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Strategic control and medial frontal negativity: Beyond errors and response conflict

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Abstract

Errors in timed choice tasks typically produce an error-related negativity (ERN) in the event-related potential (ERP). The error specificity of the ERN has been challenged by studies showing a correct response negativity (CRN). Forty-five participants engaged in a flanker task in which both compatibility between flankers and target and the probability of compatible flankers were manipulated. Correct responses elicited a CRN, the amplitude of which increased with the degree of mismatch between the presence of conflict and conflict probability, even on low-conflict (compatible) trials. The fronto-central N2 component was larger on high-conflict (incompatible) correct response trials. However, in contrast to some recent accounts, this N2 was largest for highly probable stimuli. These findings suggest revision to models of the effects of conflict on response-related negativity to account for strategic adjustments made in preparation for the response.

Descriptors: Error-related negativity, Correct-related negativity, Strategic control, Conflict detection

Monitoring performance and adjusting behavior appropriately is an adaptive, critical function for the human information-processing system (e.g., Holroyd & Coles, 2002). A negative deflection has been identified in the event-related brain potential (ERP) occurring shortly after an erroneous response (e.g., Falkenstein, Hohnsbein, Hoormann, & Blanke, 1990, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993), known as the error-related negativity (ERN) or error negativity (Ne), which appears to serve this action-monitoring function (e.g., Luu, Flaisch, & Tucker, 2000). A later-occurring positive component (the error positivity; Pe) also has been identified, although its functional relationship to the ERN/Ne remains somewhat unclear (e.g., Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000).

Initial accounts linked the ERN/Ne to the operation of an error detection mechanism (e.g., Bernstein, Scheffers, & Coles, 1995; Falkenstein et al., 1990). This view recently has been challenged by studies showing small-amplitude ERN-like activity on correct trials (correct response negativity; CRN), leading some to propose that the ERN/CRN reflects a response comparison process (Vidal et al., 2000) or an emotional reaction to the response (e.g., Luu, Collins, & Tucker, 2000) rather than error detection per se. Proponents of the error detection view have argued that the CRN occurs when participants are unsure of the correctness of a given response, or when a stimulus elicits subthreshold incorrect response activation before the correct response is emitted (Coles, Scheffers, & Holroyd, 2001). Consistent with this view, several studies have reported an association between small CRNs and the presence of coactivation of correct and incorrect responses (Luu, Flaisch, et al., 2000; Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996; Vidal et al., 2000). Thus, as noted by Coles et al. (2001), there should be no ERN-like activity (i.e., CRN) if a correct response is executed under conditions where stimuli are unambiguous.

A related explanation has been offered by Cohen and colleagues (e.g., Botvinick, Braver, Barch, Carter, & Cohen, 2001; Carter et al., 1998), who posit that the ERN reflects a response conflict monitoring function performed by the anterior cingulate cortex (ACC). This hypothesis is based largely on brain imaging data showing ACC activation following both erroneous responses and correct responses on tasks involving high response conflict (e.g., the word RED shown in blue font during the Stroop task; Stroop, 1935), but not on tasks involving low response conflict (e.g., the word RED shown in red font). According to this view, errors are simply an extreme form of response conflict but are not uniquely associated with the neural activity responsible for the

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ERN. This theory posits that errors largely result from premature responding that occurs prior to complete stimulus evaluation (see also Bernstein et al., 1995; Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988), and that the correct response channel becomes activated following commission of the incorrect response in an attempt to correct or "reverse" the error (see Botvinick et al., 2001, p. 629). The resulting simultaneous activation of both correct and incorrect response channels produces conflict in the system, which is detected by the ACC. Conflict on correct response trials, in contrast, is hypothesized to be reflected in a stimulus-related negativity, the N2, which occurs prior to the response (see Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhoff, 2003; van Veen & Carter, 2002). On such trials, the correct and incorrect response channels are hypothesized to coactivate prior to the commission of the correct response, with the correct response channel ultimately dominating the response selection process.

Both the error-detection and conflict-monitoring models, then, similarly predict that unintended response activity (resulting from a mismatch between response representations in the error-detection model and from the product of correct and incorrect response activation in the conflict monitoring model) results in enhancement of fronto-central negativities in the ERP thought to emanate from the ACC. At present, however, neither model readily accounts for the presence of a CRN when stimuli are unambiguous and participants have little doubt as to the correctness of their responses (e.g., Coles et al., 2001). Monitoring processes presumably should be involved in determining not only whether the appropriate response has been activated but also how information is processed in order to produce responses. Therefore, if the ERN and CRN are indices of these monitoring processes, they also may reflect the evaluation of whether or not the strategy adopted on a particular trial is appropriate for the conditions occurring on that trial. Van Veen and colleagues (e.g., van Veen & Carter, 2002; van Veen, Cohen, Botvinick, Stenger, & Carter, 2001) have documented that conflict occurring at the level of the response, but not conflict at the level of stimulus encoding, activates the ACC and results in enhanced ERN and N2 activity. However, as noted by van Veen et al. (2001, p. 1307), the possibility that the ACC and associated fronto-central ERP components are "responsive to conflict occurring between different goal states, plans . . . activity associated with early sensory processes, decisions which occur at intermediate levels between stimulus processing and action planning, and so on" remains open. Inappropriate strategy implementation during response-conflict tasks might represent one such type of conflict to which these components are sensitive.

Strategic control processes play a pivotal role in many aspects of executive cognitive function (e.g., Posner, Petersen, Fox, & Raichle, 1988). In general, online monitoring of task demands and task performance leads individuals to adopt strategies facilitating appropriate responses. For example, using a flanker task (Eriksen & Eriksen, 1974) in which participants respond to a central target letter flanked by either compatible (same) or incompatible (different) distractor letters, Gratton, Coles, and Donchin (1992) showed that manipulating the probability of low-conflict (compatible) and high-conflict (incompatible) trials led participants to adopt either a "parallel mode" of processing (when low-conflict trials were expected and thus flanker information was likely to facilitate correct responding) or a "focused mode" (when high-conflict trials were expected and thus flanker information was likely to facilitate incorrect responding). These data indicate that prior to the presentation of a given stimulus array, participants adjust their processing strategy according to a simple probability heuristic (Gratton et al., 1988). To date, however, the role of response strategy evaluation in producing the ERN and CRN has not been established.

Findings from two recent studies have highlighted the effects of strategic control processes on ACC activation during response conflict tasks. Using a flanker task, Botvinick, Nystrom, Fissell, Carter, and Cohen (1999) found that incompatible (high-conflict) trials following compatible (low-conflict) trials produced greater ACC activation than did incompatible trials following other incompatible trials. These findings are conceptually consistent with those of Gratton et al. (1992), who showed (using response time [RT] data) that participants adjust processing strategies on a trial-by-trial basis depending upon the presence of conflict. In a similar study, Carter et al. (2000) manipulated the probability of high- and low-conflict trials to examine the influence of strategic versus evaluative processes on ACC activation. Results showed that ACC activation on high-conflict trials was greater when low-conflict trials were expected than when high-conflict trials were expected, suggesting that the ACC is more sensitive to evaluation of conflict than to strategic control processes. Importantly, compatible trials did not produce significant enhancement of ACC activation in any condition in either of these studies, reflecting an assumption of the conflict-monitoring hypothesis that "compatible trials are unlikely to induce conflict, regardless of context" (Botvinick et al., 1999, p. 180). More recently, some authors have posited that noise in the response selection process can occasionally lead to activation of the incorrect response (and thus produce conflict) even on compatible trials (see Jones, Cho, Nystrom, Cohen, & Braver, 2002). However, such occasional noise in the system would not be expected to produce systematic variance in ACC activity or associated ERP components on compatible trials. Even if such occasional noise did systematically influence ACC activity, the conflict-monitoring theory predicts that conflict on correct response trials would be reflected in the N2 and not the CRN.

To the extent that the CRN also reflects conflict in the information processing system, the results of these recent studies suggest that a CRN should occur on high-conflict trials (particularly when low-conflict trials are expected), and that no CRN is likely on low-conflict trials regardless of context. Yet, both Carter et al. (2000) and Gratton et al. (1992) found that behavioral responses are influenced on compatible trials when the context predicts incompatible trials and thus an inappropriate processing strategy is applied. In other words, strategic control processes operate during compatible as well as incompatible trials to influence response selection. If efficient and accurate behavior is the ultimate goal of the response monitoring system, then monitoring both behavioral output and higher level processes that influence response selection seems essential. Consequently, response-related negativity in the ERP could reflect a mismatch between the processing strategy employed and an internal representation of the appropriate strategy, regardless of whether or not response conflict is present (Botvinick et al., 1999) or the incorrect response is generated (cf. Coles et al., 2001).

The primary purpose of the current study was to examine the influence of strategic control on CRN amplitude, in the context of two main hypotheses. First, if CRN amplitude is sensitive only to response conflict, the CRN should be enhanced only on high-conflict (i.e., incompatible) trials relative to low-conflict (compatible) trials. Furthermore, if the CRN also is sensitive to the

use of probability information, it should be particularly enhanced on high-conflict trials encountered in the context of low-conflict (compatible) trials (e.g., Carter et al., 2000). However, if the neural processes underlying the CRN also are sensitive to response strategy evaluation, the amplitude of the CRN should also increase for compatible trials presented in the context of incompatible trials (i.e., trials on which no response conflict occurs but for which an inappropriate response strategy was implemented). A second purpose of this study was to examine these same predictions with respect to the stimulus-related N2, thought to index response conflict on correct-response trials (e.g., Botvinick et al., 2001). The conflict-monitoring theory predicts a larger N2 on incompatible relative to compatible trials, because compatible trials should not result in coactivation of correct and incorrect response channels. However, the N2 is also sensitive to manipulations of stimulus frequency (e.g., Nieuwenhuis et al., 2003), suggesting that any low-frequency event should induce conflict prior to the response and thus increase the N2. If so, the N2 also might be enhanced on compatible (low-conflict) trials presented in the context of incompatible trials. Finally, although this study was not specifically designed to test predictions concerning error-related components (ERN and Pe), it was important to measure these components to provide a basis for comparing the CRN as a function of the manipulated variables of interest (e.g., response conflict and response strategy) and the correctness of behavioral responses.

Method[1]

Participants

Participants were 45 young adults (21 women) ages 21–30 who signed informed consent and received $8.00 per hour for their participation. They were right-handed, native English speakers, who reported themselves in good health and had normal or corrected-to-normal vision. Data from 3 participants were discarded due to excessive EEG artifacts, resulting in a final sample of 42 participants.

Stimuli and Procedures

The Eriksen flanker task (Eriksen & Eriksen, 1974) as modified by Gratton et al. (1992) was used to present high- and low-conflict stimuli to participants at varying levels of conflict probability. Each trial consisted of one of four 5-letter arrays (HHHHH, SSHSS, SSSSS, or HHSHH). Participants were instructed to respond as quickly and accurately as possible to the central "target" letter by pressing a key with one hand if it was an "H" and another key with the other hand if it was an "S" and to ignore the "flanker" (noise) letters. Note that in each array the flanker letters were either compatible (low-conflict) or incompatible (high-conflict) with the correct response. The association between target letter and responding hand was counterbalanced across participants. Each array was presented for 200 ms, with a 2500-ms intertrial interval, on a monitor positioned 60 cm in front of the participant. A fixation cross, placed just below the target letter, was present throughout the experiment. Although the probability of each target letter was kept at 50% across the experiment, the flanker-type probability (and thus conflict probability) was varied across blocks as follows: 50/50 (equal proportions of compatible and incompatible trials), 80/20 (80% compatible trials), and 20/80 (20% compatible trials), resulting in expect-neutral (EN), expect-compatible (EC), and expect-incompatible (EI) conditions, respectively. The order of these conditions was randomized for each participant. Previous research has demonstrated that the size of the noise-compatibility effect (i.e., slower response times to incompatible vs. compatible trials) depends upon the relative probability of incompatible flankers (e.g., Gratton et al., 1992).

Participants first completed three practice blocks of 60 trials each of the flanker task (EN condition), with instructions equally emphasizing response speed and accuracy. Next, participants completed twenty-four 60-trial blocks of the experimental task, with a short break after block 12.

Electrophysiological Recording

The electroencephalogram (EEG) was recorded from 20 scalp locations according to the 10-20 electrode placement system referred on-line to the left mastoid. An average mastoid reference was derived off-line. Vertical and horizontal electrooculogram (EOG) was recorded bipolarly using electrodes placed above and below the right eye and 2 cm external to the outer canthus of each eye, respectively. Ocular artifacts were corrected off-line using a standard procedure (Gratton, Coles, & Donchin, 1983). Trials including amplitude values larger than ± 75 μV following ocular artifact rejection were considered artifacts and were excluded from analyses. The EEG and EOG were recorded with a 0.01–30 Hz bandpass filter for epochs lasting 1400 ms, beginning 100 ms, before the stimulus, at a digitizing rate of 100 Hz. Impedance was kept below 10 kΩ.

Results

Stimulus- and response-related average waveforms were computed for each electrode, participant, and condition. The average voltage value for the 100 ms preceding the stimulus (for stimulus-related averages) and the average voltage value for the 50 ms preceding the response (for response-related averages) was subtracted from the waveforms prior to all further analyses. Initial analyses comparing the size of components of interest at midline scalp locations indicated that ERN/CRN amplitude was largest at the Cz electrode site, and the Pe was largest at Pz (see Figure 1). Based on this finding, the ERN/CRN was quantified as the average voltage value (with respect to the baseline) at the Cz electrode for the interval between 10 and 110 ms after the response. The Pe was quantified as the average voltage value at Pz between 200 and 400 ms after the response. The stimulus-related N2 was largest overall at Fz, but as in prior research (see van Veen & Carter, 2002) the component also was quite pronounced at Cz (see Figure 2). Inspection of the single-subject average waveforms indicated that the component peaked between 250 and 350 ms poststimulus for each subject. Therefore, the N2 was

[1]The data reported here were collected in the context of a larger project examining effects of alcohol on cognition. However, the focus of the current report is on the influence of strategic control on response-related ERPs, and not on alcohol effects. Data related to alcohol effects in this experiment are reported in a separate paper (Bartholow et al., 2003) along with complete details on those aspects of the procedure related to alcohol administration. Interestingly, a set of additional analyses showed no significant effects of alcohol on the amplitudes of the ERN/CRN and Pe. As such, we focus here exclusively on the findings associated with our other manipulations.

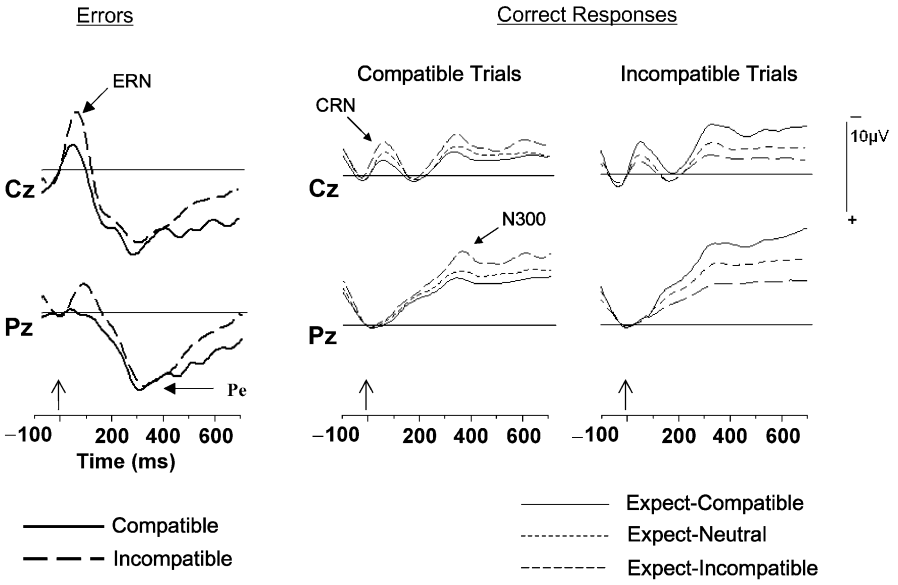

Figure 1. Response-related ERP waveforms elicited by errors (left panel) and correct responses (right panel) on compatible and incompatible trials. The vertical arrow indicates onset of behavioral responses.

quantified at both Fz and Cz as the average voltage value for the interval between 250 and 350 ms poststimulus.[2]

The percent of the total number of available trials in which errors occurred was low across participants (from $<1\%$ to 24.6%, $M = 5.2\%$, $SD = 0.06$), owing in part to the fact that, differently from previous studies (e.g., Gratton et al., 1988, 1992), instructions emphasized both speed *and* accuracy. Therefore, although we calculated separate waveforms for errors elicited on compatible and incompatible trials for each participant, attempting to further divide errors according to expectancy conditions resulted in empty cells in the full design for many participants. However, we identified a subset of participants ($n = 7$) who committed at least five errors in each condition, and whose data therefore could be examined as a function of both compatibility and expectancy factors. Hence, comparison of electrocortical activity elicited by correct responses and errors, in addition to RTs associated with correct trials versus errors, were carried out using 2 (compatibility: compatible, incompatible) $\times$ 2 (response type: correct, error) repeated measures ANOVAs. The effects of compatibility and expectancy on all dependent variables for correct response trials, as well as ERN amplitudes among the subsample with sufficient errors in each condition, were examined using 2 (compatibility: compatible, incompatible) $\times$ 3 (expectancy: expect-compatible, expect-neutral, expect-incompatible) repeated measures ANOVAs. The analysis examining the stimulus-related N2 also included an additional two-level electrode factor to test whether this component was differentially influenced by our manipulations at the Fz and Cz electrodes. Analysis of the error rate data was carried out using the arcsine of the square root of the percent of errors in each condition, which produces a more normal distribution suitable for ANOVA. Probability levels (p values) associated with repeated measures analyses including more than two levels were adjusted using the Greenhouse–Geisser correction.

Behavioral Performance

The ANOVA comparing RTs on correct trials versus errors showed significant main effects of compatibility, $F(1,41) = 8.46$, $p < .01$, and response type, $F(1,41) = 19.06$, $p < .001$, which were

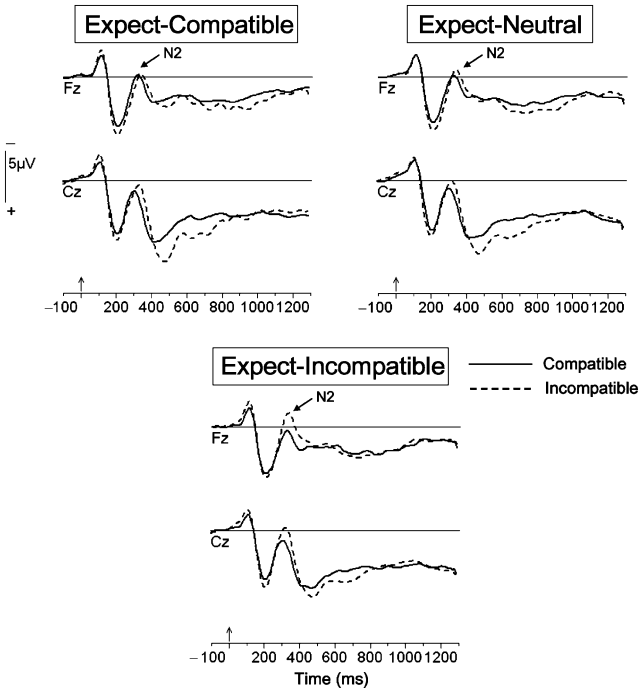

Figure 2. Stimulus-related ERP waveforms elicited on correct trials as a function of expectancy condition and compatibility. The vertical arrow indicates onset of the stimulus array. Note that the N2 component is largest in the expect-incompatible condition.

[2]As an alternative to the average voltage approach reported here, we also examined peak amplitudes by selecting for each participant the largest amplitude value associated with each component of interest. Analyses of these peak values produced results highly similar to those we report, but with slightly larger F values for most analyses. To maintain consistency with some previous reports (e.g., Gehring, Gratton, Coles, & Donchin, 1992; Gehring & Knight, 2000), we opted to present the findings based on average amplitude values.

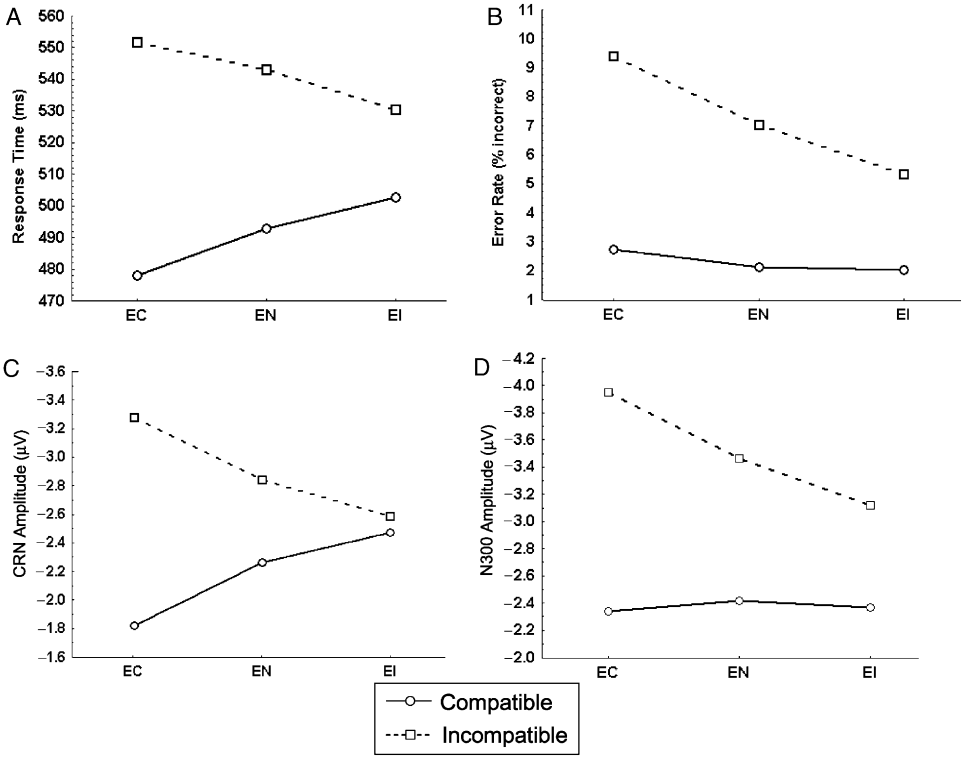

Figure 3. Modulation of the noise compatibility effect by expectancy in reaction times (correct trials; A), response accuracy (B), CRN amplitude at Cz (C), and N300 amplitude at Cz (D). EC = expect compatible; EN = expect neutral; EI = expect incompatible.

qualified by a significant Compatibility × Response Type interaction, $F(1,41) = 15.40$, $p < .01$. Simple effect tests conducted within response type showed that whereas correct responses were faster on compatible trials ($M = 493$ ms) than on incompatible trials ($M = 544$ ms), $t(41) = 20.0$, $p < .001$, errors were made equally quickly on both types of trials (Ms = 478 and 470 ms, respectively), $t(41) = 0.53$, $p > .50$.

Analysis of correct response RTs showed a main effect of compatibility, $F(1,41) = 400.01$, $p < .001$, and a significant Expectancy × Compatibility interaction, $F(2,82) = 110.39$, $p < .0001$, $\varepsilon = .75$, indicating that the size of the noise-compatibility effect depended upon the probability of incompatible noise. Inspection of Figure 3A shows that the noise-compatibility effect in RT decreased across expect-compatible ($M = 73.6$ ms), expect-neutral ($M = 50.1$ ms), and expect-incompatible ($M = 27.7$ ms) conditions (though the effect was significant in each condition, Fs > 137.00, ps < .001). Follow-up contrasts examining the linear expectancy effect for compatible and incompatible trials showed that increasing the probability of incompatible noise increased RTs to compatible trials, $F(1,41) = 62.50$, $p < .01$, and decreased RTs to incompatible trials, $F(1,41) = 45.07$, $p < .01$.

A similar ANOVA examining transformed error rates showed significant main effects for compatibility, $F(1,41) = 76.79$, $p < .001$, and expectancy, $F(2,82) = 15.48$, $p < .001$, $\varepsilon = .83$, which were qualified by a marginal Expectancy × Compatibility interaction, $F(2,82) = 3.05$, $p < .06$, $\varepsilon = .98$. Inspection of Figure 3B shows the untransformed error rates (for ease of interpretation) as a function of condition. The noise-compatibility effect decreased from expect-compatible ($M = 6.64\%$) to expect-neutral ($M = 4.88\%$) to expect-incompatible ($M = 3.29\%$) conditions. Follow-up analyses indicated that error rates for incompatible trials decreased with increasing probability of incompatible noise, $F(1,41) = 26.20$, $p < .001$, whereas error rates for compatible trials were not significantly affected by probability, $F(1,41) = 2.81$, $p > .10$.

Stimulus-Related N2 Analysis

Stimulus-related waveforms from correct response trials are presented in Figure 2. Recall that the conflict-monitoring theory predicts that response conflict on correct trials will be manifest in enhancement of the stimulus-related N2, and that conflict reflected in the N2 can arise either from high-conflict trials (i.e., trials likely to elicit activation of both correct and incorrect response channels) or from trials that occur with low frequency (Braver, Barch, Gray, Molfese, & Snyder, 2001; Jones et al., 2002; Nieuwenhuis et al., 2003). The ANOVA examining N2 amplitude produced a significant main effect of compatibility, $F(1,41) = 16.25$, $p < .001$, indicating that incompatible trials ($M = -0.3\ \mu$V) elicited a significantly larger N2 than compatible trials ($M = 1.0\ \mu$V), consistent with the notion that conflict on correct trials is reflected in this component. This main effect was qualified by a significant Compatibility × Electrode interaction, $F(1,41) = 15.43$, $p < .001$. Simple effect analyses showed that the compatibility effect was larger at Cz ($M = 1.0\ \mu$V), $t(41) = 4.41$, $p < .001$, $d = 1.38$, than at Fz (0.5 μV), $t(41) = 3.00$, $p < .01$, $d = 0.94$. Finally, all of these lower-order effects were qualified by a significant Expectancy × Compatibility × Electrode interaction, $F(2,82) = 7.07$, $p < .01$, $\varepsilon = .75$. Mean amplitudes associated with this effect are presented in Table 1, which shows that the compatibility effect was significant regardless of expectancy at Cz, but was significant only in the expect-incompatible condition at Fz.

Table 1. *Mean Amplitude of the Stimulus-Related N2 as a Function of Electrode, Expectancy, and Compatibility Factors*

	Fz			Cz		
	Expect-compatible	Expect-neutral	Expect-incompatible	Expect-compatible	Expect-neutral	Expect-incompatible
Compatible	0.4_a	0.4_a	0.9_a	1.7_a	1.3_a	1.5_a
Incompatible	0.4_a	0.3_a	-0.4_b	0.7_b	0.7_b	0.1_b

Note: Means within each column that do not share a subscript differ significantly, $p < .001$ (Tukey's HSD test). Expect-compatible = 80% compatible trial blocks; Expect-neutral = 50% compatible trial blocks; Expect-incompatible = 20% compatible trial blocks. Smaller (less positive) means indicate a larger N2 component.

Response-Related ERP Data

ERN/CRN amplitude. Both the error-detection and conflict-monitoring perspectives predict larger response-related negativity for errors than for correct trials. Consistent with this prediction, the ANOVA comparing ERN and CRN amplitudes showed a significant main effect of response type, $F(1,41) = 11.06$, $p < .001$, indicating that the ERN was significantly larger ($M = -5.0$ μV) than the CRN ($M = -2.6$ μV). A main effect of compatibility, $F(1,41) = 8.67$, $p < .01$, indicated that incompatible trials elicited larger negativity than compatible trials in both components (see Figure 1). These main effects were qualified by a Response Type $\times$ Compatibility interaction, $F(1,41) = 4.75$, $p < .05$. Planned contrasts indicated that the compatibility effect was larger in the ERN (mean difference = 3.2 μV) than in the CRN (mean difference = 0.6 μV), though it was significant for both components, Fs$(1,41) > 6.90$, ps $< .01$ (see Figure 1).

More central to the current research is whether strategic control of response selection would influence the CRN. Support for a response conflict account of the CRN would be indicated if the component was larger on incompatible trials in the expect-compatible condition than in the expect-incompatible condition, *and* if compatible trials did not elicit a CRN regardless of expectancy condition. Support for the strategy selection hypothesis, however, would be indicated by larger CRN amplitude on all trials for which inappropriate response strategies have been selected, regardless of the presence of response conflict.

The significant main effect of compatibility for correct trials was qualified by an Expectancy $\times$ Compatibility interaction, $F(2,82) = 6.99$, $p < .01$, $\varepsilon = .88$ (Figure 3C). Importantly, follow-up contrasts indicated that the CRN on compatible trials was significantly larger in the expect-incompatible ($M = -2.5$ μV) than the expect-compatible condition ($M = -1.8$ μV), $t(41) = 1.95$, $p = .05$. Conversely, the CRN on incompatible trials was larger in the expect-compatible ($M = -3.3$ μV) than in the expect-incompatible condition ($M = -2.6$ μV), $t(41) = 2.24$, $p < .05$. Planned comparisons indicated that although the noise-compatibility effect was significant in the expect-compatible (mean difference = 1.3 μV) and expect-neutral (mean difference = 0.6 μV) conditions, ts$(41) = 4.02$ and 2.33, ps $< .01$, respectively, it was not significant in the expect-incompatible condition (mean difference = 0.1 μV), $t(41) = 0.35$, n.s.

The fact that the amplitude of the CRN closely paralleled the RT and accuracy results across stimulus types (see Figure 3) raises the possibility that changes in CRN amplitude were due to differences in task difficulty across conditions, rather than to some form of conflict in the response monitoring and output system. To rule out this potential confound, we conducted a median-split analysis in which each participant's trials were sorted separately for each condition on the basis of whether the RT was faster or slower than their median for that condition, indicating that the trial was relatively easy or difficult, respectively (see also Johnson, Barnhardt, & Zhu, 2004). Doing so allowed us to essentially reverse the difficulty relationship between compatible (generally easier) and incompatible (generally harder) conditions by comparing fast incompatible trials with slow compatible trials in the EC and EI conditions. The waveforms presented in Figure 4 A indicate that despite this reversal, "easy" (i.e., fast) incompatible trials still produced a larger CRN in the EC condition than did "difficult" (i.e., slow) compatible trials (Ms $= -3.3$ and -2.0 μV, respectively), $F(1,41) = 10.52$, $p < .01$. Importantly, these easy incompatible trials were associated with faster responses ($M = 478$ ms) compared to difficult compatible trials ($M = 545$ ms), $F(1,41) = 65.48$, $p < .001$, indicating that the increased CRN amplitude on easy incompatible trials shown in Figure 4A was apparent despite significantly faster RTs on those trials. Compatible trials also elicited larger CRN in the EI condition than in the EC condition regardless of RT (see Figure 4B), although the difference was somewhat larger on slow RT trials (M difference = 1.7 μV; $t[41] = 3.42$, $p < .01$) than on fast RT trials (M difference = 1.0 μV; $t[41] = 1.90$, $p = .05$). However, the Expectancy $\times$ RT group interaction testing whether this slope differed as a function of RT was not significant, $F(2,82) = 1.20$, $p > .30$, $\varepsilon = .98$. The overall Expectancy $\times$ Compatibility $\times$ RT Group interaction also was not significant, $F(2,82) = 1.86$, $p > .16$, $\varepsilon = .99$, indicating that CRN amplitudes followed the general pattern shown in Figure 3C regardless of task difficulty as indexed by RT.

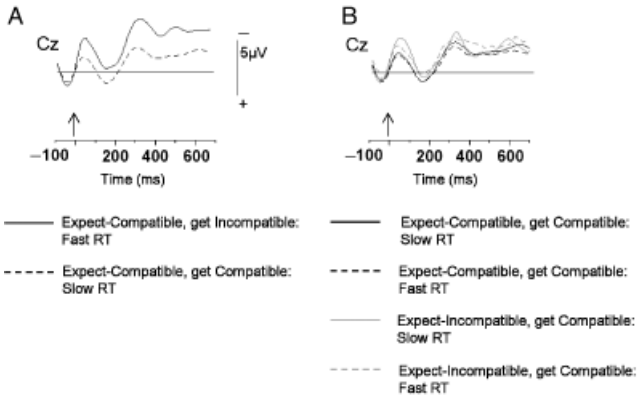

Figure 4. A: Response-locked ERP waveforms elicited in the expect-compatible condition, showing CRN amplitude for incompatible trials with relatively fast RTs compared to compatible trials with relatively slow RTs (median split). B: The CRN on compatible trials as a function of expectancy condition and RT (median split). The vertical arrow on the timeline represents response onset.

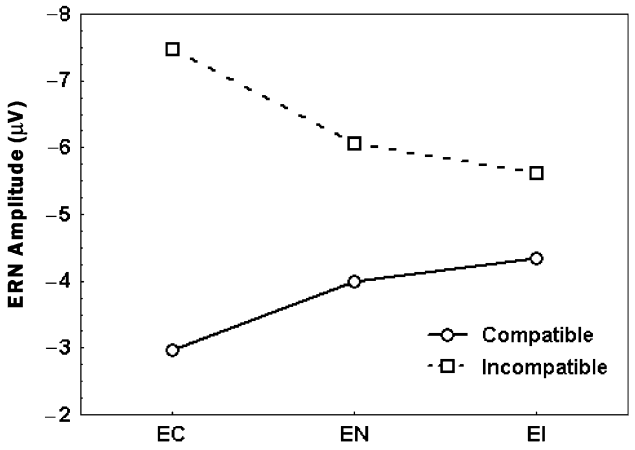

Figure 5. Modulation of the noise compatibility effect by expectancy in ERN amplitude for a subset of participants ($n = 7$) whose data contained at least five error trials in each condition. EC = expect-compatible; EN = expect-neutral; EI = expect-incompatible.

We also examined whether the amplitude of the ERN would be influenced by the compatibility and expectancy manipulations in a manner similar to the CRN, among the subsample of participants with at least five errors in each condition. This analysis produced a marginally nonsignificant Expectancy × Compatibility interaction, $F(2,12) = 3.37$, $p = .08$, $\varepsilon = .78$. Inspection of the means associated with this interaction (see Figure 5) shows that the pattern was nearly identical to that seen for the CRN (see Figure 3C). As with the analysis of the CRN, follow-up contrasts indicated that the ERN on incompatible trials was significantly larger in the expect-compatible condition ($M = -7.5$ μV) than in the expect-incompatible condition ($M = -5.6$ μV), $F(1,6) = 5.48$, $p = .05$, $d = 1.91$. The ERN on compatible trials was somewhat larger in the expect-incompatible ($M = -4.4$ μV) compared to the expect-compatible condition ($M = -3.0$ μV), but this difference was not significant, $F(1,6) = 1.79$, $p = .23$, $d = 1.09$. Also like the CRN, the noise compatibility effect in the ERN was significant in the expect-compatible (mean difference = 4.5 μV), $t(6) = 4.35$, $p < .01$, and expect-neutral (mean difference = 2.1 μV) conditions, $t(6) = 2.38$, $p = .05$, but not in the expect-incompatible condition (mean difference = 1.2 μV), $t(6) = 0.86$, $p > .40$.

Error positivity (Pe). Unlike the ERN and CRN, which were similar in polarity but simply larger for errors, the waveforms in Figure 1 indicate that errors and correct responses elicited very different ERP activity during the epoch associated with the Pe. An initial ANOVA confirmed that the Pe associated with errors ($M = 6.8$ μV) was significantly different from the prolonged negativity associated with correct responses during this epoch ($M = -5.1$ μV), $F(1,41) = 138.58$, $p < .001$, which some have labeled an N300 (Vidal et al., 2000). Thus, we conducted separate analyses to examine the effects of our manipulations on the Pe elicited by errors and on the N300 associated with correct responses.

Compatibility did not significantly affect Pe amplitude at Pz, $F(1,41) = 0.59$, $p > .50$. Given the apparent lack of variability in the data at Pz, we also examined the Pe data from the Cz electrode, but here too the compatibility effect was not significant, $F(1,41) = 2.27$, $p > .13$. Initial inspection of the N300 showed that it was largest at the Pz electrode. We therefore first examined the effects of our manipulations on N300 amplitudes using a 2 (compatibility) × 3 (expectancy) repeated ANOVA focused on data from Pz. This analysis showed no significant effects, although incompatible trials elicited marginally more negativity ($M = -5.3$ μV) than did compatible trials ($M = -4.9$ μV), $F(1,41) = 3.66$, $p < .07$. Given the lack of effects of our manipulations at Pz, we also examined the data at Cz using a similar ANOVA. This analysis showed a significant main effect of compatibility, $F(1,41) = 14.17$, $p < .001$, indicating that incompatible trials elicited a larger N300 ($M = -3.5$ μV) than did compatible trials ($M = -2.4$ μV). This analysis also showed a marginal Expectancy × Compatibility interaction, $F(2,82) = 2.95$, $p < .06$, $\varepsilon = .99$ (see Figure 3D). Follow-up linear contrasts indicated that whereas the N300 on compatible trials was unaffected by expectancy condition ($M = -2.4$ in each condition), $F(1,41) = .01$, $p > .90$, incompatible trial amplitudes decreased significantly as the probability of incompatible trials increased (Ms = −3.9, −3.5, and −3.1 μV, respectively), $F(1,41) = 6.95$, $p = .01$.

Discussion

The main purpose of this experiment was to examine variability in the CRN as a function of response strategy selection, as a means of testing whether response conflict and other types of conflict elicit increased medial-frontal negativity in the ERP. As in previous research, RTs were faster overall for errors than for correct responses, and error trial RTs were not influenced by compatibility (e.g., Pailing, Segalowitz, Dywan, & Davies, 2002; Scheffers & Coles, 2000), consistent with the notion that responding prior to complete analysis of the stimulus array contributes to errors (see also Falkenstein et al., 1991; Gratton et al., 1988, 1992). Correct response RTs indicated that selection and implementation of response strategies was influenced by the probability of high-conflict trials, with longer RTs to low-probability stimuli indicating less optimal strategy implementation (e.g., Carter et al., 2000; Gratton et al., 1992) and greater conflict.

More importantly, the amplitude of the CRN was also influenced by the probability manipulation. Most intriguing is that this effect occurred for both high-conflict (incompatible) and low-conflict (compatible) trials, suggesting that conflict between the response strategy implemented and an internal representation of the appropriate strategy can elicit a CRN, regardless of the presence of response conflict at the level of behavioral output (van Veen et al., 2001) or partial error processing (Coles et al., 2001). This notion is consistent with the assertions of other researchers (e.g., Gehring & Fencsik, 2001) that conflict is related not only to the properties of a particular stimulus but also to anticipatory actions and motor programs (e.g., Coles, Gratton, Bashore, Eriksen, & Donchin, 1985; Gratton et al., 1988; Luu, Flaisch, et al., 2000). These data suggest a broadening of current models of conflict monitoring (e.g., Botvinick et al., 2001) to account for conflict related to the strategic use of information to guide adaptive responding and for conflict in the response-locked waveform on correct trials.

The amplitude of the ERN also appears sensitive to response strategy selection. As with the CRN, the ERN was largest following errors on high-conflict (incompatible) trials occurring in the context of more probable low-conflict (compatible) trials, and smallest following errors to highly probable low-conflict trials. Bernstein et al. (1995) similarly showed that ERN amplitude

depended upon subjects' response strategy (hand vs. finger preference, in that case). However, the current ERN findings should be interpreted with caution due to the relatively small number of errors generated overall.

The current data suggest revision to aspects of error-detection theory (e.g., Coles et al., 2001; Falkenstein et al., 1990; Holroyd & Coles, 2002). Although the component was larger following errors, response-related negativity was clearly present following both correct and incorrect responses. Coles and colleagues (e.g., Coles et al., 2001) have argued that response-related negativity on correct trials results when participants believe they have erred or are uncertain of their response (e.g., when told to respond after a specified time delay, or when stimulus degradation makes response outcomes uncertain). This seems an unlikely explanation for the current data given that participants were not asked to estimate response time and stimuli were clearly visible. Moreover, it seems unlikely that participants would believe they had erred on trials involving no stimulus ambiguity or conflict at the response output level. RT data indicated that participants were using a focused response strategy in the EI condition and thus were unlikely to have activated the incorrect response, even at a subthreshold level, on compatible trials. A more likely explanation, we argue, is that participants recognized that their processing strategy, selected on the basis of probability information, was not optimal on low probability trials. Thus, the negativity observed on correct trials likely represents not error detection but rather a more general process related to the recognition of an inappropriate response strategy (in which an erroneous outcome would represent the most flagrant case of mismatch). Taken together, the results of the CRN and ERN analyses suggest that medial-frontal negativity in the ERP is the product of conflict at multiple stages of processing unfolding over time, including strategy representations, strategy implementation, and response representations. Thus, on both correct and error trials, conflict occurring both before the response (partially reflected in stimulus-locked averages) and after it (reflected in response-locked averages) needs to be considered to more fully account for the amplitude of these components.

Our findings also provide evidence of dissociation between the two components most associated with error processing (ERN and Pe; see also Vidal et al., 2000). Whereas the ERN and CRN were similar in polarity and both were similarly influenced by compatibility and expectancy factors, only errors produced a clear Pe. Correct responses—even the slowest correct responses associated with the largest CRNs—elicited an N300 during this later epoch. Furthermore, in contrast to the ERN, Pe amplitude was not significantly affected by compatibility, suggesting different functions for these components (Falkenstein, Hohnsbein, & Hoormann, 1996). It is important to note, however, that the current paradigm was not ideally suited for comparison of these components, given the relatively low error rate and the fact that the error rate differed significantly across conditions, which likely influenced the amplitude of the ERN. Nevertheless, these data add to a growing body of evidence suggesting important differences in the functional significance of the ERN and Pe (e.g., Kiehl, Liddle, & Hopfinger, 2000; van Veen & Carter, 2002), and in particular suggesting that whereas the ERN is not specific to error processing, the Pe is (also see Vidal et al., 2000).

The current data also provide some boundary conditions for the N300, a component that has received very little attention in the empirical literature on conflict and cognitive control (but see Vidal et al., 2000). In contrast to the CRN, the amplitude of the N300 was similar following compatible trials regardless of expectancy condition. However, N300 on incompatible trials decreased significantly as the probability of conflict increased. To the extent that encountering conflict signals a need to increase vigilance in order to ensure adaptive responding, these findings suggest that the N300 might reflect processes associated with bringing cognitive control to bear on future responding. If so, the N300 may be similar to the stimulus-related negative slow wave (NSW) identified in other research on cognitive control (e.g., West & Alain, 1999, 2000). West and Alain showed that the amplitude of this component is larger on trials of response conflict tasks (e.g., the Stroop task) in which cognitive conflict is successfully resolved (see also Curtin & Fairchild, 2003). In addition, Bartholow, Dickter, and Sestir (2004) found that the amplitude of the NSW (particularly at frontal electrode locations) correlated significantly with successful behavioral inhibition in a go–stop task, suggesting that this component reflects neural implementation of cognitive control. Of course, these speculations concerning the potential relationship between the stimulus-related NSW and response-related N300 remain to be tested in future research.

In contrast to predictions derived from some recent reports (e.g., Braver et al., 2001; Nieuwenhuis et al., 2003), the frontal stimulus-related N2 on correct trials, posited to reflect both response conflict and conflict associated with stimulus infrequency, was larger on incompatible (high-conflict) trials only when those trials were most frequent. The more centrally focused N2 (at Cz) was insensitive to the probability manipulation altogether. The fact that previous reports have shown larger N2 amplitudes as a function of stimulus infrequency could be at least partially attributable to differences in task parameters in the current study. For example, the N2 effects reported by Nieuwenhuis et al. (2003) were elicited using a go/no-go task (also see Heil, Osman, Wiegelmann, Rolke, & Henninghausen, 2000). These authors reported that the differing task demands on go and no-go trials significantly contributed to the N2 effect they reported, and that an implicit bias toward the go response, resulting in increased response conflict on no-go trials, was responsible for the asymmetry in the N2 that they observed. This type of bias did not exist in the current paradigm, given that participants responded on every trial and could not predict which response (left or right) would be required on a given trial. Thus, it appears that the stimulus-related N2 is sensitive to response conflict and is largely unaffected by conflict at other stages, whereas the CRN is sensitive to both response conflict and conflict associated with inappropriate response strategy.

Despite the importance of the current data for models of conflict and error processing, these findings leave a number of questions unresolved. First, stability of the effects of conflict probability on the ERN should be examined in future research using a paradigm that reliably elicits a larger number of errors. Second, the current study can say little regarding the neural sources of the components of interest, particularly whether the ERN and CRN share a common source. Researchers have debated the functional relationship between the ERN and CRN, with some studies showing that they are essentially the same component, sharing both temporal and spatial properties (e.g., Vidal, Burle, Bonnet, Grapperon, & Hasbroucq, 2003), and others providing evidence that errors activate somewhat different medial frontal areas on high-conflict trials than do correct responses (e.g., Ullsperger & von Cramen, 2001). The current findings are generally consistent with the idea that the ERN and

CRN share a common psychological foundation in that both were affected similarly by our experimental manipulations. The apparent inconsistency between the current findings and those of Carter et al. (2000), who claimed that ACC activity is not significantly enhanced on low-conflict trials under high conflict probability conditions, casts some doubt over the assertion that the scalp-recorded CRN is produced by the same ACC activity associated with response conflict. This inconsistency could indicate that the CRN does not reflect conflict per se, but rather expectancy violation. The CRN was larger to incompatible trials when compatible trials were expected, and to compatible trials when incompatible trials were expected. However, a strict expectancy violation account would predict generally equivalent CRN amplitudes to all unexpected stimuli, which is not what we found (compare, e.g., the amplitude of the CRN to incompatible arrays in the EC condition with that to compatible arrays in the EI condition in Figure 3C). Moreover, although Carter et al. did not report a significant increase in ACC activation for compatible trials in the expect-incompatible condition, the pattern of means they reported (see their Figure 2) is comparable to that seen here, and thus suggests a similar process. Nevertheless, future studies should be aimed at more firmly establishing potential links between the CRN and ERN and their respective neural generators.

A related issue concerns the functional significance of all of the components examined here. Most models of cognitive control posit that the role of the ACC is to detect conflict in the cognitive system, indicating the need to engage other brain regions in the prefrontal cortex to implement strategic control (e.g., Botvinick et al., 2001; Kerns et al., 2004; MacDonald, Cohen, Stenger, & Carter, 2000). To the extent that all of the medial frontal negativities examined here are mediated by some component(s) of ACC function, it may be that they all represent variations of a neural "distress signal" sent by the ACC to other structures in the service of enhancing cognitive control, as proposed by others (e.g., Bush, Luu, & Posner, 2000; Gehring & Willoughby, 2002; Luu, Collins, et al., 2000; Tucker, Luu, Frishkoff, Quiring, & Poulsen, 2003). In the case illustrated by the current data, recognition that a particular strategy is not optimal for executing a response may be a source of distress, though to a lesser degree than committing an error. In the expect-compatible condition when participants are using a parallel processing strategy (i.e., processing flankers along with the target), encountering incompatible trials signals the inappropriateness of that strategy for inhibiting incorrect responses and thus leads to a large CRN. In contrast, encountering compatible trials in the expect-incompatible condition also signals a need to adjust strategy, but only because the current (focused) strategy is not optimally facilitating fast and accurate responding. In other words, using a focused strategy under these conditions will not inhibit proper behavior, it will simply make behavior suboptimal. Thus, the distress associated with these trials is less than in the former scenario, producing a smaller CRN. We contend that the ACC may be responsible for detecting or computing this distress, and detection of response conflict, maladaptive response strategy, or error detection all may be sufficient to generate it.

Recently, the strategic adjustment account of the modulation of the noise compatibility effect originally observed by Gratton et al. (1992), and replicated here in both behavioral performance and the ERN and CRN, has been challenged by a "repetition priming" account (Mayr, Awh, & Laurey, 2003). According to this view, modulation of the noise-compatibility effect by probability is completely dependent upon the variance in performance associated with stimulus–response repetition trials. We believe this view would not account for the current data, for at least two reasons. First, using the same paradigm as the one used here, Gratton et al. (1992) found that when stimulus–response repetitions were separated from trials in which only the flanker information repeated, the modulation effect was still found. Second, arbitrary cues presented well before the imperative stimulus that predict flanker information but not the actual stimulus or the response also induced modulation of the noise-compatibility effect in a manner and to a degree similar to that obtained as a function of either probability or sequential (stimulus-response repetition) manipulations (see Gratton et al., 1992, Experiment 3). This cueing procedure arguably taps the same processes associated with strategic adjustments as the probability manipulation used here. Thus, higher-order processes engaged in the service of adjustments in control provide a better account for modulation of noise-compatibility effects than does simple response priming.

In summary, the findings reported here should significantly advance theorizing related to processing of error and conflict in the human cognitive system. Correct responses are associated with at least three distinct medial-frontal negativities in the ERP, including both stimulus-related (N2) and response-related components (CRN, N300), all of which respond differentially to different types of conflict during information processing. Future research should be directed at further specifying the functional relations among these components and their relation to the ERN and Pe, their neural generators, and their significance for controlling cognition and behavior. Finally, although the current data appear inconsistent with aspects of both error-detection and conflict-monitoring theories, we contend that these models need only to broaden their focus to consider how people use all relevant information in order to account for these findings.

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