observed postcorrect slowing [$t(14) = -2.56$, $p < .05$] (see Figure 1A).

Regarding error proportions, the repeated measures ANOVA revealed a main effect of accuracy condition [$F(1,14) = 547.7$, $p < .001$] and a main effect of accuracy of the previous trial [$F(1,14) = 81.2$, $p < .001$], with more errors after errors ($M = 50.54$, $SD = 1.11$) than after correct trials ($M = 34.95$, $SD = 0.95$). The interaction between previous accuracy and accuracy condition was marginally significant [$F(1,14) = 3.19$, $p < .09$], but in line with previous findings (Notebaert et al., 2009), we observed more errors after errors than after correct trials in both accuracy conditions.

ERPs

We excluded trials that occurred before a stable accuracy level was reached (35% or 75%, depending on the condition). Additionally, trials with RTs faster than 200 msec, slower than 1,000 msec, and trials following "too slow" responses were excluded. As described in the Method section, after applying these exclusion criteria, we selected a random sample of trials for the ERP analyses on the basis of the condition with the fewest trials; this was to ensure that all conditions of interest had an equal number of trials.

ERN

The grand average ERPs of all conditions at the electrodes FCz (top of figure), Cz (middle of figure), and Pz (bottom of figure) locked to the response and feedback onset are depicted in Figure 2. At FCz, the ERPs show the ERN on error responses and the correct-related negativity (CRN) on correct responses, both peaking approximately 50 msec after response onset. Because of the procedure we are using (immediate feedback), we can in principle not dissociate response-related from feedback-related processing. However, the timing (approximately 50 msec following response/feedback onset) makes it unlikely that this component reflects feedback processing. A 2 (accuracy in current trial: correct vs. error) × 2 (accuracy condition: 35% correct vs. 75% correct) ANOVA revealed a main effect of accuracy of the current trial [$F(1,14) = 6.60$, $p < .05$], with an increased amplitude for errors ($M = -5.63$, $SD = 0.84$) in comparison with correct responses ($M = -4.30$, $SD = 0.68$) and no main effect of the accuracy condition [$F(1,14) = 0.18$, $p = .68$]. Importantly, the interaction between accuracy and accuracy condition was significant [$F(1,14) = 5.20$, $p < .05$]. Post hoc t tests revealed a significant difference between the ERN and the CRN in the 75%-accuracy condition, with an increased amplitude for the ERN [$t(14) = 2.82$, $p < .001$], whereas in the 35%-accuracy condition no amplitude differences were found [$t(14) = 0.17$, $p = .93$]. As can be seen in Figure 3, the topography voltage differences between error and correct trials show the ERN maximally over frontocentral electrodes.

FRN

As illustrated in Figure 2, there is a negative deflection peaking about 400 msec following the response and feedback onset, which is only present for error feedback.

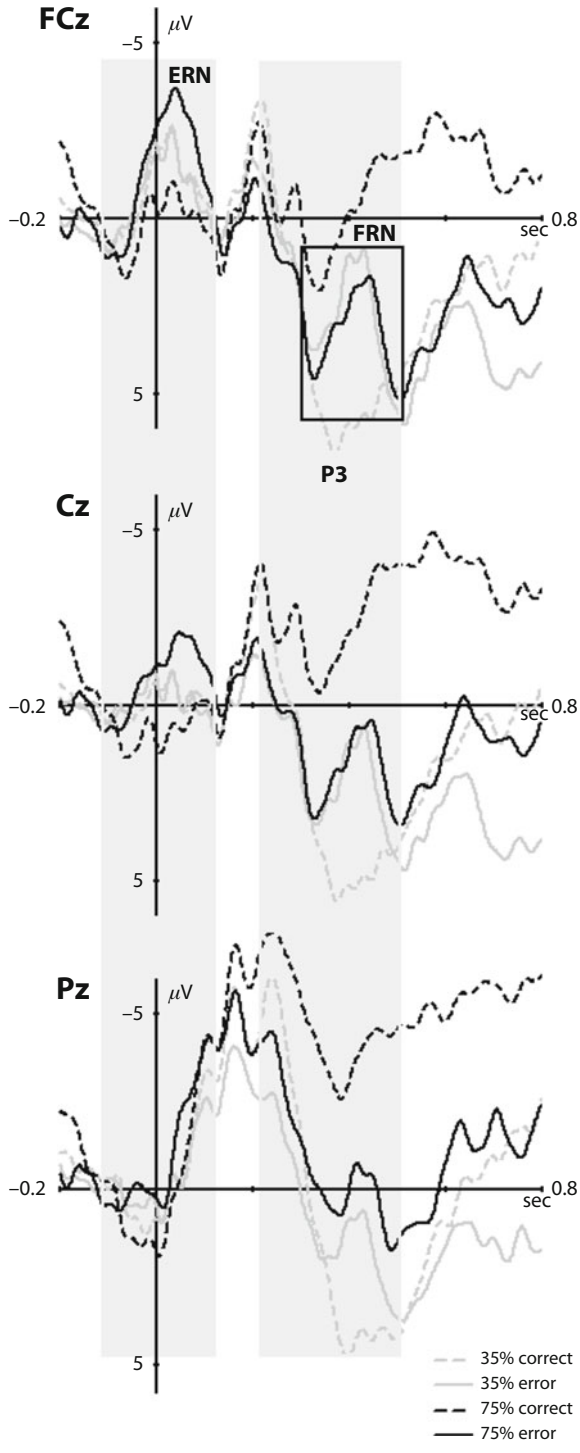

Figure 2. Grand average ERP waveforms at FCz (top of figure), Cz (middle of figure), and Pz (bottom of figure) as a function of accuracy condition and accuracy of the current trial.

A 2 (accuracy in current trial: correct vs. error) × 2 (accuracy condition: 35% correct vs. 75% correct) repeated measures ANOVA confirmed a main effect of accuracy [$F(1,14) = 10.16$, $p < .01$], showing that the FRN was larger for errors ($M = 4.24$, $SD = 0.78$) than for correct responses ($M = 2.02$, $SD = 0.37$). The effect of accuracy condition [$F(1,14) = 0.4$, $p = .53$] and the interaction did not reach significance [$F(1,14) = 1.81$, $p = .20$].

In order to rule out the possibility that the FRN variations were confounded by the differences in the P3 component, we ran the same analysis reported above after filtering the low frequencies (3- to 30-Hz band-pass). As expected, a 2 (accuracy in current trial: correct vs. error) × 2 (accuracy condition: 35% correct vs. 75% correct) repeated measures ANOVA confirmed the pattern of results previously reported: The FRN was larger for errors ($M = 4.64$, $SD = 0.73$) than for correct ($M = 1.54$, $SD = 0.38$) responses [$F(1,14) = 13.99$, $p < .01$]; but the effect of accuracy condition [$F(1,14) = 0.6$, $p = .44$] and the interaction [$F(1,14) = 0.3$, $p = .71$] were not significant. These results show that the FRN component is modulated by accuracy regardless of outcome expectancy. Importantly, the ERN and the FRN dissociate on this aspect in the sense that a clear ERN is observed only on infrequent error trials, whereas the FRN is observed on all error trials. The potential maps in Figure 3 show the scalp topography of the FRN per condition, maximally over frontocentral electrodes after filtering the low frequencies.

P3

The P3 can be observed in Figure 2. A 2 (accuracy in current trial: correct vs. error) × 2 (accuracy condition: 35% correct vs. 75% correct) repeated measures ANOVA revealed a significant main effect of accuracy condition [$F(1,14) = 6.13$, $p < .05$] and a main effect of accuracy [$F(1,14) = 6.10$, $p < .5$]. The main effect of accuracy condition showed that the P3 was larger for the 35%-accuracy condition ($M = 8.60$, $SD = 1.37$) than for the 75%-accuracy condition ($M = 6.15$, $SD = 0.87$) and the main effect of accuracy showed increased P3 for correct ($M = 8.31$, $SD = 0.99$) in comparison with error ($M = 6.41$, $SD = 1.19$) responses.

Crucially, the interaction between accuracy in current trial and accuracy condition was significant [$F(1,14) = 16.88$, $p < .01$]. Post hoc t tests revealed significant differences in the 35%-accuracy condition, with an increased amplitude for correct responses [$t(14) = 4.00$, $p < .01$], whereas in the 75%-accuracy condition we observed an increased amplitude for the error responses [$t(14) = -2.33$, $p < .05$]. As can be seen in Figure 1, the pattern of results observed for the P3 component shows a striking similarity to the pattern of the behavioral data: In the 75%-accuracy condition, where posterror slowing was found, we observed an increased P3 amplitude for errors, whereas in the 35%-accuracy condition, where a postcorrect slowing was found, we saw an increased P3 amplitude for correct responses. In other words, the data show increased P3 amplitude for unexpected outcomes regardless of accuracy.

Finally, in order to rule out the possibility that P3 variations were confounded with the differences in the FRN component, we ran the same analysis reported above after filtering high frequencies (3 Hz low pass). A 2 (accuracy in current trial: correct vs. error) × 2 (accuracy condition: 35% correct vs. 75% correct) repeated measures ANOVA replicated the previous analysis: We found a significant two-way interaction between accuracy con-

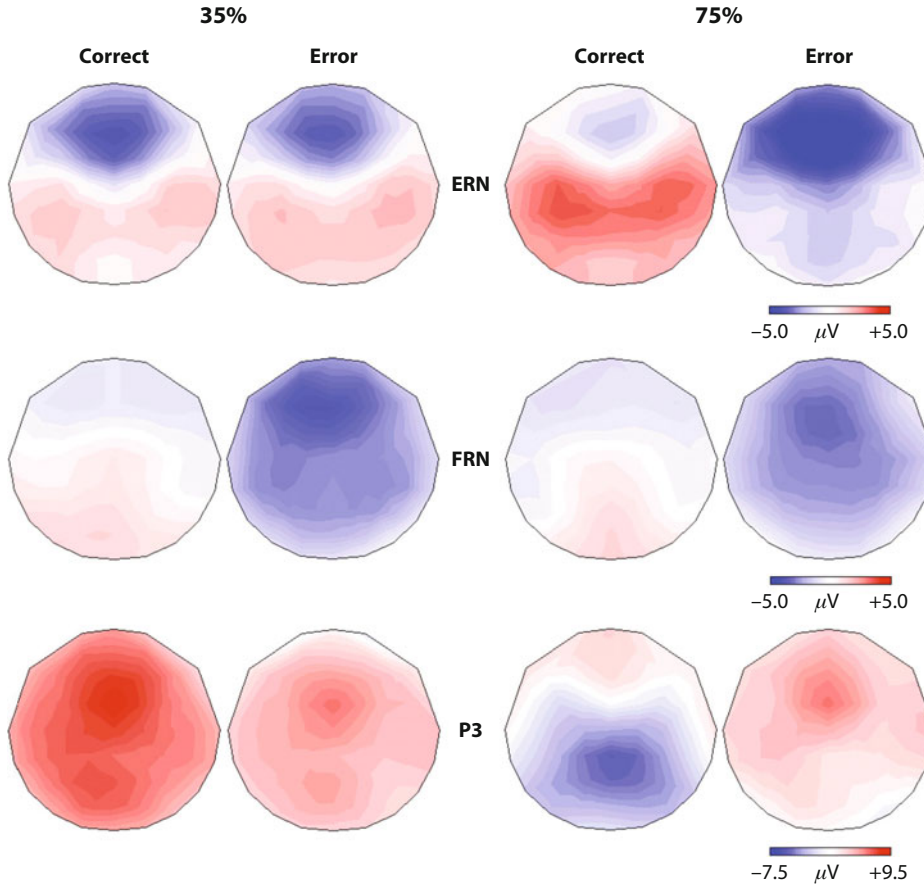

Figure 3. Scalp topography at the time point where the ERN, FRN (after filtering low frequencies), and P3 (after filtering high frequencies) are maximal. The potential maps are plotted individually by accuracy (error vs. correct) and accuracy condition (35% correct vs. 75% correct).

dition and accuracy [$F(1,14) = 21.00$, $p < .001$]. Likewise, post hoc t tests revealed that the P3 amplitude was larger in the 35%-accuracy condition for correct than for error responses [$t(14) = 3.03$, $p < .01$], whereas in the 75%-accuracy condition this component was larger for correct than for error responses [$t(14) = -3.55$, $p < .01$], confirming the pattern of results. As can be seen in Figure 3, which shows the topography voltage of this component after filtering high frequencies, the P3 was maximal over frontocentral midline electrodes.

Correlation Between ERN, FRN, P3, and Slowing

The previous analyses revealed that the P3 data match the behavioral RT data more than the ERN or the FRN data do. In order to investigate the relationship between the ERP components and the behavioral slowing effects more directly, we performed a correlational analysis. For every participant, we calculated the correlation between the ERP components (ERN, FRN, or P3) and the RT on the following trial. Therefore, we had four ERP values and four RT measures for every participant. The ERP measures were the participants' average peak measure in one condition (correct in the 75%-correct condition, error in the 75%-correct condition, correct in the 35%-correct condition, and error in the 35%-correct condition). The four RT measures were the participants' mean RTs for correct trials following this particular condition. This rendered a regression coefficient for every participant, and we then checked to see whether the standardized beta coefficient was significant over all participants (see Lorch & Myers, 1990).

As expected, the results showed that the average of the regression coefficients for the ERN ($M = -0.06$, $SD = 0.63$) [$t(14) = -0.34$, $p = .74$] and the FRN ($M = 0.07$, $SD = 0.56$) [$t(14) = 0.46$, $p = .64$] did not significantly differ from zero, whereas for the P3 ($M = 0.28$, $SD = 0.41$) [$t(14) = 2.60$, $p < .05$], the results showed a significant positive correlation, which confirms that increased P3 amplitude predicts increased RTs (see Figure 4).

DISCUSSION

With the present study, we explored the effects of outcome expectancy and accuracy on response/feedback processing. Participants performed an adaptive four-choice RT task that allowed us to manipulate error rates. Every participant completed a 35%- and 75%-accuracy condition. Behaviorally, the results showed posterror slowing in the 75%-correct condition and postcorrect slowing in the 35%-correct condition, indicating that outcome expectancy mismatch is crucial for posterror slowing.

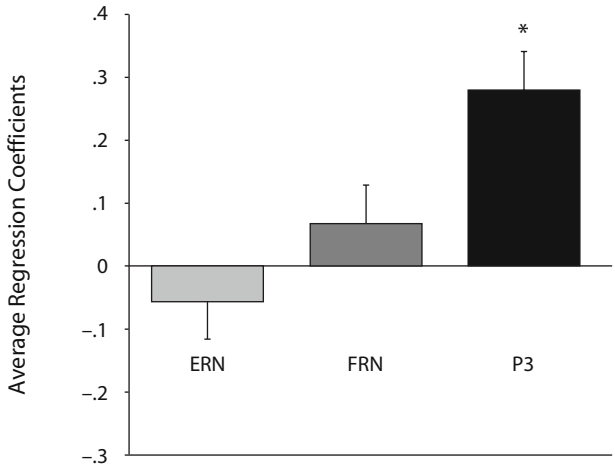

Figure 4. Average of the regression coefficients for the ERN, FRN, and P3. The figure shows that only the P3 amplitude correlates with the behavioral adjustments. The significant positive correlation indicates that increased P3 amplitude predicts increased RTs. $^{*}p < .05$.

The present study aimed at investigating the ERP correlates of these posterror and postcorrect behavioral adjustments. Consistent with our main prediction, we found that neither the ERN nor the FRN showed a pattern similar to the behavioral data. If the slowing was mainly related to error processing, one would expect a significant relationship between this behavioral effect and the ERN and FRN components. We did not find support for this hypothesis. Interestingly, we found that it was the P3 component that revealed a striking similarity with the behavioral data: In the 75% condition, we observed increased amplitude for errors, whereas in the 35% condition we observed increased P3 amplitude for correct trials. Moreover, a correlation analysis revealed that the P3 component predicts RTs on the following trial, supporting the idea that behavioral adjustments observed after the commission of an error may, at least partly, be caused by the low frequency of errors and the orienting response this generates (Notebaert et al., 2009).

The topography suggests that our P3 might be a P3a, which is sensitive to novel events that involuntarily capture attention (e.g., Friedman et al., 2001). This fits the orienting account in the sense that the slowing is believed to be related to the involuntary capture of the surprising feedback. This orienting turns attention away from the task at hand and results in slow RTs on the following trial, but considering the fact that the P3a may often be elicited in tandem with the P3b (Friedman et al., 2001; Polich, 2003), we do not want to make strong claims about differences between the P3a and the P3b.

At this point, we would have to speculate about the precise relationship between the P3 and the subsequent slowing, but an interesting suggestion is provided by the locus coeruleus (LC)–norepinephrine (NE) theory for the P3 (Niewenhuis, Aston-Jones, & Cohen, 2005). The theory holds that the P3 reflects activity of the locus coeruleus–norepinephrine system, which increases the response (or gain) to motivationally significant events. Interestingly, LC phasic responses to motivationally significant stimuli are followed by a period of relative silence, and this silent period has been related to the attentional blink effect, which is the inability to detect a new target briefly after a first target was presented (Nieuwenhuis, Gilzenrat, Holmes, & Cohen, 2005). Arguing that slowing after unexpected feedback is also related to this silent period would be bold at this stage, but the idea is certainly worth investigating.

The results of the present study seem to be at odds with previous studies reporting posterror slowing predicted by the ERN (Debener et al., 2005; Gehring et al., 1993; Marco-Pallares et al., 2008; West & Travers, 2008). It is possible that methodological differences across studies can explain part of the empirical differences. Most studies describing a relationship between the ERN and posterror slowing investigated this on a within-subjects level, whereas most studies describing no such relationship tested for correlations across participants (for an exception, see Gehring & Fencsik, 2001, who also observed no relationship in a within-subjects analysis). On top of that, there are substantial task differences—inducing different errors—among studies. There are meaningful differences between errors in our four-choice color-discrimination task, on the one hand, and the intrusion errors in a Stroop task (West & Travers, 2008) and errors in a flanker task (Gehring et al., 1993; Marco-Pallares et al., 2008), on the other hand. One potentially interesting difference is that errors in Stroop and flanker tasks are characterized by response conflict and therefore also trigger conflict adaptation mechanisms. In line with the conflict interpretation of the ERN (Yeung, Botvinick, & Cohen, 2004), what is perhaps really observed in these studies is conflict detection and postconflict slowing (e.g., Ullsperger, Bylsma, & Botvinick, 2005). In our task, on the other hand, no irrelevant conflicting information is presented and thus it is less likely that response conflict is the primary source of errors. This clearly is an interesting avenue for further research.

The ERPs showed two early components that have been traditionally associated with response monitoring activity: the ERN and the CRN, both peaking approximately 50 msec after the response/feedback onset. Analyses revealed significant differences between the ERN and the CRN in the 75%-accuracy condition, with an increased amplitude for the ERN, whereas in the 35%-accuracy condition no amplitude differences were found; in this condition we observed a reduction of the ERN amplitude at the same time that the CRN amplitude increases. Interestingly, all major theories can account for the pattern of results for the ERN. The error likelihood model (Brown & Braver, 2005) predicts increased ERN amplitude for low-error conditions in comparison with high-error conditions, the reinforcement learning theory (Holroyd & Coles, 2002) predicts increased ERN amplitude when the number of errors decreases, and Falkenstein et al. (2000) predicted increased ERN amplitude as errors become easier to detect.

Although none of the major ERN theories makes specific predictions about the CRN, our CRN data do not contradict these theories. One could argue that CRN differences reflect different probabilities for correct responses

(Brown & Braver, 2005) or differences in the error detection process (Falkenstein et al., 2000). Alternatively, the CRN can be interpreted as an ERN on correct trials caused by our stimulus degradation manipulation that compromised the representation of the correct or actual response (Coles, Scheffers, & Holroyd, 2001).

Interestingly, we observed an FRN on error trials in both accuracy conditions, unaffected by outcome expectancy. Consequently, we showed a dissociation between the ERN and the FRN in the sense that ERN interacted with outcome expectancy whereas the FRN did not, which does not support the hypotheses that the FRN is related directly to the recognition of violations of expectations (Oliveira, McDonald, & Goodman, 2007) or elicited by the first evidence that events are worse than expected (Holroyd & Coles, 2002). Instead, our findings are in line with a previous study (Hajcak et al., 2005) that also failed to find an interaction between valence and expectancy in the FRN, but future studies should explore this empirical instability (e.g., Holroyd & Coles, 2002; Holroyd, Nieuwenhuis, Yeung, & Cohen, 2003).

The present study revealed one unexpected finding—namely, a negative slow wave for correct responses in the 75%-accuracy condition (see Figure 2). Figure 2 shows that this is more pronounced at posterior sites, indicating that our frontal ERP components are unaffected by this negativity. However, rather than considering this negativity as a component, one could consider this as the absence of a positivity on these trials. In fact, these trials are the only ones in which responses and feedback are both correct and expected.

Finally, a possible limitation of this study is that in our effort to prevent expectancies (and expectancy violations) for the timing of feedback onset by giving immediate feedback, we entangled response- and feedback-related processes. This makes it in principle difficult to determine whether the differences observed in the P3 amplitude are associated with response generation or with feedback evaluation. Nevertheless, the experimental variables known to affect the P3 have been generally classified as influencing attentional processes (for a review, see Nieuwenhuis, Aston-Jones, & Cohen, 2005), and P3 amplitude has been shown to be relatively insensitive to variables related to response generation (e.g., Kok, 1978; McCarthy & Donchin, 1981). Furthermore, the scalp topography of the P3 component associated with the slowing in RTs shares similarities with the typical distribution of the P3a, also associated with attentional orienting to unexpected novel stimuli. Therefore, we are confident that the P3 is indeed triggered by the feedback rather than by the response.

In summary, our study investigated the nature of the posterror slowing effect, which has been widely regarded as a cognitive control effect, reflecting strategic adjustments. The present study provides additional support for the alternative view that posterror slowing is driven by attentional mechanisms elicited by the unexpected nature of the error (Notebaert et al., 2009). Our results show that neither the ERN nor the FRN, but the P3, is correlated to the behavioral pattern of posterror RTs. This suggests that the onset of the unexpected feedback triggers an orienting response as indexed by the P3, which in turn delays processing of the following stimulus, resulting in posterror slowing. We also demonstrated that ERN and FRN are related to error monitoring, so further studies are required to explore the functional significance of these two dissociable error (feedback) detectors.

AUTHOR NOTE

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