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Electrophysiological Analysis of Error Monitoring in Schizophrenia

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In this study, the authors sought to determine whether abnormalities exhibited by schizophrenia patients in event-related potentials associated with self-monitoring—the error-related negativity (ERN) and the correct response negativity (CRN)—persist under conditions that maximize ERN amplitude and to examine relationships between the ERN and behavior in schizophrenia. Participants performed a flanker task under 2 contingencies: one encouraging accuracy and another emphasizing speed. Compared with healthy participants, in schizophrenia patients the ERN was reduced in the accuracy condition, and the CRN was enhanced in the speed condition. The amplitude of a later ERP component, the error positivity, did not differ between groups in either task condition. Reduced self-correction and increased accuracy following errors were associated with larger ERNs in both groups. Thus, ERN generation appears to be abnormal in schizophrenia patients even under conditions demonstrated to maximize ERN amplitude; however, functional characteristics of the ERN appear to be intact.

Keywords: self-monitoring, schizophrenia, error-related negativity

Accurate and rapid online monitoring of one's thoughts and actions is essential for the completion of goal-directed behavior and for maintaining the internal organization of intentions, information, and reasoning. Investigators have hypothesized that failure of this internal monitoring process may contribute to the clinical symptoms experienced by schizophrenia patients. It has been suggested, for example, that misattribution of internally generated thoughts may result in auditory hallucinations (Frith, 1987). Similarly, failure to detect one's intention to execute a behavior may lead to the experience that these behaviors are under external control (e.g., Frith & Done, 1989), and faulty self-monitoring of speech may contribute to formal thought disorder (McGrath, 1991). Detection of behavioral errors is another important form of internal monitoring, allowing an individual to adjust his or her behavior in the absence of external feedback. In light of the possible disruption of self-monitoring in schizophrenia, examination of error detection provides an empirical basis for testing hypotheses regarding these internal processes.

Accordingly, researchers have examined the ability of schizophrenia patients to monitor their own behavior. Malenka, Angel, Hampton, and Berger (1982) reported that schizophrenia inpatients were less likely than nonpsychiatric participants to correct incorrect responses and more likely to reverse correct moves on a motor tracking task. In addition, medicated (Turken, Vuilleumier, Mathalon, Swick, & Ford, 2003) and unmedicated (Frith & Done, 1989) schizophrenia patients showed reduced rates of spontaneous error correction compared with nonpatients when visual feedback concerning their performance was not available. Schizophrenia patients also appear to be impaired in discriminating their own movements from those made by others (Daprati et al., 1997). In other studies, however, schizophrenia patients have not exhibited impairment in self-monitoring (e.g., Fourneret, Franck, Slachevsky, & Jeannerod, 2001; Kopp & Rist, 1994). Thus, there is intriguing but inconsistent behavioral evidence of impaired self-monitoring in schizophrenia.

The present study relied on a component of the event-related brain potential (ERP), the error-related negativity (ERN; Gehring, Goss, Coles, Meyer, & Donchin, 1993), which is theorized to be related to self-monitoring. The ERN peaks approximately 60–100 ms after the execution of incorrect responses and is a robust signal that has been elicited in a variety of paradigms (e.g., Bernstein, Scheffers, & Coles, 1995; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001; Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996). Efforts to localize the neural generators of the ERN using electroencephalography (EEG; Dehaene, Posner, & Tucker, 1994; Holroyd, Dien, & Coles, 1998; Luu, Flaisch, & Tucker, 2000) and magnetoencephalography (Miltner et al., 1997) have converged on the anterior cingulate cortex (ACC). Functional

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MRI studies provide further evidence of the involvement of the ACC in self-monitoring (e.g., Carter et al., 1998; Holroyd et al., 2004; MacDonald, Cohen, Stenger, & Carter, 2000). This region of the brain is activated during tasks that require selective attention, working memory, language generation, and controlled information processing (Cabeza & Nyberg, 1997). It also has been suggested that the ACC is involved in the executive control of cognition (D'Esposito et al., 1995; Posner & Dehaene, 1994). Of importance, there is evidence suggesting that the ACC may be compromised in schizophrenia (e.g., Benes, Majocha, Bird, & Marotta, 1987; Benes, McSparren, Bird, SanGiovanni, & Vincent, 1991; Gabriel et al., 1997).

Several research groups have observed diminished ERN amplitude in patients with schizophrenia. Kopp and Rist (1999) reported that paranoid patients exhibited reduced ERNs compared with nonparanoid schizophrenia patients and healthy comparison participants during performance of a flanker task. Mathalon et al. (2002) examined response-related ERPs during a picture–word matching task and similarly found that schizophrenia patients exhibited reductions in ERN amplitude, with paranoid patients showing smaller ERNs than nonparanoid patients. In addition, ERN-like activity following correct responses, the correct response negativity (CRN), was larger in patients than in healthy comparison participants. Alain and colleagues (Alain, McNeely, He, Christensen, & West, 2002) reported a similar pattern of a diminished ERN and an enhanced CRN among schizophrenia patients during performance of a Stroop task. Using a go/no-go paradigm, Bates and colleagues (Bates, Kiehl, Laurens, & Liddle, 2002) also observed ERN reductions in schizophrenia patients. In contrast to Mathalon et al., patients did not show enhancement of the CRN, possibly because patients were less confident in the accuracy of their responses while associating words with pictures than while discriminating letters (Bates et al., 2002). Recently, Bates, Liddle, Kiehl, and Ngan (2004) reported that although the ERN increased in schizophrenia patients tested following 6 weeks of antipsychotic treatment, patients' ERN was diminished relative to that of healthy comparison participants regardless of medication status. In contrast to Bates et al. (2002), Bates et al. (2004) observed a CRN in patients and healthy participants, although the magnitude of the activity was not substantial. Taken together, reductions in ERN activity have been observed reliably in schizophrenia patients across a variety of paradigms, whereas enhanced CRN amplitude was found in only a subset of studies.

In considering its functional significance, the ERN was initially theorized to reflect the activity of an error-detection system, with heightened responses indicative of a mismatch between the intended and actual response (e.g., Bernstein et al., 1995; Coles, Scheffers, & Holroyd, 2001; Gehring et al., 1993). An alternate theory proposes that the ERN arises from the presence of response conflict rather than the detection of an error (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Carter et al., 1998). More recently, Yeung, Botvinick, and Cohen (2004) modeled various conflict and response parameters and demonstrated that the ERN arises during continued processing of the stimulus and activation of the correct response even following the commission of errors. They further showed that the appearance of an ERN on correct trials (e.g., Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Luu, Flaisch, & Tucker, 2000) may reflect conflict that is resolved prior to the execution of a correct response and is better characterized as an N2 (van Veen & Carter, 2002a). They proposed that conflict monitoring and error detection are not mutually exclusive but that conflict monitoring serves as a computationally simple method for detecting errors. This theory, however, does not account for the presence of ERN-like activity following correct responses appearing in response-locked averages (Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). It is possible that such activity reflects error processing due to the presence of some aspect(s) of erroneous responding even on correct responses, such as a slow reaction time (RT) during a task with an RT cutoff (Coles et al., 2001). Thus, it appears that the ERN may reflect a comparison of intended and actual responses, simultaneous activation of correct and incorrect responses, processing of a response in which one aspect is erroneous, or a combination of these factors.

Both error monitoring and response conflict detection can be expected to be affected by task context, which is governed by factors that include reward and penalty contingencies, instructions, difficulty, and recent and remote task-related experience (Cohen & Servan-Schreiber, 1992). These factors determine, at least in part, the significance of errors and the strength of activation (and potential coactivation) of responses. Among nonpsychiatric participants, for example, ERN amplitude appears to be highly sensitive to differences in task context and is largest when participants are instructed to focus on responding accurately, smaller when accuracy and speed of responding are emphasized equally, and smallest when participants are instructed to respond quickly (Gehring et al., 1993). Similarly, ERN is reduced under high compared with moderate time pressure (Falkenstein et al., 2000). These findings are consistent with recent evidence that the ERN is also modulated by affective or motivational factors (Luu, Collins, & Tucker, 2000) and demonstrate the critical importance of task instructions in studies of response-related ERPs.

One aim of the present study was to investigate the scope of error monitoring difficulties in schizophrenia patients by using a task devised to optimize ERN amplitude and by varying the motivational significance of errors. Specifically, performance demands were introduced to examine whether ERN activity is intact when schizophrenia patients are provided with a task context that enhances response monitoring in healthy individuals. In prior studies, participants were instructed either to respond quickly at the expense of accuracy (Kopp & Rist, 1999) or to respond quickly but without sacrificing accuracy (Alain et al., 2002; Bates et al., 2002, 2004; Mathalon et al., 2002). A key question is whether ERN activity in schizophrenia patients can be optimized when accuracy of performance is emphasized. It was hypothesized that compared with healthy participants, schizophrenia patients would exhibit a diminished ERN and an enhanced CRN across task conditions. Given prior reports of disruption in the representation and maintenance of task context in schizophrenia patients (e.g., Barch, Carter, MacDonald, Braver, & Cohen, 2003; Cohen, Barch, Carter, & Servan-Schreiber, 1999; MacDonald & Carter, 2003), it also was expected that any impact of instructional context on the ERN would be reduced in schizophrenia patients compared with healthy comparison participants. Among healthy participants, the ERN was predicted to be larger when task instructions and contingencies favored accurate responding than when they favored fast responding. In contrast, it was anticipated that schizophrenia patients would have difficulty sustaining the task context and would have a weakened representation of task-appropriate re-

sponses, such that the ERN in these participants would not differ between instruction conditions.

To examine whether abnormalities observed in schizophrenia patients are specific to the ERN, we compared the error positivity (Pe) between the two groups. The Pe is a positively deflected ERP that appears approximately 160–500 ms after the execution of a response and is more prominent following errors than correct responses (Falkenstein, Hohnsbein, & Hoormann, 1991; Falkenstein et al., 2000). Dipole modeling of the Pe suggests that its neural source lies in the rostral ACC (van Veen & Carter, 2002b), consistent with the theory that it is related to subjective assessment of errors (Bush, Luu, & Posner, 2000; Falkenstein et al., 2000). In prior studies that did not emphasize accuracy of performance, schizophrenia patients have not shown differences from healthy comparison participants in Pe amplitude (Alain et al., 2002; Bates et al., 2004; Mathalon et al., 2002). Thus, we hypothesized that group differences would be limited to the ERN and not extend to the Pe.

Another aim of this research was to evaluate the relationship between the ERN and response-related behaviors such as error correction and posterror slowing in schizophrenia patients. Increased error correction and slowing of RTs on trials following errors have been associated with a larger ERN in some studies (Gehring et al., 1993; Nieuwenhuis et al., 2001; Scheffers & Coles, 2000) but not in others (Gehring & Fencsik, 2001; Hajcak, McDonald, & Simons, 2003). Investigations conducted with schizophrenia patients (Alain et al., 2002; Mathalon et al., 2002) also have failed to find a relationship between the ERN and posterror slowing. It is possible that the impact of differences in experimental tasks, instructions to participants, analytic methods, and statistical power may be obscuring what might be only moderate relationships between the ERN and response behaviors. Because the methods used in the present study were most similar to those of Gehring et al. (1993), similar ERN-behavior relationships were predicted. Specifically, it was expected that in healthy participants, larger ERNs would be associated with greater posterror slowing, improved accuracy on trials following errors, and increased probability of error correction. Given evidence for normal posterror slowing (Alain et al., 2002; Laurens, Ngan, Bates, Kiehl, & Liddle, 2003; Mathalon et al., 2002) and self-correction (Kopp & Rist, 1994) in schizophrenia, it was hypothesized that the relationship between the ERN and response-related behaviors would not differ between patients and healthy comparison participants.

The association between ERP amplitude and response-related behaviors also was examined for the Pe. There is some suggestion that Pe amplitude does not differ between corrected and uncorrected errors (Falkenstein, Hohnsbein, & Hoormann, 1996). In contrast, Pe amplitude and posterror slowing have been found to be greater for perceived errors compared with those that are unperceived (Nieuwenhuis et al., 2001). Older participants, however, exhibit a reduced Pe but increased posterror slowing compared with younger participants (Falkenstein et al., 2000), suggesting a possible uncoupling as a function of aging. To our knowledge, the present study is the first to examine the relationship between Pe and behavior in schizophrenia. On the basis of similarities in experimental tasks between the present study and that of Falkenstein et al. (2000), we hypothesized that Pe amplitude would not be related to error-related behaviors in healthy participants. A normal pattern of relationships also was anticipated among schizophrenia patients, assuming that the disjunction between Pe and posterror slowing is restricted to older persons.

Method

Participants

A total of 19 schizophrenia patients and 11 healthy comparison participants participated in the study. Data from 3 patients were excluded because of equipment problems ($n = 2$) or excessive artifact from movement ($n = 1$), yielding a final sample of 16 patients. The groups did not differ in age, $F(1, 25) = 0.01$, $p = .92$; years of education, $F(1, 25) = 3.74$, $p = .06$; years of parental education, $F(1, 25) = 0.00$, $p = .99$; gender, $\chi^2(1, N = 27) = .08$, $p = .78$, or ethnicity, $\chi^2(5, N = 27) = 3.24$, $p = .66$. Demographic characteristics of the groups are summarized in Table 1.

Outpatients were recruited from a longitudinal study of recent-onset schizophrenia (see Nuechterlein et al., 1992). They were assessed with the Structured Clinical Interview for the *DSM–IV* (SCID; First, Spitzer, Gibbon, & Williams, 1994) and diagnosed with schizophrenia ($n = 11$; subtypes: 7 undifferentiated, 3 paranoid, 1 disorganized), schizoaffective disorder, depressive type ($n = 2$), or schizophreniform disorder ($n = 3$). Subsequently, diagnoses of schizophrenia ($n = 2$) and schizoaffective disorder, depressed type ($n = 1$) were assigned to the schizophreniform patients. Patients were treated with atypical antipsychotics (risperidone: 9, olanzapine: 4, clozapine: 2), except for 1 patient who received fluphenazine decanoate. The mean number of years between the onset of psychotic symptoms and participation in this study was 7.38 ($SD = 6.57$).

All healthy comparison participants were screened with the SCID to exclude for any personal history of schizophrenia, schizoaffective disorder or other major psychopathology, and any alcohol or substance abuse in the last 3 months. Individuals were excluded if they reported serious medical conditions (e.g., neurological disorders, history of major head trauma, more than a 5-min loss of consciousness) or a family history of schizophrenia or schizoaffective disorder. Except for 1 healthy comparison participant, all participants reported that they were right-handed. All participants were fluent in English and provided written informed consent.

Table 1
Demographic and Clinical Status Data for Schizophrenia Patients and Healthy Comparison Participants

	Healthy comparison participants ($n = 11$)		Schizophrenia patients ($n = 16$)	
Characteristic	*M*	*SD*	*M*	*SD*
Age (years)	31.6	6.1	31.4	7.4
Education (years)	15.2	1.2	14.1	1.6
Parent's highest education (years)	15.5	2.5	15.7[a]	2.3
Gender				
Male	7		11	
Female	4		5	
Ethnicity				
European American	9		10	
Asian American			2	
African American	1		1	
Latino/Latina	1		1	
Biracial			2	
BPRS 18-item total score			27.9	5.4
SANS total score			6.4[b]	3.8

Note. BPRS = Brief Psychiatric Rating Scale; SANS = Scale for the Assessment of Negative Symptoms.
[a] $n = 13$. [b] $n = 15$.

Experimental Task

Participants performed a flanker task similar to that used by Kopp and Rist (1999). Each trial began with a 1-cm fixation cross displayed for 500 ms in the center of the screen, followed by the onset of the flanker stimuli, which were two equilateral triangles or squares arranged in a vertical array. The length of each side of the triangles was 21 mm, and the squares were 15-mm high. Flanker stimuli were displayed for 100 ms before the middle triangle, the target, appeared. The entire array was displayed for 50 ms. Participants were instructed to respond with the hand that corresponded with the direction in which the target was pointing. RT was determined relative to the onset of the target stimulus. Response feedback (see below) was displayed for 500 ms, beginning 1400 ms after the response. The delay between the offset of the target (or feedback when provided) and the onset of the subsequent fixation cross ranged within 1 to 2 s. The flanking triangles were oriented either in the same (facilitation condition) or opposite (interference condition) direction as the target. On some trials, flanking squares were used instead of triangles (neutral condition). Flanker stimuli were positioned either close (2 cm) or far (6.3 cm) from the target. The facilitating and interfering effects of the flankers on performance have been shown to be related to the distance of the flankers from the target (Eriksen & Eriksen, 1974). The array subtended 1° of vertical visual angle in the close condition and 3° in the far condition. The six different types of trials were presented with equal probability in random order. All stimuli were gray and presented on a black background with a 24-cm × 32-cm monitor placed 1.5 m from the participant's face. The response device consisted of two 3.1-cm buttons mounted on a lapboard, with a minimum force of 2 N (.45 lb) required to make a response.

Procedure

EEG recordings were obtained from a 128-channel electrode cap (Neuroscan, El Paso, TX) with Quick-gel applied to the sensors. Only data from primary midline sites are reported. The first 10 participants (7 patients and 3 comparison participants) were tested in a semidarkened, sound-attenuated room, and the remaining participants were tested with the same equipment but in a soundproof chamber. No differences in ERPs were observed between the two rooms.

Before testing, participants were informed they could win a bonus of up to $10 (beyond the payment given for their participation) depending on their task performance. Feedback was provided to reinforce the task contingencies, as indicated below in parentheses. In the accuracy condition, participants were instructed to respond as accurately as possible and were penalized 5¢ for incorrect responses regardless of RT (*Incorrect − 5¢)* and rewarded 1¢ for correct responses made within 300 ms of target onset (*Fast response + 1¢*). These contingencies had the effect of encouraging correct responding while also introducing a modest time pressure that elicited errors sufficient for computation of the ERN and Pe. In the speed condition, participants were instructed to perform the task as quickly as possible and were given a bonus of 4¢ for responding within 270 ms regardless of the accuracy of their response (*Fast response + 4¢*). If participants responded after the deadline, they were penalized 1¢ if their response was incorrect (*Incorrect − 1¢*). This contingency had the effect of encouraging rapid responding, and because participants inevitably missed the RT deadline on a subset of trials, the contingencies also minimized purely random responding that would, in effect, introduce a fundamental difference in the nature of the task between the two instruction conditions.

Task instructions and information regarding reward contingencies were displayed on the video monitor. Participants were questioned prior to beginning the task to ensure comprehension. The order of the speed and accuracy conditions was counterbalanced across participants. Participants completed 60 practice trials at the beginning of each instruction condition. Each task was then administered in 60-trial blocks with brief rest periods between blocks. The experimenter reminded participants of the performance emphasis at the beginning of each trial block. Participants performed 7 blocks of trials for a total of 420 trials in the speed condition and 14 blocks for a total of 840 trials in the accuracy condition. The entire testing session lasted approximately 90 min. At the end of the session, participants were debriefed about the nature of the study and given the entire $10 bonus, regardless of task performance.

Ratings of patients' symptoms during the 2-week ($n = 12$) or 3-week ($n = 4$) period preceding the date of testing were obtained by a clinic staff member from the expanded version of the Brief Psychiatric Rating Scale (Ventura et al., 1993) and the Scale for the Assessment of Negative Symptoms (Andreasen, 1982).

Psychophysiological Recording, Data Reduction, and Analyses

EEG and electrooculogram (EOG) were recorded with Synamps amplifiers and Scan 4.1 software (Neuroscan, El Paso, TX). All physiological data were recorded at a rate of 1000 Hz. EEG was filtered online at .05 and 100 Hz with a gain of 1000. To identify eye movement artifact in the EEG, EOG activity was recorded from electrodes placed above and below the left eye, filtered online at .05 and 200 Hz with a gain of 500. EOG artifact was removed from the EEG with singular value decomposition (see Picton et al., 2000). In data in which cardiac activity was visible, the same method was used to minimize this artifact. Epochs contaminated by participant movement were excluded. Response-locked epochs beginning 1000 ms before and extending 1000 ms beyond the response were then created for each trial. A 1–10 Hz, 96 dB filter was applied, and a 50-ms preresponse baseline was subtracted. Four averages were computed for each participant: correct and incorrect responses in the two instruction conditions.

The amplitude of the ERN and CRN was scored at Fz, FCz, Cz, and Pz with the use of methods similar to those of Gehring et al. (1993) and Luu, Flaisch, and Tucker (2000). The latency of the most negative point from 40 to 160 ms after the response was determined from the FCz channel, relying on the average waveform derived from incorrect trials during the accuracy condition. The average amplitude in a 50-ms period centered on this latency was then computed for each of the four averages for each channel and participant. This method eliminates the opportunity to analyze latency differences but permits measurement of the CRN, which is typically a positive deflection. The Pe was scored at the same sites as the ERN and was computed as the mean amplitude from 170 to 400 ms after the response.

To assess the relationships between ERP amplitude and response characteristics, averages characterized by small, medium, and large ERNs and Pes were created for each participant (see Gehring et al., 1993). All error trials from the accuracy condition and an equal number of correct trials with the same range of RTs from the same task were used. Trials with a missing response on the next trial were excluded. Data from the FCz site (where the ERN and Pe were observed to be maximal) were entered into a stepwise discriminant analysis (SWDA) using SPSS Version 11. The SWDA was performed once for the ERN data and again for the Pe data. For the ERN analysis, the SWDA included data from 20 to 180 ms following the response. For the Pe analysis, data from 170 to 410 ms following the response were used. The SWDA procedure produced a probability value for each trial that indicated the likelihood of the trial being an error. Error trials were sorted on the basis of these probabilities into three bins containing an equal number of trials for each participant (M number of trials per bin per ERP for each participant = 25.9, $SD = 11.9$, range = 5–60). Epochs were averaged within bins to create small, medium, and large ERNs and Pes. Four additional variables were computed for each size category for the ERN and Pe: RT, probability of error correction, and RT and accuracy on the next trial. An error correction was defined as a response occurring with the opposite hand after an error at any time before the onset of the trial feedback or, on trials in which no feedback occurred (e.g., a correct trial not fast enough to earn the bonus for fast responding), before the onset of the fixation cross. The probability of error correction

was computed by dividing the number of error corrections for each ERN size by the total number of error corrections for each participant. Data from participants who made fewer than three response corrections (2 patients and 1 comparison participant) were excluded from these analyses.

An alpha level of .05 was adopted, and the Greenhouse–Geisser method was used to adjust for repeated measures. Simple effects analyses of variances (ANOVAs) with the Bonferroni correction were used for post hoc comparisons on between-groups measures.

Results

Flanker Task Performance

Response accuracy. The manipulation had the intended effect of maximizing correct responding in the accuracy instruction condition. A Group × Instruction ANOVA performed on the percentage of correct responses indicated a main effect for instruction condition. Both schizophrenia patients and comparison participants responded more accurately during the accuracy condition ($M = 89\%$ correct, $SD = 5\%$) than the speed condition ($M = 68\%$ correct, $SD = 13\%$), $F(1, 25) = 84.53$, $p = .00$, $\eta_p^2 = .77$. There was no group difference in response accuracy, $F(1, 25) = 0.21$, $p = .65$, which is consistent with prior studies using the flanker task (e.g., Kopp & Rist, 1999).

The effects of the type of flanker and distance of the flankers from the target on performance accuracy were examined separately for the two instruction conditions with Group × Flanker Type × Distance ANOVAs. Accuracy data are shown in Table 2. In the speed instruction condition, a Group × Flanker Type interaction was observed, $F(2, 50) = 4.22$, $p = .04$, $\eta_p^2 = .14$, in addition to a Distance × Flanker Type interaction, $F(2, 50) = 8.79$, $p = .001$, $\eta_p^2 = .26$, and main effects of distance, $F(1, 50) = 7.94$, $p = .009$, $\eta_p^2 = .24$, and flanker type, $F(2, 50) = 60.70$, $p = .00$, $\eta_p^2 = .71$. Post hoc analyses revealed that the effects of flanker distance and group were significant only in the interference condition, such that accuracy was reduced when flankers were close compared with far from the target, $F(1, 25) = 19.59$, $p = .00$, $\eta_p^2 = .44$. Unexpectedly, schizophrenia patients were more accurate than comparison participants when speed of performance was emphasized, regardless of flanker distance, $F(1, 25) = 5.65$, $p = .025$, $\eta_p^2 = .18$.

Table 2
Task Performance Data as a Function of Instructional Emphasis, Flanker Type, and Flanker Distance

	Healthy comparison participants ($n = 11$)		Schizophrenia patients ($n = 16$)	
Flanker type and distance	RT	% Correct	RT	% Correct
Accuracy instruction condition				
Facilitating				
Close	292 (32)	99 (1)	344 (58)	96 (4)
Far	297 (32)	98 (2)	346 (56)	96 (3)
Neutral				
Close	335 (29)	97 (2)	382 (50)	92 (6)
Far	328 (29)	97 (2)	372 (46)	94 (5)
Interfering				
Close	404 (34)	65 (12)	438 (61)	66 (16)
Far	377 (32)	85 (8)	416 (52)	81 (12)
Speed instruction condition				
Facilitating				
Close	225 (45)	82 (12)	287 (82)	80 (17)
Far	228 (37)	80 (11)	283 (74)	78 (17)
Neutral				
Close	258 (55)	70 (12)	306 (81)	70 (18)
Far	247 (48)	68 (15)	303 (83)	73 (16)
Interfering				
Close	272 (95)	39 (16)	331 (114)	55 (15)
Far	273 (87)	49 (15)	338 (100)	61 (16)

Note: Mean reaction times are presented in milliseconds and standard deviations are in parentheses.

In the accuracy instruction condition, main effects of flanker type and distance were modified by an interaction between these variables, $F(2, 50) = 116.43$, $p = .00$, $\eta_p^2 = .82$. In both flanker distance conditions, accuracy was reduced in the interference compared with the neutral conditions: close, $F(1, 25) = 124.30$, $p = .00$, $\eta_p^2 = .83$, and far, $F(1, 25) = 39.79$, $p = .00$, $\eta_p^2 = .61$; and with the facilitation conditions: close, $F(1, 25) = 118.11$, $p = .00$, $\eta_p^2 = .82$, and far, $F(1, 25) = 42.22$, $p = .00$, $\eta_p^2 = .63$. Accuracy was also reduced in the neutral compared with the facilitation conditions: close, $F(1, 25) = 15.34$, $p = .001$, $\eta_p^2 = .38$, and far, $F(1, 25) = 8.07$, $p = .01$, $\eta_p^2 = .24$. In the interference, $F(1, 25) = 148.29$, $p = .00$, $\eta_p^2 = .86$, and neutral, $F(1, 25) = 4.65$, $p = .04$, $\eta_p^2 = .16$, conditions, but not the facilitation condition, $F(1, 25) = .145$, $p = .71$, the effect of flanker distance was significant, with reduced accuracy when flankers were closer to the target compared with when they were more distant.

RT. A Group × Instruction Condition × Response Accuracy ANOVA on RT revealed an Instruction Condition × Response Accuracy interaction, $F(1, 25) = 7.39$, $p = .012$, $\eta_p^2 = .74$. When performance during the two instruction conditions was examined separately, main effects for response accuracy (i.e., faster responding on error than on correct trials) were observed for both the speed, $F(1, 25) = 85.22$, $p = .000$, $\eta_p^2 = .77$, and accuracy, $F(1, 25) = 317.96$, $p = .000$, $\eta_p^2 = .93$, conditions. However, the magnitude of the effect was larger in the accuracy (correct: $M = 357$ ms, $SD = 9$ ms; incorrect: $M = 272$ ms, $SD = 8$ ms) than in the speed (correct: $M = 277$ ms, $SD = 15$ ms; incorrect: $M = 213$ ms, $SD = 10$ ms) condition. In addition, a main effect of group indicated that schizophrenia patients ($M = 303$ ms, $SD = 11$ ms) responded more slowly than healthy comparison participants ($M = 256$ ms, $SD = 13$ ms), $F(1, 25) = 7.95$, $p = .009$, $\eta_p^2 = .24$.

There were group differences in the rates at which participants received bonuses and penalties in the speed instruction condition, results that are consistent with this RT slowing. Although a repeated measures ANOVA comparing the proportion of trials on which the groups obtained bonus, penalty, or neither feedback did not show a significant Group × Feedback Type interaction, $F(2, 50) = 3.93$, $p = .057$, $\eta_p^2 = .14$, schizophrenia patients earned bonuses for fast responses on fewer trials: patients, $M = 49\%$, $SD = 28\%$, and comparison participants, $M = 69\%$, $SD = 19\%$; $F(1, 25) = 4.45$, $p = .045$, $\eta_p^2 = .15$; and they received penalties for slow erroneous responses on more trials than did comparison participants: patients, $M = 7\%$, $SD = 4\%$, and comparison participants, $M = 3\%$, $SD = 3\%$; $F(1, 25) = 5.17$, $p = .032$, $\eta_p^2 = .18$. In the accuracy instruction condition, there was no Group × Feedback Type interaction, $F(2, 50) = 2.25$, $p = .15$, and no significant differences between groups in the rates at which the

different types of feedback were obtained: bonus, $F(1, 25) = 3.91$, $p = .06$; penalty, $F(1, 25) = 1.71$, $p = .20$; and none, $F(1, 25) = 1.32$, $p = .26$. Comparison participants earned a larger total bonus across instruction conditions ($M = \$9.07$, $SD = \$3.38$) than did schizophrenia patients ($M = \$3.68$, $SD = \$3.51$), $F(1, 25) = 15.84$, $p = .001$, $\eta_p^2 = .39$.

The effects of group, flanker type, and distance on RT were examined for correct responses in the speed and accuracy instruction conditions separately. In the speed instruction condition, a main effect of flanker type was observed, $F(2, 50) = 19.98$, $p = .000$, $\eta_p^2 = .44$. As expected, RTs were slower in the interference condition than in the neutral, $F(1, 25) = 10.88$, $p = .003$, $\eta_p^2 = .30$, and facilitation, $F(1, 25) = 24.14$, $p = .000$, $\eta_p^2 = .49$, conditions and in the neutral compared with the facilitation condition, $F(1, 25) = 26.16$, $p = .000$, $\eta_p^2 = .51$. In the accuracy instruction condition, a main effect of group was observed, $F(1, 25) = 6.42$, $p = .02$, $\eta_p^2 = .20$, characterized by slower responding among schizophrenia patients. In addition, a Flanker Type × Distance interaction modified the main effects of these variables, $F(2, 50) = 27.22$, $p = .000$, $\eta_p^2 = .52$. In the interference and neutral flanker conditions, RTs were slower when the flankers were closer to the target than when they were further away, $F(1, 25) = 48.38$, $p = .000$, $\eta_p^2 = .66$; and $F(1, 25) = 14.63$, $p = .001$, $\eta_p^2 = .37$, respectively. In the facilitation condition, however, there was no effect of flanker distance on RT, $F(1, 25) = 2.45$, $p = .13$, $\eta_p^2 = .09$. These effects are generally consistent with previous reports of an enhanced impact of flankers that are closer to the target (Eriksen & Eriksen, 1974) and of normal or reduced interference effects in schizophrenia patients (Elkins & Cromwell, 1994; Kopp, Mattler, & Rist, 1994).

Midline Distribution of the ERN and Pe

To confirm that the midline distribution of the ERN in the current study was consistent with that of previous reports, we compared ERN amplitude in the accuracy instruction condition at four midline sites (Fz, FCz, Cz, and Pz). One patient was excluded from these analyses because of missing data from the Cz site. A Group × Site ANOVA revealed a main effect for site, $F(3, 72) = 28.19$, $p = .000$, $\eta_p^2 = .54$, such that the ERN was maximal at FCz as expected. Pe amplitude also was found to be maximal at FCz, $F(1, 24) = 6.81$, $p = .004$, $\eta_p^2 = .22$ (as in the study by Bates et al., 2004), although more anterior than the central–parietal maximum reported by others (e.g., Falkenstein et al., 2000; Leuthold & Sommer, 1999). Because prior studies have not detected qualitative topographical differences in the ERN or Pe between schizophrenia patients and healthy comparison participants (Alain et al., 2002; Bates et al., 2004; Mathalon et al., 2002), we conducted all further analyses using only data from the FCz site.

ERN Amplitude, Response Accuracy, and Task Context

Grand average waveforms are presented in Figure 1, and mean ERN–CRN and Pe amplitudes are shown in Figure 2. The hypothesis that schizophrenia patients would exhibit a reduced ERN and enhanced CRN was supported by a Group × Response Accuracy interaction, $F(1, 25) = 16.30$, $p = .000$, $\eta_p^2 = .39$. This interaction remained significant when patients with a schizoaffective or schizophreniform diagnosis were omitted, $F(1, 20) = 11.13$, $p = .003$, $\eta_p^2 = .36$, and when only patients treated with risperidone were examined, $F(1, 18) = 9.84$, $p = .006$, $\eta_p^2 = .35$. Post hoc analyses compared the ERN and CRN separately in the two groups during the two instruction conditions. Consistent with previous reports were our findings that the schizophrenia patients' ERN was diminished relative to that of comparison participants during the accuracy condition, $F(1, 25) = 4.24$, $p = .05$, $\eta_p^2 = .14$. Contrary to our expectations, there were no group differences in ERN during the speed condition, $F(1, 25) = 1.42$, *ns*. As hypothesized, schizophrenia patients also showed a larger CRN than healthy comparison participants during the speed instruction condition, $F(1, 25) = 9.25$, $p = .005$, $\eta_p^2 = .27$. A similar pattern of enhanced CRN in schizophrenia patients relative to healthy comparison participants was observed during the accuracy instruction condition, although this difference did not reach statistical significance, $F(1, 25) = 4.10$, $p = .054$.

To test the hypothesis that the ERN would be larger during the accuracy than the speed instruction condition for healthy participants but not schizophrenia patients, we compared the ERN during the two conditions and conducted post hoc analyses addressing within-group differences. A Group × Instruction interaction in this analysis, $F(1, 25) = 7.50$, $p = .01$, $\eta_p^2 = .23$, modified the main effect of instruction condition, $F(1, 25) = 34.67$, $p = .000$, $\eta_p^2 = .58$. Healthy comparison participants' ERN was larger following errors during the accuracy condition than during the speed condition, $F(1, 10) = 24.43$, $p = .001$, $\eta_p^2 = .71$, results that were consistent with our hypothesis. Unexpectedly, schizophrenia patients' ERN also exhibited sensitivity to task context; the ERN in the accuracy condition was larger than in the speed condition, although the difference was much smaller than that observed in healthy comparison participants, $F(1, 15) = 7.51$, $p = .015$, $\eta_p^2 = .33$.

In comparison to previous reports (Alain et al., 2002; Bates et al., 2004; Bush et al., 2000; Falkenstein et al., 1991, 2000; Mathalon et al., 2002; van Veen & Carter, 2002b), the Pe component in both groups was generally smaller and negative in amplitude, although positively deflected. The broad, centrally maximal distribution and sensitivity of the component to response accuracy, however, suggested that the Pe had been correctly identified and scored. No group differences were observed in either the speed, $F(1, 25) = 1.35$, $p = .25$, or accuracy, $F(1, 25) = 0.33$, $p = .57$, instruction conditions, results consistent with the hypothesis that Pe would not discriminate between schizophrenia patients and comparison participants. Main effects of response accuracy were significant in both the speed, $F(1, 25) = 30.91$, $p = .000$, $\eta_p^2 = .55$, and accuracy, $F(1, 25) = 23.72$, $p = .000$, $\eta_p^2 = .49$, conditions, with larger Pes occurring on error trials compared with correct trials. Pe also was sensitive to task context, with greater amplitude occurring following errors in the speed instruction condition, $F(1, 25) = 12.10$, $p = .002$, $\eta_p^2 = .33$. No effects involving group differences were significant in these Pe analyses. Taken together, results of the ERN and Pe analyses are largely consistent with the hypothesis that schizophrenia patients' ERN is reduced and CRN is enhanced relative to healthy comparison participants and that these group differences have some specificity relative to the Pe.

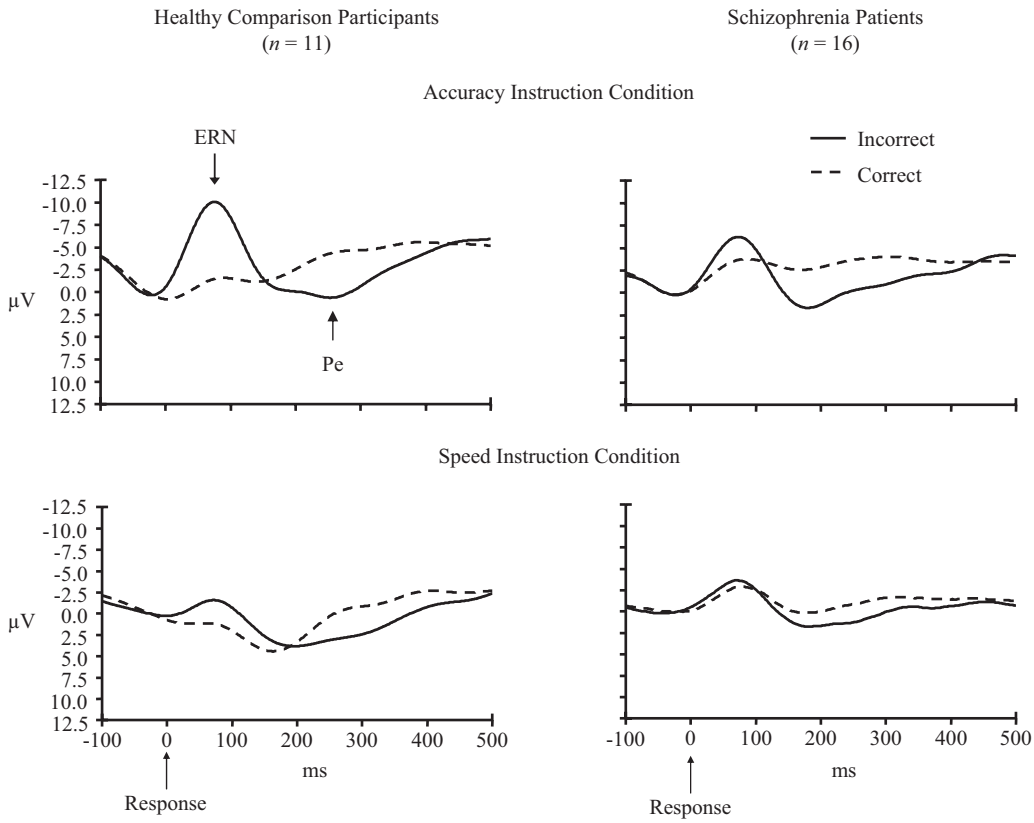

Figure 1. Grand average waveforms for healthy comparison participants and schizophrenia patients during accuracy and speed instruction conditions. Data displayed are from the FCz site. ERN = error-related negativity; Pe = error positivity.

Response-Related Behaviors

The discriminant analysis procedure produced three distinct ERN waveforms for each group. As shown in Figure 3A, a small, medium, and large ERN is apparent for the healthy comparison participants. Among healthy participants, it appears that the activity that best distinguished errors from correct trials occurred in the early portion of the segment included in the analysis, as the greatest discrimination is apparent in the 60–100 ms latency range. For schizophrenia patients, a similar pattern is present, although the medium and small waveforms are distinguished from each other more by temporal characteristics than by amplitude. This may indicate that error-related activation associated with medium and especially small ERNs occurred over a more extended or variable time period for patients than for comparison participants.

To examine the relationships between the ERN and error-related behaviors, we performed Group × ERN Size ANOVAs on RT, RT on the next trial, response accuracy on the next trial, and probability of error correction. RT did not differ between the three ERN size categories, $F(2, 50) = 1.83$, $p = .17$, although responses of schizophrenia patients were slower than those of comparison participants, $F(1, 25) = 6.33$, $p = .019$, $\eta_p^2 = .2$. There also was no effect of ERN size on RT on trials following errors, $F(2, 50) = 2.19$, $p = .49$. In analyses including all trials (not only those used in the discriminant analysis), a Group × Instruction Condition repeated measures ANOVA revealed a main effect of instruction condition on posterror slowing, $F(1, 25) = 15.46$, $p = .001$, $\eta_p^2 = .38$, but no difference between the groups, $F(1, 25) = 0.48$, $p = .49$. Specifically, RTs following errors were an average of only 6 ms slower than those following correct responses in the accuracy condition, whereas RTs following errors in the speed condition were an average of 23 ms *faster* than RTs following correct responses.

Consistent with the hypothesis that schizophrenia patients and healthy comparison participants would exhibit similar ERN-behavior relationships, the ERN was associated with posterror accuracy and error correction in both groups. As shown in Figure 4A, the hypothesized relationship between ERN size and accuracy on trials following errors was observed, in which accuracy was improved following larger ERNs, $F(2, 50) = 3.13$, $p = .05$, $\eta_p^2 = .11$. A main effect of group revealed that schizophrenia patients were generally less accurate following errors than were healthy comparison participants, $F(1, 25) = 6.65$, $p = .02$, $\eta_p^2 = .21$. Error correction data are presented in Figure 4B. A main effect of ERN size on the probability of error correction was observed, $F(2, 44) = 4.10$, $p = .02$, $\eta_p^2 = .16$. The direction of this relationship, however, was the reverse of the hypothesized relationship with corrections more likely following smaller ERNs than larger ERNs. The overall percentage of errors corrected by schizophrenia patients ($M = 27\%$, $SD = 15\%$) did not differ from that of healthy comparison participants ($M = 31\%$, $SD = 18\%$), $F(1, 23) = 0.29$, $p = .54$.

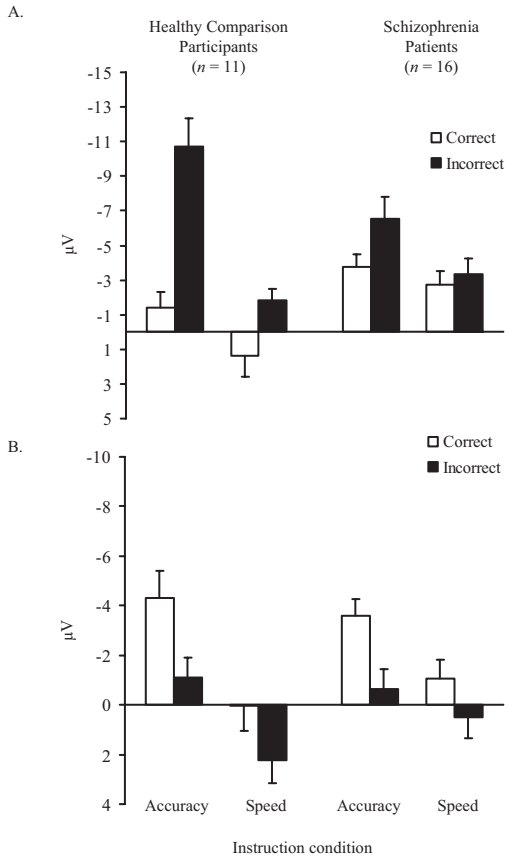

Figure 2. Mean error-related negativity and correct response negativity (A) and error positivity (B) amplitudes for healthy comparison participants and schizophrenia patients during accuracy and speed instruction conditions. Data displayed are from the FCz site. Error bars represent standard error.

Examination of relationships between Pe size and response-related behaviors showed some similarities with those observed in the ERN analyses. Waveforms showing small, medium, and large Pes are presented in Figure 3B. Similar to the relationship observed with ERN size, error correction rates also differed between the three Pe size categories, $F(2, 44) = 6.45$, $p = .005$, $\eta_p^2 = .23$, with more frequent error correction following smaller Pes (see Figure 4C). There were no differences between the three Pe sizes in RT on trials following errors, $F(2, 50) = 2.16$, $p = .13$, or accuracy on trials following errors, $F(2, 50) = 0.16$, $p = .80$, and no effects involving group.

Discussion

The aims of this study were to compare the amplitude of response-related ERPs under varying task demands in schizophrenia patients and healthy comparison participants and to examine the relationships between ERN amplitude and behavioral variables in these groups. As predicted, the ERN was reduced in schizophrenia patients but maximized in healthy comparison participants when performance accuracy was emphasized. Somewhat unexpectedly, ERN amplitude did not differ between patients and comparison participants when speed of responding was rewarded. The CRN was larger in schizophrenia patients than in healthy comparison participants during the speed instruction condition and, to a lesser extent, when contingencies favored accurate responding. These results largely replicate and extend previous findings of reduced ERN amplitude in schizophrenia by demonstrating that this impairment is observed even under task demands that have been shown to potentiate the component. In addition, the findings are consistent with other reports of enhanced CRN in schizophrenia patients (Alain et al., 2002; Mathalon et al., 2002). The picture that emerges, therefore, is not so much one of a lack of monitoring in schizophrenia but of imprecise monitoring, characterized by enhanced activity when it is not appropriate and diminished activity when an erroneous response is executed under conditions in which errors are most costly. This interpretation, however, depends upon the still-controversial assumption that the ERN and CRN reflect the same underlying process. Until the precise nature of the CRN and its properties are fully understood, conclusions regarding its significance must remain tentative.

The theoretical import of these findings differs depending upon the model that is adopted regarding the generation of the ERN. Considered within the framework of error detection theory (e.g., Bernstein et al., 1995; Coles et al., 2001; Scheffers & Coles, 2000), the present results suggest that schizophrenia patients have difficulty distinguishing between correct and incorrect responses, perhaps because of disruptions in phasic dopamine (DA) activity that render the ACC unable to detect changes in the success or failure of ongoing events (Holroyd & Coles, 2002). This interpretation is consistent with long-standing theories of the involvement of DA in schizophrenia (for a review, see Davis, Kahn, Ko, & Davidson, 1991) and reports of abnormal DA transmission in the ACC of schizophrenia patients (Benes, 2000; Suhara et al., 2002). Challenging this idea, however, is the absence of group differences in response accuracy rates despite the ERN abnormalities observed in this and other studies of schizophrenia (Alain et al., 2002; Bates et al., 2004; Kopp & Rist, 1999; Mathalon et al., 2002) and essentially normal modulation of the Pe according to response accuracy (Alain et al., 2002; Bates et al., 2004; Mathalon et al., 2002). It appears improbable that schizophrenia patients could perform experimental tasks with essentially normal rates of accuracy if their ability to discriminate correct from incorrect responses is fundamentally disrupted. One possibility is that error monitoring difficulties in schizophrenia manifest themselves in general slowing of responses rather than increased error rates. Given the sensitivity of the ACC to the loss of reward, it is also possible that group differences in ERN amplitude in the face of normal response accuracy might point to insensitivity to reward and loss of reward (see Holroyd & Coles, 2002).

Within the conflict detection model of ERN generation (Botvinick et al., 2001; Carter et al., 1998; MacDonald et al., 2000; Yeung et al., 2004), results of the current study suggest that schizophrenia patients experience diminished response competition compared with healthy participants during trials on which accuracy of performance is emphasized and an incorrect response is executed. This may be due to premature selection of a response and, thus, the absence of concurrently activated responses. In contrast, during trials on which a correct response is made, it appears that schizophrenia patients experience enhanced response competition compared with healthy comparison participants and that simultaneous

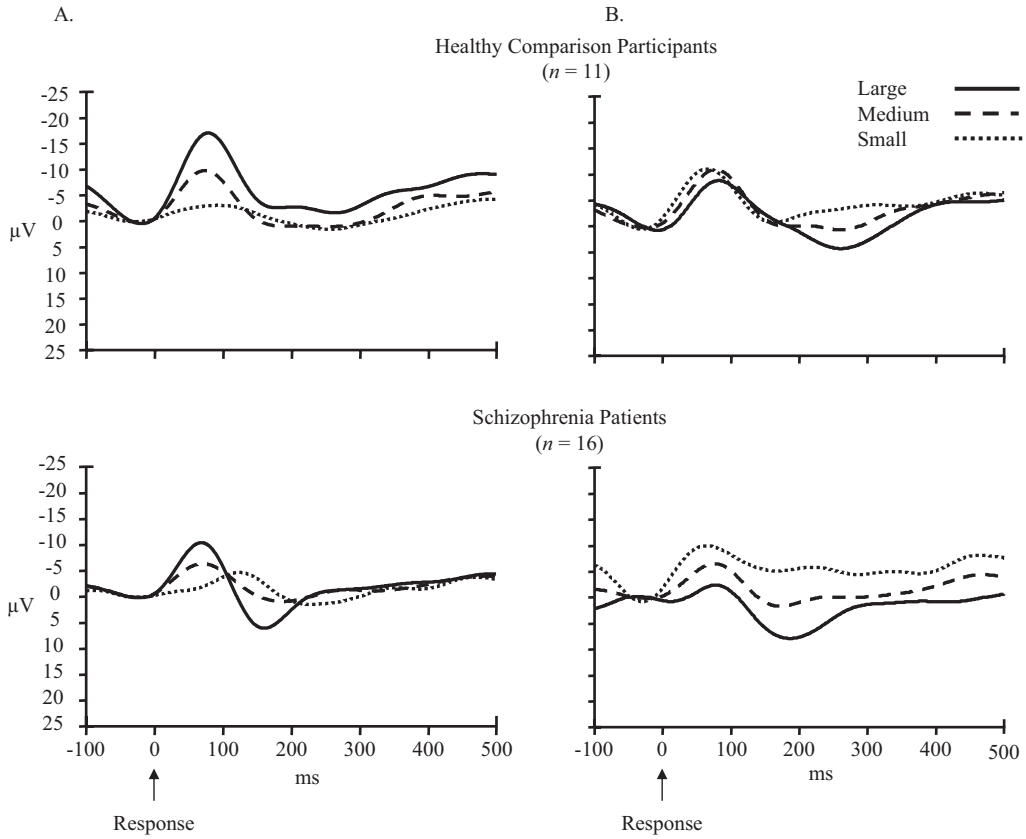

Figure 3. Error-related negativity (A) and error positivity (B) waveforms on error trials in the accuracy instruction condition grouped according to size for healthy comparison participants and schizophrenia patients. Data displayed are from the FCz site.

coactivation of response units persists even though a correct response is executed. If enhanced CRN amplitude in schizophrenia patients is due to increased response competition, however, slower RTs and higher error rates would be expected in patients than in healthy comparison participants. Although increased RTs were observed among schizophrenia patients as compared with healthy participants, it is difficult to discern if this was due to increased response competition or the psychomotor slowing that is consistently observed in schizophrenia patients (for a review, see Nuechterlein, 1977). The absence of group differences in performance accuracy and greater response accuracy in schizophrenia patients than in healthy comparison participants on interference trials during the speed instruction condition argues against the possibility of heightened response competition in patients.

The observed pattern of results also could be explained by failures of context maintenance in schizophrenia (Barch et al., 2003; Braver, Barch, & Cohen, 1999; Cohen et al., 1999; MacDonald & Carter, 2003). Failure by schizophrenia patients to accurately hold online response representations of task demands and appropriate responses could result in disruption in the generation of the ERN, as a result of either the unpredictable occurrence of response competition according to the conflict detection model or failure to accurately discriminate errors from correct responses, consistent with the error detection model. Contrary to this notion, however, results from the present study suggest that the ERN in schizophrenia patients is sensitive to task instruction condition, showing increased amplitude when accuracy of responding is emphasized compared with when speed of responding is encouraged. Results of the flanker task manipulation could be interpreted as providing further evidence of appropriate context maintenance in schizophrenia given that patients performed even more accurately than comparison participants on interference trials during the speed condition. It may be that schizophrenia patients adopted a response strategy of emphasizing accuracy to compensate for their slower RTs and to avoid the penalty for slow incorrect responses, thus demonstrating sensitivity to task context. Alternately, the performance difference between groups on interference trials could be interpreted as reflecting failure on the part of schizophrenia patients to understand task contingencies or an inability to modify their behavior by responding quickly without regard to response accuracy.

Although it is apparent that the present investigation does not lend overwhelming support for one theoretical model over the others, the results do suggest various interesting possibilities. The discussion above also underscores the range and complexity of issues associated with evaluating self-monitoring in schizophrenia patients. Yet another factor to consider is the possible effects of antipsychotic medications on DA activity and, according to the Holroyd and Coles (2002) model, the generation of the ERN. With the exception of a small subset of patients who were tested while

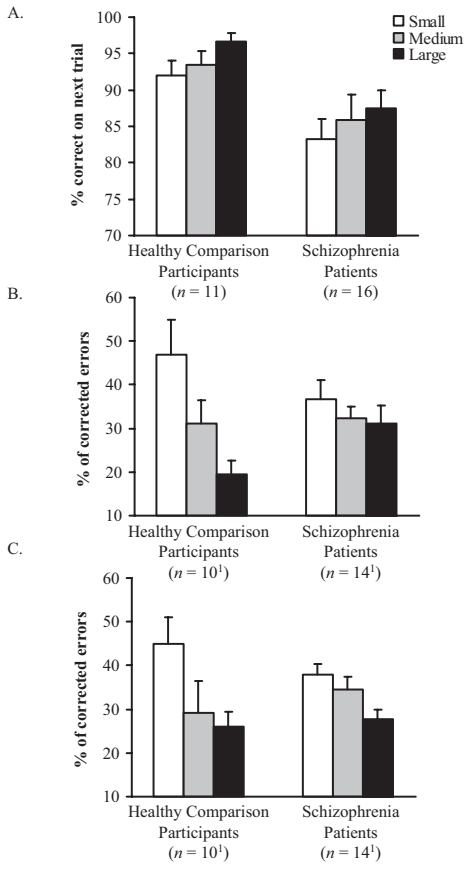

Figure 4. Rates of response accuracy on trials following errors (A) and error correction (B) for healthy comparison participants and schizophrenia patients for small, medium, and large error-related negativities. Error correction rates for small, medium, and large error positivities (C) for healthy comparison participants and schizophrenia patients. Error bars represent standard error. [1]Data from 2 schizophrenia patients and 1 normal comparison subject who made fewer than 3 error corrections are excluded.

unmedicated (Alain et al., 2002) or while their medication status was uncertain (Bates et al., 2004), ERN studies of schizophrenia have included only patients receiving antipsychotic medications. Because these medications act to block DA receptors, they may diminish the effects of phasic DA release, resulting in an attenuation of the ERN. Experimental evidence for this hypothesized effect of antipsychotic medication on the ERN is mixed. Acute administration of haloperidol to healthy participants has been shown to diminish ERN amplitude (Zirnheld et al., 2004), although Kopp and Rist (1999) found medication dose not to be related to ERN amplitude in schizophrenia patients. Moreover, it is unclear how antipsychotic medications could account for the finding of increased CRN amplitude detected in the current and prior studies (Alain et al., 2002; Mathalon et al., 2002).

Another factor that may have resulted in the appearance of a diminished ERN in schizophrenia patients is differential variability in the latency of the response-related brain activity between the two groups. If the variability of the ERN latency from trial to trial (i.e., "latency jitter") is greater in schizophrenia patients than in healthy comparison participants, it would have the effect of "smearing" the component and reducing the amplitude of the peak that is observed in averaged data. The differences in ERN latency observable in the waveforms resulting from the SWDA suggest that there may indeed have been latency jitter or shift among schizophrenia patients, particularly on trials associated with smaller ERNs. It is difficult to conclude, however, that diminished ERN amplitude in schizophrenia patients is due predominantly to shifts or jitter in latency when these participants also exhibited enhanced CRN activity. These issues require careful further study, perhaps using techniques that allow for adjustment of latency differences on a single trial basis, before it can be concluded that the ERN is diminished in schizophrenia as a result of heightened latency variability.

In contrast to the ERN and consistent with previous findings (Alain et al., 2002; Bates et al., 2004; Mathalon et al., 2002), the Pe did not differ between schizophrenia patients and healthy comparison participants. The observation of greater Pe amplitude in the speed condition relative to the accuracy condition was somewhat unexpected. Considering the theorized relationships between Pe amplitude and subjective evaluation of error significance (Bush et al., 2000; Falkenstein et al., 2000), it would be reasonable to expect Pe to be larger in the accuracy instruction condition because of the importance of minimizing errors for successful task performance. In the speed condition, however, an additional evaluative component may have been present, reflecting reinterpretation of the meaning of an error within a task context in which the prepotent behavioral tendency (i.e., to respond correctly) is of reduced relevance compared with the less dominant tendency to respond quickly. This additional level of processing may be reflected in the increased Pe amplitude observed in the speed instruction condition.

Examination of relationships between ERP measures and error-related behaviors supported the hypothesis that these associations would be similar in the two groups. Specifically, both schizophrenia patients and healthy comparison participants showed increased rates of error correction following smaller ERNs and Pes and increased accuracy on trials that followed larger ERNs, although patients were generally less accurate following errors than were comparison participants. The overall similarity of the relationships between the ERN and response-related variables suggests that the functional significance of the ERN does not differ between the two groups. It may be that despite weaker error-related activation detected at the scalp for schizophrenia patients, the neural response to errors is adequate to engage systems involved in error correction and response selection. In contrast to our findings, however, Gehring et al. (1993) reported increased error correction with *larger* ERNs as well as a relationship between ERN amplitude and posterror RT, which was not present for either group in the current study. In the earlier paper, however, corrected responses were determined on the basis of forearm flexor electromyography activity rather than overt responses used in the present study to define error correction. This methodological difference likely accounts for the large difference in error correction rates between the two studies (70% in Gehring et al., 1993, vs. 27% for schizophrenia patients and 31% for healthy comparison participants in the present study) and suggests that partial or weak error corrections, occurring in the present study but not included in the analyses, could account for the inconsistency between the findings. The relationships observed in the current study, nonetheless, are help-

ful in understanding the potential significance of ERN reduction in schizophrenia, suggesting that the effects of diminished ERN and the associated decrease in posterror accuracy might be apparent in failures of executive control in schizophrenia.

The possibility of overlap between stimulus-related ERP components and the ERN cannot be definitively ruled out by the analyses conducted in the present research. It should be noted, however, that ERN size was not associated with RT in either participant group. If stimulus-locked activity was co-occurring with the ERN, larger ERNs would be expected on trials with relatively faster RTs, resulting from the combined N2–ERN. This pattern was not detected, suggesting that the ERN in the present study was largely independent of stimulus-related activity.

In sum, results of the present study advance research in the area of self-monitoring in schizophrenia by demonstrating that schizophrenia patients show diminished differentiation in response-related ERPs between correct and incorrect responses and that this pattern persists across task contexts varying in response demand and reward contingencies. Such anomalies may be a manifestation of neuroanatomical abnormalities found in the ACC in schizophrenia patients as well as disruptions in DA signals to the ACC that may reflect insensitivity to the state of ongoing events. This deficit appears to demonstrate some specificity, as Pe amplitude did not differ between the groups. Despite these abnormalities in ERN generation, schizophrenia patients exhibited normal modulation of both the ERN and the Pe in response to alterations in task context. In addition, ERP-behavior relationships did not differ between the groups, suggesting that the functional characteristics of these components are intact in schizophrenia.

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