

The Timing of Action-Monitoring Processes in the Anterior Cingulate Cortex

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Abstract

■ The anterior cingulate cortex (ACC) has been shown to respond to conflict between simultaneously active, incompatible response tendencies. This area is active during high-conflict correct trials and also when participants make errors. Here, we use the temporal resolution of high-density event-related potentials (ERPs) in combination with source localization to investigate the timing of ACC activity during conflict and error detection. We predicted that the same area of the ACC is active prior to high-conflict correct responses and following erroneous responses. Dipole modeling supported this prediction: The frontocentral N2, occurring prior to the response on correct conflict trials, and the ERN, occurring immediately following error responses, could both be modeled

as having a generator in the caudal ACC, suggesting the same process to underlie both peaks. Modeling further suggested that the rostral area of the ACC was also active following errors, but later in time, contributing to the error positivity (P_E), and peaking at 200–250 msec following the ERN peak. Despite the inherent limitations of source localization, these data may begin to shed light on the timing of action-monitoring processes. First, the time course of caudal ACC activity follows the time course as predicted by the conflict theory of this region. Second, caudal ACC activity might be temporally dissociated from rostral ACC activity during error trials, which possibly reflects a separate, affective component of the evaluative functions of the ACC. ■

INTRODUCTION

The concept of monitoring, and the distinction between attentional control and monitoring, has been highly influential in cognitive science, in both classical and recent literature (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Efklikes, Samara, & Petropoulou, 1999; Levelt, Roelofs, & Meyer, 1999; Koriat & Goldsmith, 1996; Ohlsson, 1996; Wegner, 1994; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Carver & Scheier, 1981; Miller, Galanter, & Pribram, 1960). Control refers to a top-down, limited-resource cognitive mechanism modulating information processing based on an attentional or instructional set or goal representation; monitoring refers to a cognitive mechanism that applies a simple algorithm to a limited domain of information to evaluate the quality of information processing and executive control and to alert the control mechanisms if information processing does not proceed adequately. Thus, monitoring and control are complimentary processes. Without a monitoring mechanism, theories about the nature and implementation of executive control are homuncular because control cannot exert control on itself (Botvinick et al., 2001). In recent years, the rise of cognitive neuroscience has generated a plethora

of research into the neural correlates of executive control; strangely, however, the complimentary and necessary concept of evaluative monitoring has received much less attention.

It has recently been hypothesized that (a) one such evaluative monitoring process is accomplished through the detection of processing conflicts between simultaneously active, task-relevant representations that are incompatible; (b) this conflict detection function is carried out by a distinct area of the brain, the anterior cingulate cortex (ACC), specifically, the caudal, “cognitive” part of the ACC; and (c) the input to this monitoring process consists mainly of representations active at response-related or later levels of information processing (van Veen, Cohen, Botvinick, Stenger, & Carter, 2001; Botvinick et al., 2001; Barch, Braver, Sabb, & Noll, 2000; Cohen, Botvinick, & Carter, 2000; MacDonald, Cohen, Stenger, & Carter, 2000; Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Carter et al., 1998, 2000). This hypothesis regarding ACC functioning in cognition is in contrast to the view that it implements a top-down control function, such as selection (Turken & Swick, 1999; Posner & Petersen, 1990) or response inhibition (Smith & Jonides, 1999; George et al., 1994). It bears more resemblance to (though it is distinct from) theories that assign other monitoring algorithms to this area, notably, the detection of overt errors based on a comparator process (Kiehl, Liddle, & Hopfinger, 2000;

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Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996; Scheffers & Coles, 2000; Gehring et al., 1993) or responsiveness to general difficulty (Paus, Koski, Caramanos, & Westbury, 1998). Although evidence has been provided against the views that ACC activity reflects a (controlled) selection or inhibition process (Carter et al., 2000; Casey et al., 2000; MacDonald et al., 2000; Botvinick et al., 1999), or a difficulty or error detection process (van Veen et al., 2001; MacDonald et al., 2000; Carter et al., 1998), the broad role of the ACC in higher cognitive functioning remains the subject of debate (Bush, Luu, & Posner, 2000).

The view that the ACC participates in conflict detection is partially based on data from studies using event-related potentials (ERPs). Immediately following errors, a large negative-going peak has been observed, with a frontocentral maximum (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Gehring et al., 1993), which has been dubbed as the error negativity (N_E) or error-related negativity (ERN). The ERN has repeatedly been modeled by a single dipole source, located in the vicinity of the ACC (Gehring, Himle, & Nisenson, 2000; Holroyd, Dien, & Coles, 1998; Dehaene, 1996; Dehaene, Posner, & Tucker, 1994). In support of these dipole models, studies using functional MRI have shown increased activation of the ACC during error trials relative to correct trials (Kiehl et al., 2000; Carter et al., 1998). Although, often, the ERN is considered to reflect the detection of overt errors based on a comparator process (Falkenstein et al., 2000; Gehring et al., 1993), we have argued that the ERN reflects the detection of conflict rather than of overt errors (Carter et al., 1998).

Errors in speeded response tasks are typically premature, impulsive responses executed while stimulus evaluation is still incomplete (Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988); while the error is being committed, stimulus evaluation continues, leading to activation of the correct response and a very fast correction of the erroneous response (Kopp, Rist, & Mattler, 1996; Rabbitt & Rodgers, 1977; Rabbitt & Vyas, 1981). Therefore, not much response conflict prior to an error is to be expected; rather, response conflict should be maximal immediately after an error is made and both responses are active—which is when the ERN occurs. This has been shown using a connectionist network (Botvinick et al., 2001). Evidence suggests that during correct trials high in response conflict, however, conflict occurs prior to the response rather than after: At first, the erroneous response is prepared, but the correct response manages to override the incorrect response tendency (Kopp et al., 1996; Gratton et al., 1988). The conflict theory thus predicts a temporal rather than a functional dissociation between ACC activity during error trials and correct conflict trials: In the former, we expect to see the ACC to be active following the response, and in the latter, we expect to see the ACC to be active prior to the response.

ERN research has provided evidence that the ACC is indeed activated immediately following an error response; however, there is no established evoked potential correlate of ACC activity prior to the response. A likely candidate for this is the frontocentral N2. This ERP component has a scalp topography similar to the ERN and appears to be increased in conditions in which response conflict is high (Liotti, Woldorff, Perez, & Mayberg, 2000; Wang, Kong, Tang, Zhuang, & Li, 2000; Smid, Jakob, & Heinze, 1999; Lange, Wijers, Mulder, & Mulder, 1998; Kopp et al., 1996; Kenemans, Kok, & Smulders, 1993; Wijers, Mulder, Okita, Mulder, & Scheffers, 1989). Like the ERN, the N2 has previously been hypothesized to have an ACC generator (Liotti et al., 2000; Lange et al., 1998). One of the goals of the present study was to test the hypothesis that the N2 might reflect conflict detection by the ACC.

To date, however, no direct comparison has been made between dipole models of the ERN and the N2 within the same study. Although our theory predicts that the same region of the ACC is involved in generation of both the ERN and the N2, recent fMRI evidence may possibly suggest otherwise. Menon, Adelman, White, Glover, and Reiss (2001) and Kiehl et al. (2000) used fMRI to study ACC activity in response to errors and correct conflict trials and reported that while the caudal ACC was activated during correct conflict trials, error trials additionally and uniquely engaged a more rostral area of the ACC. These authors have suggested that whereas the caudal activation of the ACC reflects conflict detection, the rostral activation reflects error detection. There are, therefore, two possibilities as to the N2 and ERN generators. First, it is possible that the caudal ACC generates the N2, reflecting conflict detection, whereas the ERN is generated by the rostral ACC, reflecting error detection. If this were the case, we would expect the ERN to have a more anterior generator than the N2. This would provide support for the comparator hypothesis of the ERN. Alternatively, the ERN and N2 might both be generated by the caudal ACC, reflecting conflict detection. In this case, modeling the generators of the two peaks should result in similar dipole models, located in the same region of the ACC. If this proves to be the case, it is possible that the rostral ACC activity during error trials does not contribute to the generation of the ERN per se, but might instead have a different time course, contributing to some other ERP components.

In the present task, we recorded high-density ERPs from participants while they were performing a version of the Eriksen flanker task (Eriksen & Eriksen, 1974; Eriksen & Schultz, 1979). In this task, the participant has to respond to a target presented centrally on the screen, while trying to ignore simultaneously presented flanker stimuli. In our version of the flanker task, flankers could either be the same as the target (congruent, CO); mapped to the opposite response

(response-incongruent, RI); or different from the target but still mapped to the same response (stimulus-incongruent, SI). Since participants respond slower to SI stimuli than to CO stimuli, and again slower to RI stimuli (Eriksen & Schultz, 1979), both the SI and RI conditions involve conflict relative to the CO condition. Since in the SI condition, the irrelevant information is mapped onto the same response as in the CO condition, this condition is often thought to involve competition between target representations, thus reflecting conflict at an earlier or central processing stage, possibly involved in stimulus evaluation or target detection (Zhang, Zhang, & Kornblum, 1999; Kornblum, Hasbroucq, & Osman, 1990). In addition, since the flankers and target of RI stimuli are mapped onto incompatible responses, this condition involves competition between task-relevant responses, and, thus, this condition induces conflict at a later or response-related stage. Using event-related fMRI, we have shown that the ACC is only activated to RI stimuli, even when controlling for reaction time (RT) differences, which suggests that the ACC is selectively responsive to conflict occurring at later or response-related levels of processing (van Veen et al., 2001). Thus, if the frontocentral N2 is indeed generated by the ACC, it should follow the pattern of activity seen in the fMRI study and should only be enhanced to RI stimuli.

In summary, we sought to test the following hypotheses. First, we predicted that a frontocentral N2 should be elicited during correct incongruent trials, an ERN should be elicited following errors and we hypothesize that both are generated by the ACC. Thus, we hypothesize that the timing of ACC activity differs between error trials and correct incongruent trials: ACC activity immediately follows the response during error trials and is reflected in the ERN; ACC activity precedes the response during correct incongruent trials and is reflected in the N2. We therefore predict that source localization should indicate an ACC generator for both peaks. Second, we predict that, if the N2 is indeed generated

by the ACC, it should only be enhanced to RI trials and be similar in amplitude to CO and SI trials. Third, if the ERN reflects the operation of an error detector that is more rostral than the conflict detector (Menon et al., 2001; Kiehl et al., 2000), source localization should indicate a more anterior generator for the ERN and a relatively more posterior one for the N2. In contrast, the conflict theory predicts that the frontocentral N2 and the ERN will have similar scalp distributions and a generator in the same region of the ACC.

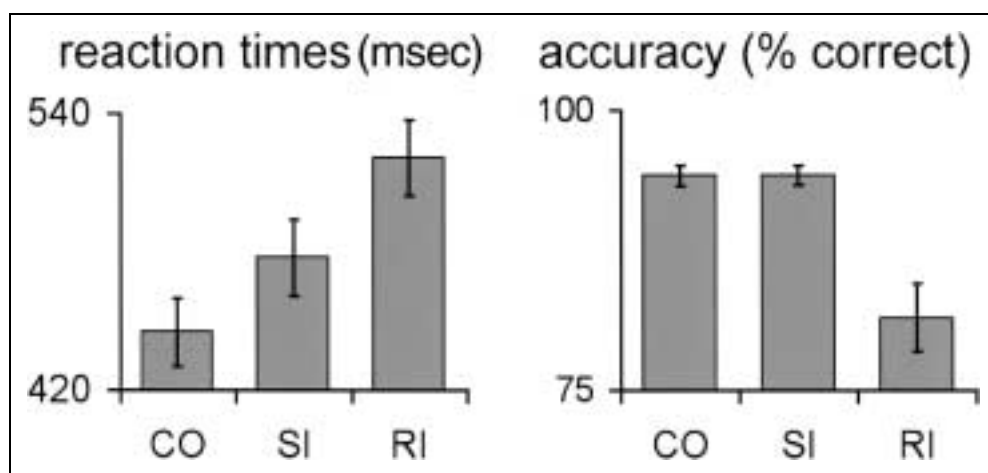
RESULTS

Behavioral Data: RTs and Accuracy

Mean correct RTs were 445 msec for CO stimuli ($SD = 51$), 478 msec for SI stimuli ($SD = 58$), and 521 msec for RI stimuli ($SD = 57$). A repeated-measures ANOVA indicated that the differences between RTs were highly significant [$F(2,11) = 84.87, p < .001$]. Subsequent paired t tests were carried out between CO and SI ($t = 8.64, p < .001$), between CO and RI ($t = 12.56, p < .001$), and between RI and SI ($t = 6.04, p < .001$), indicating that RTs increased with the level of conflict. Thus, RTs to SI stimuli were longer than RTs to CO stimuli; RTs to RI stimuli were longer than to both SI and CO stimuli (see Figure 1).

Accuracy per trial type was determined in percentage correct. CO trials were responded to with 94.2% accuracy ($SD = 3.1$), SI stimuli with 94.3% accuracy ($SD = 3.0$), and RI stimuli with 81.5% accuracy ($SD = 10.6$). Because of heteroscedasticity, nonparametric tests were used; accuracy differed significantly between the three groups of trials [Friedman's $\chi^2 = 18.17, p < .001$]. Specifically, Wilcoxon signed ranks revealed significant differences between SI and RI ($z = 3.06, p < .005$) and CO and RI ($z = 3.06, p < .005$); in contrast, a paired t test indicated that the difference between CO and SI stimuli was not significant ($t = .16, p = .878$). Thus, accuracy did not differ significantly between CO and SI trials, but

Figure 1. RT (left) and accuracy (right) data for each of the three conditions. RT increases with stimulus conflict, and again with response conflict. Accuracy, however, is only modulated by response conflict. Error bars represent $\pm 1 SEM$.



participants responded significantly less accurately to RI trials (see Figure 1).

ERPs: Frontocentral N2 and ERN

Inspection of the grand averaged waveforms and their topographies showed, first, in the stimulus-locked waveform, the frontocentral N2, with a latency of 340–380 msec after target onset, to be enhanced in response to RI stimuli. Second, in the response-locked waveform, a clearly defined ERN followed errors, occurring 40–80 msec after button press (see Figure 2). Inspection of the scalp topographies of the difference waves showed both peak differences to be maximal at Cz (channel 129); thus, values obtained from Cz were used for statistical analyses of both the N2 and the ERN. The amplitude of the N2 was defined as the amplitude of the largest negative peak in the 340–380 msec interval after target onset; the amplitude of the ERN was defined as the amplitude of the largest negative peak in the 40–80 msec interval after button press.

For stimulus-locked waveforms, a repeated-measures ANOVA indicated that the amplitude of the frontocentral N2 differed between the three conditions [$F(2,11) = 11.06, p < .001$]. Subsequent paired t tests showed significant differences between CO and RI ($t = 4.41, p < .001$) and between RI and SI ($t = 3.90, p < .005$). No significant difference emerged between CO and SI ($t = 1.30, ns$). Thus, the N2 was largest in response to RI stimuli but did not differ significantly between CO and SI stimuli. For response-locked waveforms, a paired t test indicated the difference between correct and error waveforms to be significant ($t = 7.54, p < .001$), indicating the ERN to be significantly more negative than the waveform following correct responses.

To test whether the frontocentral N2 and the ERN had the same scalp distribution, a correlation was conducted between the values for each channel at the peak of the difference wave (ERN, 52 msec; N2, 356 msec). Regress-

ing the value for each channel of the N2 on the value of the ERN resulted in a regression line with the formula $(N2) = -0.00095 + 0.101(ERN)$. This was highly significant ($r = .89, p < .001$). Apart from a significant positive correlation, if the ERN and N2 have the same scalp distribution but a different amplitude, we would expect the regression line to go through the origin. The confidence interval for the constant was $[-0.20, 0.19]$; the constant, thus, was not different from 0 ($t = -.96, p = .92$). We can thus conclude that, except for the difference in amplitude, the scalp topographies of the ERN and the N2 difference waves are the same (see Figure 3, left).

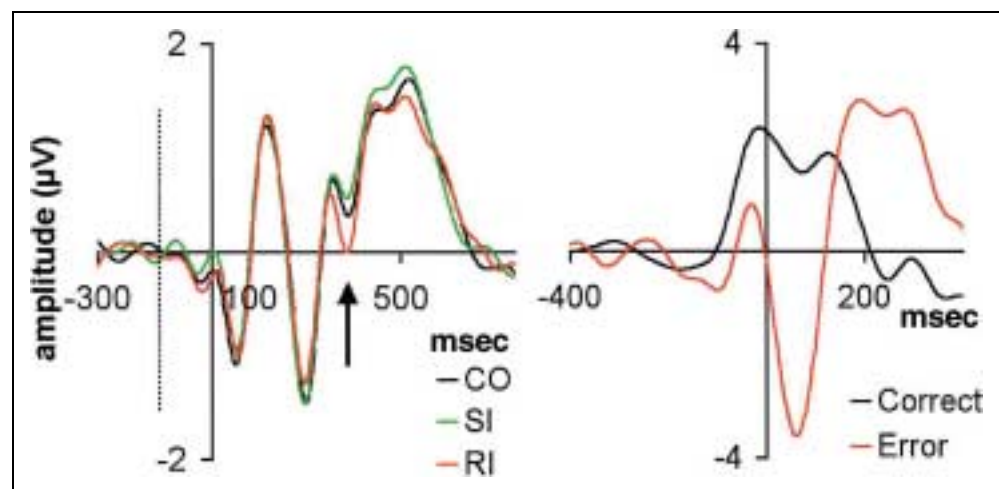
Dipole Source Localization: Frontocentral N2 and ERN

Source localization, using BESA 2000 (MEGIS Software, Munich, Germany), was first performed on the peak maximum of the difference waveforms between errors and correct responses (latency 52 msec following button press) and between RI stimuli and SI stimuli (latency 356 msec following target onset), using a three-shell spherical head model approximation (Scherg, 1990; Ary, Klein, & Fender, 1981). A single dipole model located in the vicinity of the ACC was able to account for most of the variance in the observed data for both peaks (ERN location: $x = -0.03, y = 0.20; z = 0.50$; residual variance (RV): 3.71%. N2 location: $x = 0.03; y = 0.12; z = 0.49$; RV: 8.34%).¹ Both solutions were obtained without introducing constraints and were consistent across starting positions. Superimposing the dipole locations on a standard reference brain showed that both dipoles were indeed located in the same region of the ACC (see Figure 3, middle and right).

ERPs: Error Positivity (P_E)

The finding that the frontocentral N2 and the ERN appear to be both generated by the same area of the

Figure 2. ERPs at Cz (Channel 129). Left: stimulus-locked waveforms for correct trials, separated by stimulus condition. Target onset is at 0 msec; flanker onset, occurring 100 msec prior to target onset, is indicated by a vertical dashed line. The N2 (indicated by an arrow) is visible at around 350 msec. Right: response-locked waveforms separated by correct and error trials. Response occurs at 0 msec. A clear, large ERN can be seen following error responses, peaking at around 50 msec, followed by a positive component (P_E).



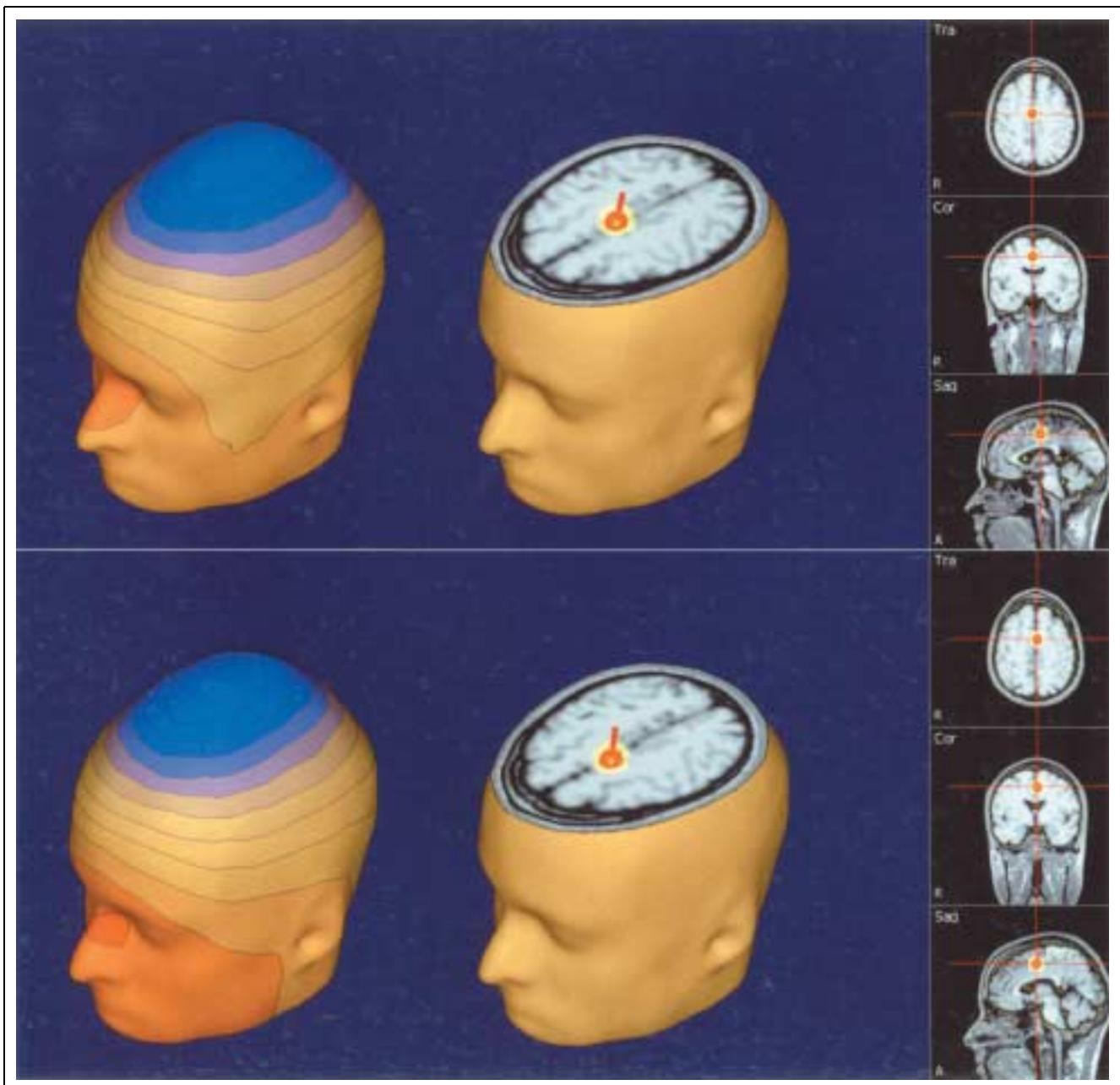


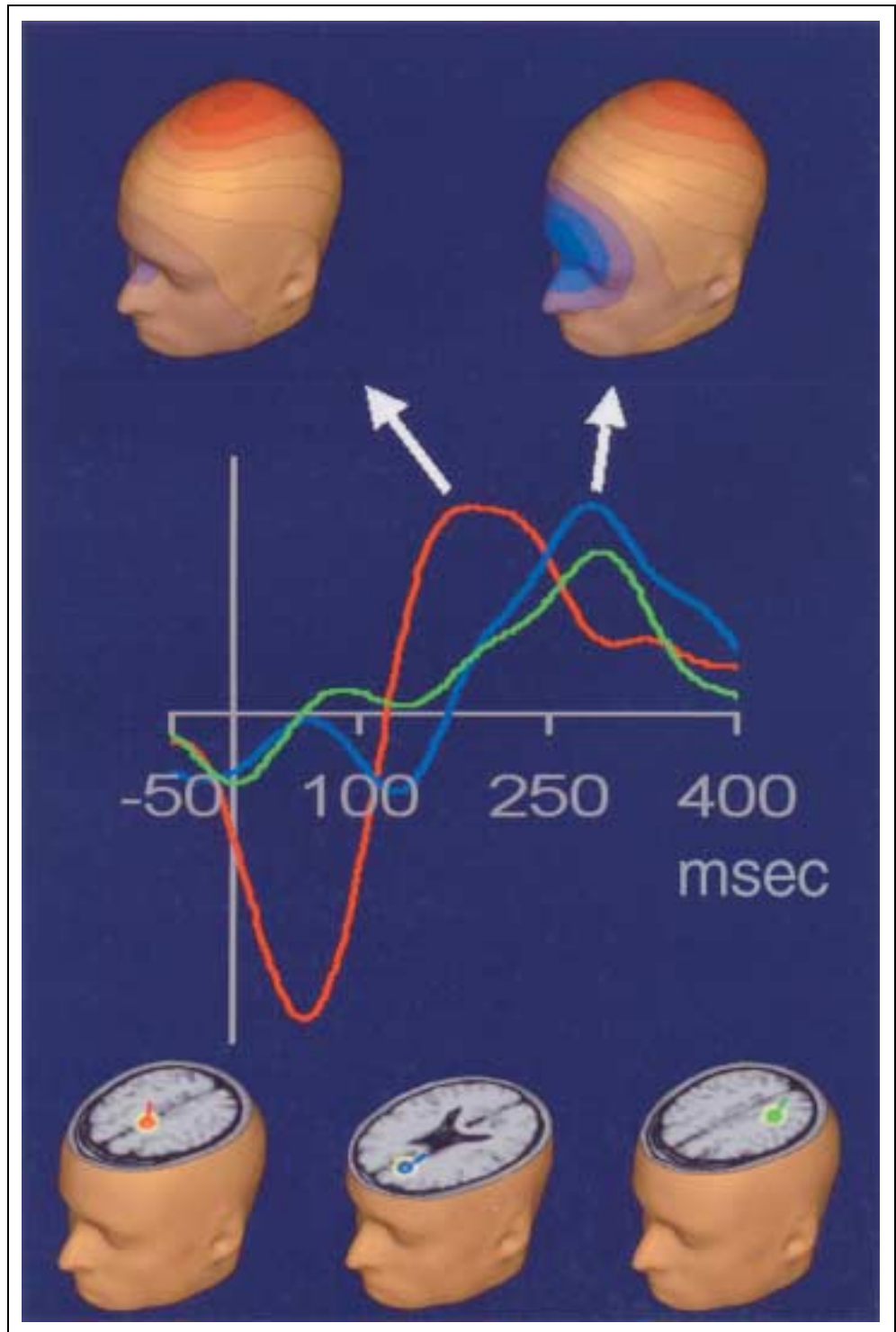
Figure 3. Voltage scalp distribution maps (large, left) and dipole source models (large, right) for the N2 (top) and ERN (bottom), superimposed on a standard realistic, MR-based head model. For each waveform peak, the scalp distribution explained by the dipole model is shown in the small top middle, the residual scalp distribution is shown in the small bottom middle head (blue = more negative; red = more positive). Transversal, coronal, and sagittal views of dipole superimposed on MR-based realistic head model are shown on the right (small). As can be clearly seen from these figures, both dipoles are located in the same region of the ACC.

ACC supports our hypotheses regarding the timing of activation and function of this area. However, the results seem at odds with recent fMRI studies that have implied a role for the rostral ACC in error processing (Menon et al., 2001; Kiehl et al., 2000). It is therefore possible that the rostral ACC contributes to a different ERP component than the ERN. To investigate this possibility, the entire interval following error responses was analyzed.

Following the ERN, a clear positivity was observed, which has been referred to as the P_E (Falkenstein et al.,

2000). Inspection of the difference waveforms showed that this component appeared to have two subcomponents: One early subcomponent peaked at around 180 msec following the response and was maximal at Cz; one later subcomponent peaked at around 300 msec and peaked at Pz (Channel 68). To test whether these are different, the average values of the error and correct waveforms for the intervals from 160 to 200 msec, and from 280 to 320 msec following the response at Fz, Cz, and Pz, were entered into a three-way repeated-measures

Figure 4. Source time course and dipole locations for the -50 to 400 msec interval following errors. The caudal ACC dipole (red; bottom, left) explains the ERN and early P_E ; the rostral ACC dipole (blue; bottom, center) and the left superior parietal cortex dipole (green; bottom, right) explain the late P_E . Top, left: scalp distribution of early P_E (188 msec). Top, right: scalp distribution of late P_E (298 msec). Blue = more negative; red = more positive.



ANOVA, with accuracy, interval, and channel as factors. This was significant [$F(2,22) = 3.34, p < .05$], indicating that the peak differences are different for the electrodes between these two time intervals. Paired t tests carried out separately at both time intervals, for the electrode at which the signal peaks, indicated, too, that the P_E was significantly more positive at both intervals, at both peaks: The early peak was significant at Cz

($t = 2.92, p < .05$); the late peak was significant at Pz ($t = 5.47, p < .001$).

Dipole Source Localization: P_E

Next, source analysis was performed on the entire error-correct difference wave, from 50 msec prior to response to 400 msec following response, with the constraint that

the location and orientation of the ERN dipole were held constant (see Figure 4). A physiologically plausible model ($RV = 5.05\%$, best = 2.90%) was obtained with a three-dipole model (see Figure 4). In addition to the ERN dipole, this model included one dipole located in the vicinity of the left superior parietal cortex (location: $x = -0.30, y = -0.22, z = 0.51$) and one dipole located in the rostral ACC (location: $x = -0.12, y = 0.59, z = 0.22$). As can be seen in Figure 4, the moment of the dipole located in the caudal ACC corresponds to the ERN, followed by the “early” P_E , which was maximal at Cz; in contrast, the dipoles in the rostral ACC and superior parietal cortex explain the “late” part of the P_E , which was maximal at Pz.

DISCUSSION

Analysis of the behavioral data confirmed our hypotheses regarding conflicts between different kinds of representations. First, RTs were longer to SI than to CO stimuli, in the absence of an accuracy difference. This indicated that the SI stimuli generated conflict at the stimulus level, while these two conditions generated similar amounts of response conflict. The longer RTs and reduced accuracy to the RI stimuli indicated increased conflict at the response level. These findings replicate earlier findings using this task (van Veen et al., 2001; Eriksen & Eriksen, 1974; Eriksen & Schultz, 1979). Thus, we have evidence of conflict occurring at both stimulus-related and response-related processing levels (Zhang et al., 1999; Kornblum et al., 1990).

Analyses of the stimulus-locked ERPs confirmed our hypothesis regarding the frontocentral N2: It was enhanced to RI stimuli, replicating previous findings using the flanker task (Kopp et al., 1996). Moreover, it was enhanced *only* to RI stimuli. Dipole source modeling supported our hypothesis regarding the location of the N2 and ERN generators: Both peaks could be well explained by a dipole located in the ACC, replicating earlier dipole models (Gehring et al., 2000; Holroyd et al., 1998; Lange et al., 1998; Dehaene et al., 1994; Dehaene, 1996). Importantly, we found that the ERN and N2 dipoles were located in the same region of the ACC. This reinforces the notion that they reflect the same process (Kopp et al., 1996). Notably, this finding has now been replicated by two other groups (Liotti, Kothmann, Perez, & Woldorff, 2001; Yeung, Gelfand, Scanlon, & Cohen, 2001). Functional MRI evidence had already shown that the caudal ACC is activated during both errors and correct conflict trials (Kiehl et al., 2000; Carter et al., 1998), suggesting that this area of the brain is involved in conflict detection. Thus, because the present dipole models imply the involvement of an area of the brain that has already been shown to be activated during these trial types, we consider these models to be plausible. Importantly, the plausibility of the models is

further reinforced by the fact that the time course of activation (preresponse during correct conflict, postresponse during error trials) exactly matches that as predicted by the conflict theory, as explained in the Introduction. We therefore suggest that conflict detection by the (caudal) ACC is the process underlying both peaks. The notion that the ERN and the N2 reflect the operation of the same underlying mechanism argues against the hypothesis that the ERN reflects error detection by a comparator process (Scheffers et al., 1996; Scheffers & Coles, 2000; Gehring et al., 1993), since the N2 is elicited in correct trials. We hope that the hypothesis that the frontocentral N2 and ERN reflect conflict detection will prove to be of great value in interpreting results from studies using ERPs.

The fact that the N2 was only enhanced to RI stimuli is also consistent with our prediction based on our earlier fMRI experiment, which used almost the same task (van Veen et al., 2001). In that experiment, we have shown that the ACC is activated to RI but not to SI stimuli. This is another reason to consider the ACC dipole model of the N2 generator to be a plausible model. We thus hypothesize that the ACC signal, which we measured in the fMRI experiment, is, in fact, also reflected in the N2 as measured by scalp electrodes. These convergent findings from the current ERP and our previous fMRI studies suggest that the ACC detects conflict occurring at the response level of processing but not at the stimulus level. This notion is consistent with the rich interconnectivity of the ACC with the motor system (Picard & Strick, 1996) and unites the view that the ACC has an attentional or motivational role in cognition with the view that the ACC is part of the motor system.

The ERN was followed by the P_E . Post hoc modeling of the generators of the P_E suggested a contribution of the rostral ACC. This model may shed light on recent data by Menon et al. (2001) and Kiehl et al. (2000), who found the rostral ACC to be engaged during error trials, but not during correct conflict trials. The distant rostral and caudal ACC activations are convolved together in fMRI due to the more limited temporal resolution of this methodology; so they appear to reflect the same functional component. Our model, on the contrary, can explain these findings by suggesting that the time course of activation of the rostral ACC following error responses may be rather different from that of the caudal ACC. Whereas the error-related activity of the caudal ACC might be reflected in the ERN and early P_E , activity of the rostral ACC might be reflected in late P_E . Considering that the rostral ACC is often thought of as associated with affective processing (Bush et al., 2000; Devinsky, Morrell, & Vogt, 1995), our model appears consistent with the hypothesis that the P_E might reflect a subjective emotional error assessment process, as suggested by Falkenstein et al. (2000). It is also possible that the P_E might reflect the operation of some other error monitoring (e.g., Ohlsson, 1996) or more complex

performance monitoring process (e.g., Wegner, 1994). The model suggested an additional contribution of a left parietal source to the late P_E (see Figure 4), although it is unclear whether this source represents activation of the medial or lateral parietal cortex. The medial parietal cortex (precuneus) has previously been implied in error processing (Menon et al., 2001). These activations might reflect the attentional resource allocation that follows response conflict or error detection. Future research is needed to evaluate the validity of this dipole model and to better characterize the significance and function of the P_E and the rostral ACC.

It should be emphasized that the brain areas implied by source localization are only tentative—modeling the scalp potential topographies by assuming a limited number of equivalent dipole sources is an inverse problem for which there is no unique solution (Peters & de Munck, 1990; Scherg, 1990). Thus, the current results only provide a model, rather than empirical data, regarding the involvement and timing of activation of brain areas in response to conflict and errors. Nevertheless, we believe the present models to be plausible and parsimonious because they converge with data obtained from fMRI research. These findings demonstrate the potential value of the temporal resolution provided by ERPs (and ERFs) in cognitive neuroscience by suggesting a separation, both within and between brain regions, between distinct processes that contribute to performance monitoring. This would not have been possible when solely relying on the spatial information provided by fMRI. The use of temporal information as provided by ERPs has allowed us to test more specific predictions made by the conflict monitoring theory. However, because of the limited spatial resolution of ERPs, future studies will be needed to further evaluate the relationship between the frontocentral N2, the ERN and the caudal ACC, and the relative contribution of the rostral ACC to error processing. A useful approach that might achieve this is to carefully crossregister participants using both ERP and fMRI methodologies and let the dipolar sources be constrained by the fMRI results.

METHODS

Participants

Twelve healthy young people participated in the experiment (six women, six men; mean age 23.4 years, $SD = 2.8$). Participants received a monetary payment for participating. Prior to the test, participants provided written informed consent in accordance with the Institutional Review Board of the University of Pittsburgh.

Procedure

Stimuli were presented using E-Prime (Psychological Software Tools, Pittsburgh, PA). Participants were in-

structed to make a left-hand button-press response if the central letter was an S or an X, and a right-hand button-press response if the central letter was an H or P. Participants were instructed to be fast but accurate. On each trial, a line of five letters was presented, the central one of which was the target, and the remaining letters the flankers. In CO trials, the flankers were identical to the target (e.g., SSSSS or HHHHH). In SI trials, the flankers were different but mapped onto the same response hand (e.g., SSXSS or HHPHH). In RI stimuli, the flankers were mapped onto the opposite response hand than the target stimulus (e.g., SSPSS or HHXHH). Trials were presented in random order: 50% of trials were CO stimuli, and the SI and RI conditions each accounted for 25% of trials. Participants received four blocks of 384 trials each.

Each trial started with the presentation of a central fixation cross “+”. The flanker stimuli appeared 100 msec prior to the target stimulus, the target stimulus appearing in the same place as the fixation cross. Target and flankers disappeared from screen simultaneously when a response was made and were immediately replaced by the fixation cross, marking the intertrial interval. The intertrial interval was randomized between 500 and 1500 msec. Each block of trials started with a reminder to respond fast, followed by a fixation cross, which lasted for 3000 msec before the start of the first trial.

Electroencephalogram (EEG) Recording

EEG was recorded using a 128-channel Geodesic Sensor Net (Tucker, 1993) at 500 Hz and was recorded and analyzed using EGI software (EGI, Eugene, OR). Impedance of all channels was kept below 40 k Ω . All channels were referenced to Cz (Channel 129) during recording. Stimulus-locked data were segmented into epochs of 300 msec before to 800 msec after target onset; response-locked data were segmented into epochs of 400 msec before to 400 msec after button press. Editing of the EEG for eye blink and eye movement artifacts (threshold 70 μ V), signals exceeding 200 μ V or fast transients exceeding 100 μ V, was performed by a computer algorithm during averaging. Also during averaging, data were rereferenced against the average reference (Tucker, Liotti, Potts, Russell, & Posner, 1993; Bertrand, Perrin, & Pernier, 1985). For statistical analyses, the average of each participant was filtered with an FIR bandpass filter with a high cutoff frequency of 30 Hz and a low cutoff frequency of 1 Hz prior to grand averaging; for the source localization analyses and for display purposes, data were filtered with a 1–12 Hz FIR bandpass filter. For both stimulus-locked and response-locked waveforms, a baseline calculated over the first 200 msec subtracted from each channel. For display of the scalp topographies and for source localization, difference waveforms were obtained by subtraction.

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Note

1. Although the RV of a one-dipole model for the N2 was sufficiently small, inspection of the N2 scalp topography indicated two additional posterior positivities, suggesting the presence of two additional, symmetrical dipoles in the temporal occipital cortex. Post hoc adding a symmetrical dipole pair to the one-dipole model resulted in a model with an ACC dipole ($x = 0.05, y = 0.22, z = 0.56$) and two occipital temporal sources ($x = \pm 0.54, y = -0.70, z = -0.05$) with a RV of 6.31%. Thus, the location of the ACC dipole of this three-dipole model of the N2 was still in the same region of the ACC as the ERN dipole, thus, our conclusions would not be different had we rejected the one-dipole model and accepted the three-dipole model.

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