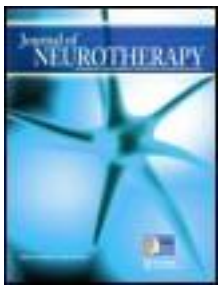

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Impaired Error Monitoring and Correction Function in Autism

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ABSTRACT. *Introduction.* Error monitoring and correction is one of the executive functions and is important for effective goal-directed behavior. Deficient executive functioning, including reduced error monitoring ability, is one of the typical features of such neurodevelopmental disorders as autism, probably related to perseverative responding, stereotyped repetitive behaviors, and an inability to accurately monitor ongoing behavior. Our prior studies of behavioral and event-related potential measures during performance on visual oddball tasks in high-functioning autistic (HFA) children showed that despite only minor differences in reaction times (RTs) HFA children committed significantly more errors.

Method. This study investigated error monitoring in children with autism spectrum disorder (ASD) with response-locked event-related potentials—the error-related negativity (ERN) and error-related positivity (Pe) recorded at fronto-central sites. The ERN reflects early error detection processes, whereas the Pe has been associated with later conscious error evaluation and attention reallocation. RTs in correct trials and posterror slowing in RTs were measured. In this study 14 participants with ASD and 14 age- and IQ-matched controls received a three-category visual oddball task with novel distracters.

Results. ERN had a lower amplitude and longer latency in the ASD group but was localized in the caudal part of anterior cingulate cortex in both groups. The Pe component was significantly prolonged in the ASD group but did not reach significance in amplitude differences

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compared to controls. We found significant posterror slowing in RTs in controls and posterror acceleration in RTs in the ASD group.

Conclusion. The reduced ERN and altered Pe along with a lack of posterror RT slowing in autism might be interpreted as insensitivity in the detection and monitoring of response errors and a reduced ability of execute corrective actions. This might result in reduced error awareness and failure in adjustment when dealing with situations where erroneous responses may occur. This deficit might be manifested in the perseverative behaviors often seen in individuals with ASD. The results are discussed in terms of a general impairment in self-monitoring and other executive functions underlying behavioral and social disturbances in ASD.

KEYWORDS. Autism, cingulate cortex, error monitoring, executive functions, oddball task, reaction time

INTRODUCTION

Autism spectrum disorders (ASD) are pervasive developmental disorders characterized by the early onset of impairments in social and communication skills along with restricted and repetitive interests and activities. These conditions range from a severe form called autistic disorder, through cases known as pervasive developmental disorders not otherwise specified (PDD-NOS), the category that ''includes 'atypical autism'—presentations that do not meet the criteria for autistic disorder because of late age of onset, atypical symptomatology, or subthreshold symptomatology'' (American Psychiatric Association, 2000, p. 84), to a much milder form known as Asperger's syndrome. There is growing evidence that executive function deficits may contribute to these core symptoms (Hill, 2004). One important executive function known to be compromised in ASD is the ability to select a contextually appropriate response among several competing ones, and simultaneously inhibit contextually inappropriate responses to avoid committing an error. Another executive deficit observed during performance on speeded reaction time (RT) tasks in autism is manifested in an abnormality related to response error monitoring and posterror response correction.

Current theory and research suggests that deficits in response monitoring may contribute to social-emotional and social-cognitive impairments in autism (Henderson et al., 2006). Executive deficit hypotheses of autism emphasize that many of the everyday behaviors of autistic individuals, such as perseverative responding, repetitive behaviors, poor imitation skills, and joint attention impairments, may involve an inability to consistently and accurately monitor ongoing behaviors (Mundy, 2003). Therefore, impairments specific to self-monitoring function have been already outlined in earlier models of autism (Russell, 1997; Russell & Jarrold, 1998). Several recent reports (Bogte, Flamma, van der Meere, & van Engeland, 2007; Henderson et al., 2006; Thakkar et al., 2008; Vlamings, Jonkman, Hoeksma, van Engeland, & Kemner, 2008) indicate that children and adult patients with ASD show reduced error processing and deficient behavioral correction after an error is committed. This finding could be explained as a reflection of ASD patients' lower sensitivity to behavioral errors and/or reduced behavior correction ability.

Performance on behavioral tasks is monitored by a brain system that is responsive to errors (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Gehring & Knight, 2000; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Luu, Flaisch, & Tucker, 2000; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003). Evidence from functional magnetic resonance imaging, electroencephalographic (EEG), and event-related potential (ERP) studies outlines that error monitoring is a function of the medial frontal cortex, including the supplementary eye fields, rostral cingulate motor area, and dorsal anterior cingulate cortex (ACC; reviewed in Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). Recent neuropathological studies in autism

suggest the presence of significant minicolumnar abnormalities in brain regions related to error monitoring, that is, medial frontal cortex and ACC (for references, see Casanova, van Kooten, Switala, van England, Heinsen, Steinbuch, Hof, & Schmitz, 2006; Casanova, van Kooten, Switala, van England, Heinsen, Steinbuch, Hof, Trippe, et al., 2006).

Error sensitivity can be readily examined by measuring response-locked ERP components associated with brain responses to errors. Two specific components relevant in this context are the error-related negativity (ERN; more rarely referred to as Ne) and the error-related positivity (Pe). The ERN is a response-locked negative ERP deflection, emerging between 0 and 150 ms after the onset of the incorrect behavioral response—a commission error. Usually this ERN is followed by a positive wave referred to as the Pe potential. Although there is discussion about the exact meaning of the Pe (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005), most studies indicate that the Pe is related to the conscious recognition of the error (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001) or the attribution of motivational significance to the committed error (Falkenstein et al., 2000). This suggests that the ERN reflects an initial automatic brain response as a result of an error, and the Pe possibly indicates the conscious reflection and comprehension of the error (Overbeek et al., 2005). The magnitude of the ERN is associated with behavioral evidence of self-monitoring (i.e., self-correction and posterror slowing responses) and therefore is interpreted as a biomarker of error processing (van Veen and Carter, 2002). Dipole modeling has localized ERN sources to the caudal ACC, whereas Pe has been localized to the more rostral ACC division (Bush, Luu, & Posner, 2000; Gehring & Knight, 2000; Herrmann, Remmler, Ehlis, Heindrich, & Fallgatter, 2004; van Veen & Carter, 2002; West, 2003). ERN and Pe are generally accepted as neural indices of response-monitoring processes in psychophysiological research and clinical neurophysiology.

One of the important research questions is whether this error-related frontal activity is associated with a premorbid trait reflecting an initial deficiency of behavioral control and regulation and whether this deficit can be generated as a result of neuropathological states associated with behavioral control deficits (e.g., PDDs). Several clinical research studies have demonstrated an excessive error processing in patients with obsessive–compulsive disorders (Johannes et al., 2001), anxiety disorders (Markela-Lerenc et al., 2004), and Tourette syndrome (Gehring, Himle, & Nilsenson, 2000). On the contrary, reduced error-processing manifestations were reported in borderline personality disorder (de Bruijn et al., 2006) and schizophrenia (Mathalon et al., 2002). In psychiatric studies, a decreased ERN is typically related to increased severity of psychomotor poverty symptoms (Bates, Liddle, Kiehl, Ngan, 2004). Furthermore, error processing has also been found to be reduced in nonclinical traits such as high impulsivity (Ruchsow, Spitzer, Groen, Grothe, & Kiefer, 2005).

Neuroanatomically and functionally, the ACC provides an interface between frontal action selection processes, limbic emotion or motivation processes, and motor output regulation (Coles, Scheffers, & Holroyd, 2001; Holroyd & Coles, 2002; Taylor, Stern, & Gehring, 2007). The integral role of the ACC in self-monitoring and guiding attention in goal-directed actions suggests that it may be an important focus for autism research. Disturbances in attention regulation and behavioral rigidity may result in social orienting deficits and a chronic disruption of social information processing and social learning that together may contribute to the social, cognitive, and emotional deficits observed in autistic children (Dawson, Meltzoff, Osterling, Rinaldi, & Brown, 1998; Klin, Warren, Schultz, & Volkmar, 2003; Mundy, 1995; Mundy & Neal, 2001).

Several neuroimaging studies (Barnea-Goraly et al., 2004; Hall, Szectchman, & Hahmias, 2003) suggest that anomalous functioning of the ACC may distinguish between individuals with autism and controls. Haznedar et al. (2000) observed that a sample of children with autism displayed hypometabolism in the right ACC relative to

controls, whereas an Asperger's disorder subsample displayed left ACC hypometabolism relative to controls. There have been also several ERN-based empirical demonstrations of connections between ACC function and autism. Children with high-functioning autism displayed longer ERN latencies but did not differ in amplitude of the ERN relative to children in the control group in the Eriksen flanker task (Henderson et al., 2006). There is other evidence of abnormal response monitoring in autism, in particular reduced error self-correction (Russell & Jarrold, 1998) and reduced posterror slowing, a compensatory mechanism to improve performance on the subsequent trial (Bogte et al., 2007). Because the evaluation of ongoing behavior and its consequences is necessary to determine whether current behavior adjustment strategies should be maintained, abnormal response monitoring and deficient adaptive correction may contribute to the behavioral inflexibility associated with ASD.

The current study examined the possibility that patients with autism exhibit a deficiency in the processing of error, reflected by a reduction in the ERN and Pe response-locked brain potentials. Further, we expected that ASD patients would have higher rates of error in the cognitive task. In addition, in normal participants it has been observed that after an error has been committed, participants show slower RT and decreased error rates. These changes have been interpreted as revealing changes in the speed–accuracy strategy of the participant possibly due to error-induced control processes. We investigated the possibility that patients with ASD show a deviant posterror response pattern.

METHODS

Participants

Participants with ASD (study enrollment eligibility age range 9–21 years) were recruited through the University of Louisville Weisskopf Child Evaluation Center. Diagnosis was made according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev. [*DSM–IV–TR*]; American Psychiatric Association, 2000) and further ascertained with the Autism Diagnostic Interview–Revised (Le Couteur, Lord, & Rutter, 2003). All ASD patients were also clinically evaluated with the Autism Diagnostic Observation Schedule (Lord et al., 1989) and had a medical evaluation by a developmental pediatrician. All participants had normal hearing based on past hearing screens. Participants either had normal vision or wore corrective lenses. Participants with a history of seizure disorder, significant hearing or visual impairment, a brain abnormality conclusive from imaging studies, or an identified genetic disorder were excluded. All participants were high-functioning persons with ASD with full scale IQ greater than 80 assessed using the Wechsler Intelligence Scale for Children, Fourth Edition (WISC–IV; Wechsler, 2003) or the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 2004).

Social and behavioral functioning for participants was evaluated utilizing caregiver questionnaires and clinician ratings. The following sections describe the measures:

Aberrant behavior checklist (ABC). The ABC (Aman & Singh, 1994) is a clinician-administered rating scale assessing five problem areas: irritability, lethargy/social withdrawal, stereotypy, hyperactivity, and inappropriate speech, and is based on caregiver reports. Each area contains multiple items receiving a rating from 0 to 3. Items are summed and high scores for each area reflect severity of the problem area.

Social responsiveness scale (SRS). The SRS (Constantino & Gruber, 2005) is a caregiver-completed rating scale assessing social interest and interaction. The scale provides a dimensional measure of social interaction allowing the rating of social skills in autism as well as nonautistic individuals.

Repetitive behavior scale–revised (RBS). The RBS (Bodfish, Symons, & Lewis, 1999) is a caregiver-completed rating scale assessing repetitive and restricted behavior patterns. The RBS is a measure of different behaviors: stereotyped, self-injurious, compulsive, ritualistic, sameness, and restricted range (Bodfish, Symons, Parker, & Lewis, 2000). Items from scales are summed to obtain a measure of the severity of repetitive behavior.

Half of the participants with autism in this study were taking medication: One patient was taking stimulants (Concerta—*Methylphenidate HCl*), and 6 patients were taking antidepressants (Prozac—*Fluoxetine HCl*, Zoloft—*Sertraline HCl*) and mood stabilizers (Depakote—*Divalproex Sodium*, Abilify—*Aripiprazole*).

Controls were recruited through advertisements in the local media. All control participants were free of neurological or significant medical disorders; had normal hearing and vision; and were free of psychiatric, learning, or developmental disorders based on self- and parent reports. Participants were screened for history of psychiatric or neurological diagnosis using the Structured Clinical Interview for *DSM–IV* Non-Patient Edition (First, Spitzer, Gibbon, & Williams, 2001). Participants within the control and autism groups were attempted to be matched by age, full-scale IQ, and socioeconomic status of their family. Socioeconomic status of ASD and control groups was compared based on parent education and annual household income. Participants in both groups had similar parent education levels.

Participants and their parents (or legal guardians) were provided with full information about the study including the purpose, requirements, responsibilities, reimbursement, risks, benefits, alternatives, and role of the local Institutional Review Board. The consent and assent forms approved by the Institutional Review Board were reviewed and explained to all participants who expressed interest to participate. All questions were answered before a consent signature was requested. If the individual agreed to participate, she or he signed and dated the consent form and received a copy countersigned by the investigator who obtained consent.

ERP Data Acquisition and Signal Processing

EEG data were acquired with a 128-channel Electrical Geodesics Inc. (EGI) system (v. 200) consisting of Geodesic Sensor Net electrodes, Net Amps and Net Station software (Electrical Geodesics Inc., Eugene, OR) running on a Macintosh G4 computer. EEG data are sampled at 500 Hz and 0.1 – 200 Hz analog filtered. Impedances were kept under 50 KΩ. According to the Technical Manual of EGI (2003) this Net Sensor electrode impedance level is sufficient for quality recording of EEG with this system.

The Geodesic Sensor Net is a lightweight elastic thread structure containing silver/silver chloride electrodes housed in a synthetic sponge on a pedestal. The sponges are soaked in a potassium chloride solution to render them conductive. EEG data are recorded continuously. EEG channels with high impedance or visually detectable artifacts (e.g., channel drift, gross movement, etc.) were marked as "bad" using Net Station event marker tools in "on-line" mode for further removal in "off-line" mode using Net Station Waveform Tools.

Response-locked EEG data are segmented off-line into 1,000-ms epochs spanning 500 ms prestimulus to 500 ms poststimulus around the critical stimulus events—commission error. Data are digitally screened for artifacts (eye blinks, movements), and contaminated trials are removed using artifact rejection tools. The Net Station Waveform Tools' Artifact Detection module in "off-line" mode marks EEG channels "bad" if the fast average amplitude exceeds 200 μV, the differential average amplitude exceeds 100 μV, or if the channel has zero variance. Segments are marked bad if they contain more than 10 bad channels or if eye blinks or eye movements are detected ($>$70 μV). After detection of "bad" channels, Net Station's "Bad channel replacement" function is used for the replacement of data in "bad" channels with data interpolated from the remaining good channels (or segments) using spherical splines (more information on interpolation methods used in EGI Net Station systems can be found in Fletcher, Kussmaul, & Mangun, 1996; Luu et al., 2001; and Srinivasan, Tucker, & Murias, 1998).

Remaining data are digitally filtered using 60 Hz Notch and 0.3–20 Hz bandpass filters and are then segmented by condition and averaged to create ERPs. Averaged ERP data are baseline corrected (500 ms) and

re-referenced into an average reference frame. All stimulus presentation and behavioral response collection is controlled by a PC computer running E-prime software (Psychology Software Tools, Inc., Pittsburgh, PA). Visual stimuli are presented on a 15-in. display. Manual responses are collected with a five-button keypad (Serial Box, Psychology Software Tools, Inc, Pittsburgh, PA).

Three Stimuli Visual Oddball with Novel Distracters

This test represents a traditional visual three-stimuli oddball task. Stimuli letters "X," "O," and novel distracters ("v," "∧," ">," and "<" signs) are presented on the screen after a fixation mark "+." One of the stimuli ("O") is presented on 50% of the trials (frequent standard); the novel stimuli stimulus (e.g., ">") is presented on 25% of the trials (rare distracter), whereas the third ("X") is presented on the remaining 25% of the trials and represents the target. Participants are instructed to press a key when they see the target letter on the screen. Each stimulus is presented for 250 ms, with a 1,100-ms to 1,300-ms intertrial interval. There are 480 trials in total, with a break every 240 trials. The complete sequence takes 20 min.

Behavioral Measures

Behavioral response measures were mean RT (in milliseconds) and response accuracy (percentage of correct hits). Number and percentage of commission and omission errors were calculated for each participant.

ERP

ERP dependent measures were adaptive mean amplitude and latency of two ERP peaks (i.e., ERN, Pe) within a temporal window across two region-of-interest (ROI) channel groups at the midline fronto-central area. Each ROI contained at least four electrodes. A list of ERP-dependent variables included stimulus-averaged amplitude and latency of the fronto-central ERP components: ERN (40–150 ms poststimulus) and Pe (100–200 ms).

The frontal and fronto-central ROIs for both ERN and Pe components included the following EGI channels: midline frontal and fronto-central ROI—Fz and FCz, and the extended fronto-central ROI contained five EEG sites—FCz, two left EGI channels 7 and 13 (between FCz and FC3 and C1) and two right EGI channels 113 and 107 (between FCz and FC2 and C2). The layout of these ROIs is presented in Figure 1.

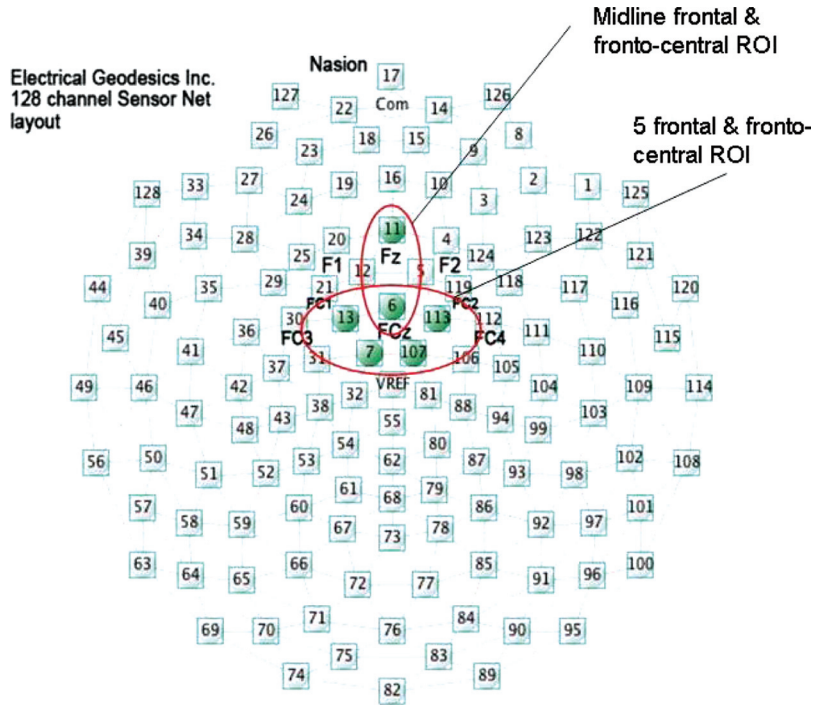
Dipole Source Localization

Dipole source localization was performed on grand average ERP files using the Brain Electrical Source Analysis software package (BESA v.5.2., MEGIS, Munich, Germany). BESA is one of the most widely used software packages for source analysis and dipole localization in EEG research (Schreg, 2005; Schreg & Berg, 1996). BESA provides a standardized, realistic head model and allows for hypothesis testing for EEG/ERP data.

Statistical Data Analysis

Statistical analyses were performed on the subject-averaged behavioral and ERP data with the subject averages being the observations. The primary analysis model is the repeated measures analysis of variance (ANOVA), with dependent variables being RT, accuracy, commission and omission error rate (percentage), posterror RT, and all the specific ERP components' (ERN, Pe) amplitudes and latencies at selected ROIs. The data of each behavioral and ERP dependent variable for each relevant ROI was analyzed using a one-way ANOVA. The between-subject factor was group (ASD, control [CNT]). Post hoc analysis was conducted where appropriate. A priori hypotheses were tested with student's t tests for two groups with unequal variance. In all ANOVAs, Greenhouse-Geisser corrected p values were employed where appropriate. SPSS v.14 and Sigma Stat 3.1 packages were used for statistical analysis.

FIGURE 1. Electrical Geodesics Inc. Sensor Net layout (2.1 version) for 128-channel EEG sites with channel numeration. *Note.* Midline frontal (Fz) and fronto-central (FCz), and a 5 channel fronto-central region-of-interest are highlighted.



RESULTS

Participants

The mean age of 14 participants enrolled in the ASD group was 13.0 ± (standard deviation) 2.5 years (range = 9–18 years, 11 male, 3 female), whereas the mean age of the CNT group ($N = 14$) was 14.1 ± 3.9 years (9–21, 9 male, 5 females). The age difference between groups was not significant ($p = .39$, *ns*). All children with autism were high functioning. Mean Full-Scale IQ score for children with autism was 92.2 ± 15.3 and was not significantly different from the group IQ of the typical children. The tests were Full-Scale IQ scores from either the WISC–IV (Wechsler, 2003) or the WASI (Wechsler, 2004). Descriptive statistics of behavioral evaluations using ABC (Aman & Singh, 1994), SRS (Constantino & Gruber, 2005), and RBS (Bodfish et al., 1999) are presented in Table 1. The approximate household incomes used to assess socioeconomic status of children did not reveal any statistically significant group differences. All participants except two in the ASD group and one in the CNT group were right-handed (assessed using Edinburgh handedness inventory, Oldfield, 1971).

Behavioral Responses

RT to targets in the ASD group was not different from the typical CNT group, $F(1, 27) = 0.29$, $p = .59$, *ns*, but the difference in commission error rate was significantly higher in the ASD group (21.7 ± 29.1% in ASD vs. 4.8 ± 6.1% in CNT), $F(1, 27) = 4.41$, $p = .046$. Mean post-error RT was faster in the ASD group compared to the CNT group (420 ± 94 ms in ASD vs. 519 ± 99 ms in CNT), $F(1, 27) = 7.21$, $p = .012$. The difference between mean RT in correct trials and posterror trials (i.e., mean posterror RT minus correct trial RT) was negative in ASD but positive in CNT,

TABLE 1. Descriptive statistics of behavioral evaluations.

Scale	*N*	*M*	*SD*
Repetitive behavior[a]	14	27.79	15.34
Social awareness[b]	14	83.00	8.20
Irritability[c]	14	9.86	6.16
Hyperactivity[d]	14	14.07	8.25

[a]Raw score for Repetitive Behavior Scale–Revised; higher score indicates more impairment (Bodfish et al., 1999).
[b]*T* score for Social Awareness subscale of Social Responsiveness Scale; higher score indicates more impairment (Constantino & Gruber, 2005).
[c]Raw score for Irritability subscale of the Aberrant Behavior Checklist; higher score indicates more impairment (Aman & Singh, 1994).
[d]Raw score for Hyperactivity subscale of the Aberrant Behavior Checklist; higher score indicates more impairment (Aman & Singh, 1994).

and this between-group difference was significant, $F(1, 27) = 5.22$, $p = .031$. Figure 2 illustrates posterror slowing of RT in CNT and posterror speeding in the ASD group.

A histogram of RT distribution in both groups shows that most ASD children were increasing speed after errors (13 of 14), whereas most typical children were slowing RT after committed error (11 of 14). See Figure 3.

ERPs

Frontal and fronto-central ERN and Pe amplitude. Amplitude of the ERN across the frontal and fronto-central ROI in the ASD group compared to controls was significantly less negative ($-0.29 \pm 6.68\,\mu V$ in ASD vs. $-5.50 \pm 5.76\,\mu V$ in CNT), $F(1, 27) = 4.88$, $p = .036$. Amplitude of the Pe was not different ($p = .14$, *ns*). See Figure 4.

Topographic amplitude mapping. Two-dimensional topographic maps (Figures 5 and 6) illustrate group differences in ERN (Figure 5) and Pe (Figure 6). Figures 7 and 8 show 3D topographic maps around peaks of ERN (Figure 7) and Pe (Figure 8) only in the ASD group ($N = 14$). Note slightly more leftward oriented peaks of scalp posterror positivity in group of children with autism (visible both at 2D and 3D topographic maps).

Latency. Latency of the ERN was significantly prolonged in the ASD group (midline Fz-FCz ROI, 106 ± 41 ms in ASD vs. 76 ± 22 ms in CNT), $F(1, 26) = 4.97$, $p = .035$; five channel ROI, respectively (103 ± 46 ms vs. 71 ± 22 ms), $F(1, 26) = 5.22$, $p = .031$. The data of one ASD patient

FIGURE 2. Posterror reaction time (RT) differences from correct response RTs (means with standard errors [SE]). *Note.* The group of children with autism show faster posterror RT, whereas the group of typical children slowed down the speed of RT after error.

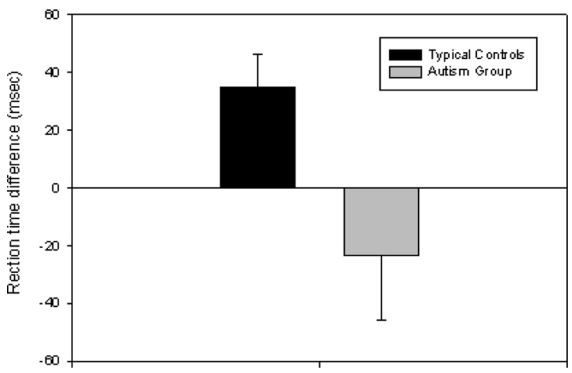
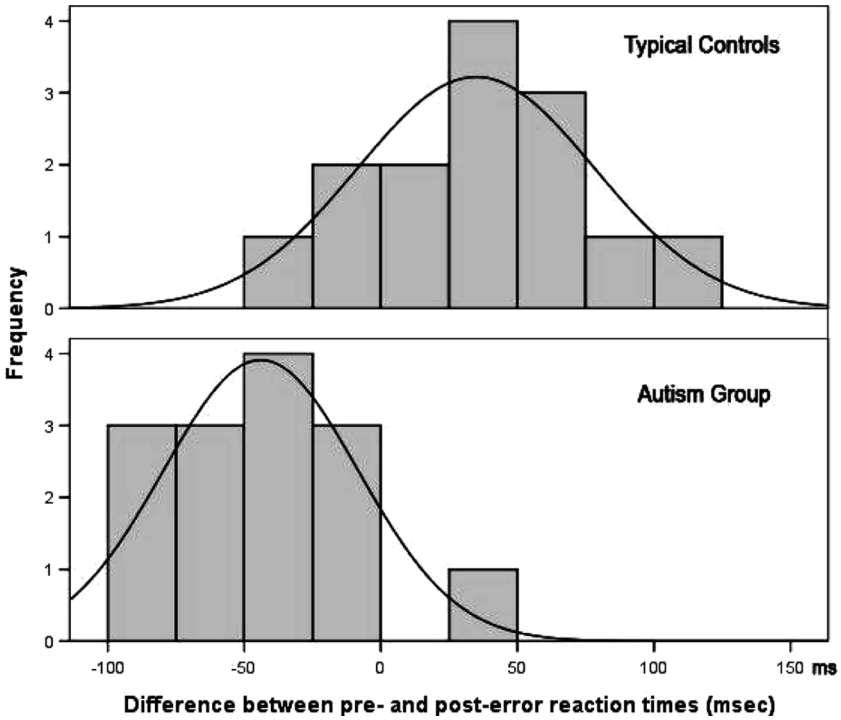


FIGURE 3. Histogram of distribution of individual posterror RTs in autism and typical control groups. *Note.* Except one child all patients with autism demonstrate faster posterror RT compared to correct response RTs.



were excluded from latency analysis due to artifacts. Latency of the Pe across the midline frontal and fronto-central channels was also significantly prolonged in the ASD group (200 ± 44 ms vs. 169 ± 30 ms), $F(1, 27) = 4.47$, $p = .045$.

Dipole Source Localization

Dipole source localization analysis allowed for placement of a dipole (Principal Component Analysis–based loading 93.6%) for ERN in a more caudal division of the

FIGURE 4. Error-related negativity (ERN) and error-related positivity from the frontal and fronto-central midline EEG sites. *Note.* Grand average waveforms ($N = 14$ per group) show lower amplitude of the ERN at all EEG recording sites in the autism group.

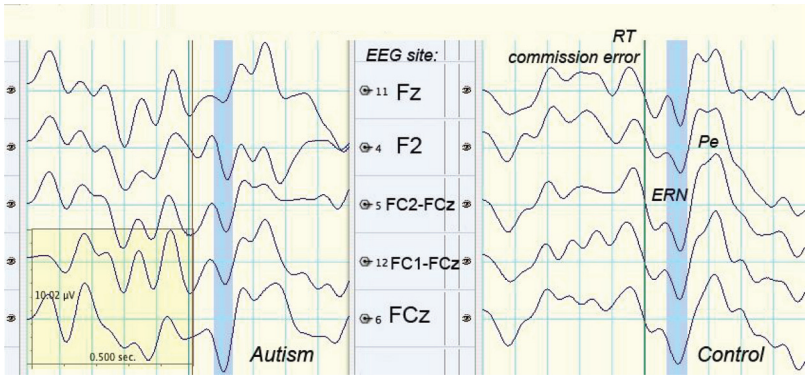


FIGURE 5. Two-dimensional topographic maps with spline interpolations show more negative error-related negativity (ERN; dark blue) in the typical controls group (peak at 100 ms posterror in control group and at 110 ms posterror in the autism group).

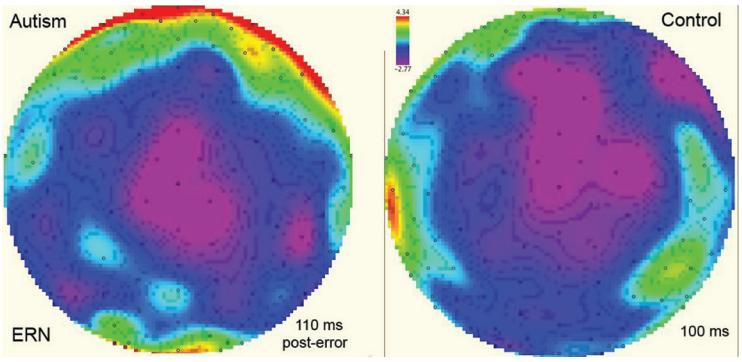


FIGURE 6. Two-dimensional topographic maps with spline interpolations show a more positive fronto-centrally distributed error-related positivity (Pe; red) in the typical controls group (210 ms posterror in control group; 220 ms posterror in the autism group).

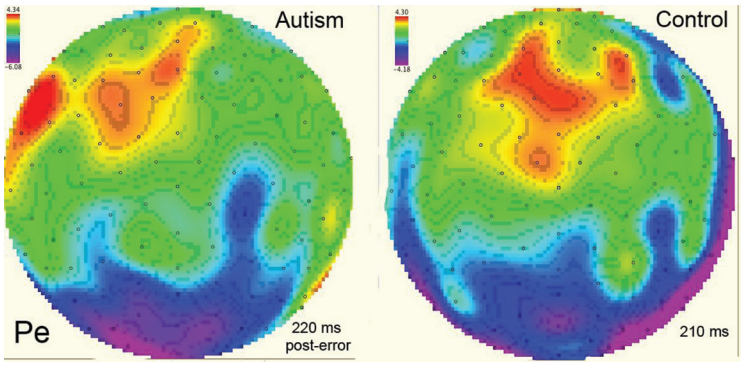


FIGURE 7. Three-dimensional topographic map created in Brain Electrical Source Analysis software shows error-related negativity (blue) over the fronto-central EEG recording sites (grand average, autism group; $N = 14$).

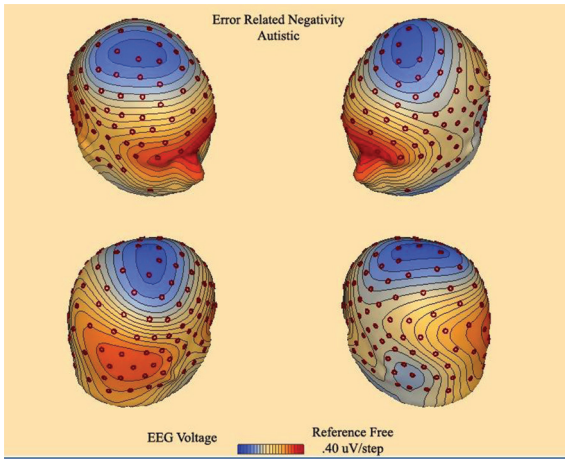
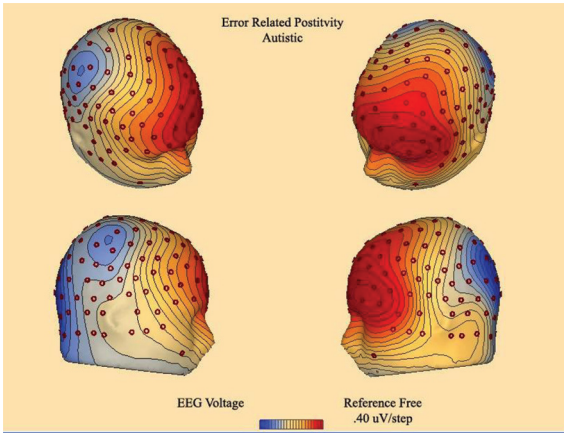


FIGURE 8. Three-dimensional topographic map created in Brain Electrical Source Analysis software shows error-related positivity (red) over the frontal EEG recording sites (grand average, autism group; $N = 14$).



ACC, whereas for Pe a dipole (Principal Component Analysis loading 76.9%) was placed in a more rostral division of the ACC. The dipole source placing is illustrated for the CNT group in Figures 9 and 10. Statistical analysis was not performed because individual participants' dipole placements were not analyzed but rather only group grand average ERPs were used.

FIGURE 9. Dipole source localization and orientation created in Brain Electrical Source Analysis software shows a single dipole with 93.6% loading (using Principal Component Analysis) placed in the caudal division of the anterior cingulate cortex for the error-related negativity (grand average for the control group, $N = 14$).

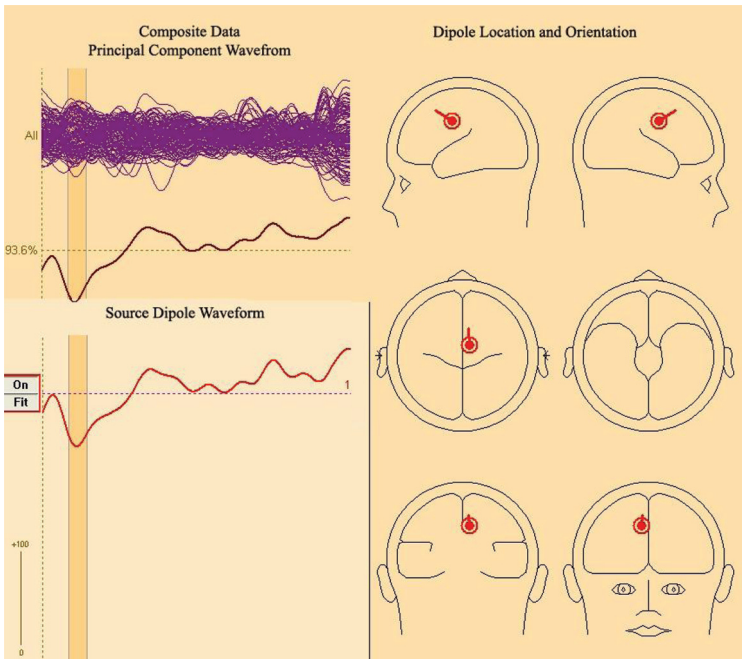
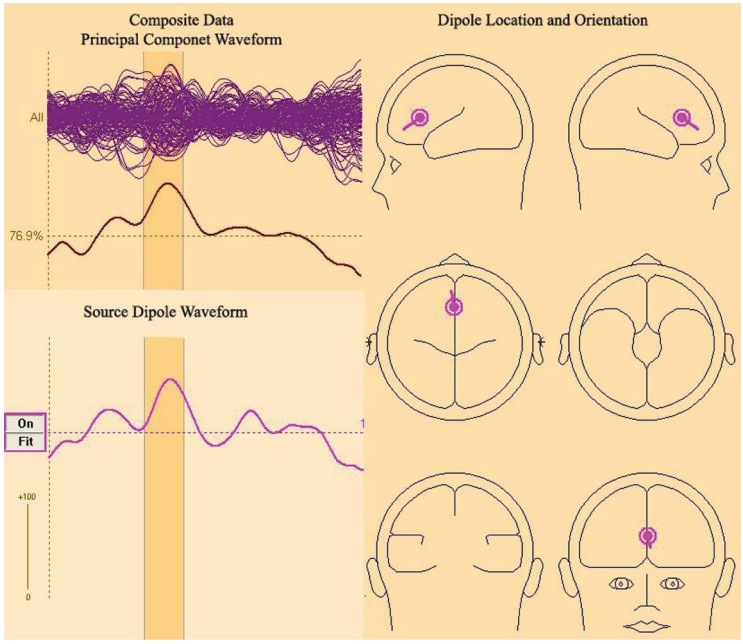


FIGURE 10. Dipole source localization and orientation created in Brain Electrical Source Analysis software shows a single dipole with 76.9% loading (using PC A) placed in the rostral division of the anterior cingulate cortex for the error-related positivity (grand average for the control group, $N = 14$).



DISCUSSION

The current study shows that the ERN and the Pe component of the response-locked ERP were substantially decreased in children with autism as compared to typical controls. In particular the amplitude of ERN was less negative and latency of both ERN and Pe were prolonged in the ASD group as compared to the typically developing children. The ERN is an electroencephalographic measure associated with the commission of errors, thought to be independent of conscious perception (Franken, van Strien, Franzek, & van de Wetering, 2007), whereas the Pe is thought to reflect the motivational or emotional significance of the error or, in another words, the conscious evaluation of the error (Overbeek et al., 2005). The findings that both ERN and Pe are altered in autism may suggest that ASD patients are not only less sensitive to committed errors but also less aware of their errors probably attributing less significance to them. Inadequate and inflexible responsiveness to errors may underlie one of the typical characteristics of autism spectrum disorders, namely, the persistence of stereotyped behaviors.

It cannot be ruled out that the present ERN and Pe findings are influenced by deficits in earlier perceptual processes, or attentional and working memory processes in children with autism, that might be reflected in altered stimulus-locked early and late ERPs. Although we did not observe a significant effect of group on the frontal N200 amplitude (Sokhadze et al., 2009), we found a significantly delayed latency of the N200 to novel distracters in a similar three-category oddball task suggesting that early processes taking place before the response may also be affected in autism. It has been suggested (Yeung & Cohen, 2006; Yeung, Cohen, & Botvinick, 2004) that both the response-locked ERN and the stimulus-locked frontal N200 might reflect similar processes (i.e., response conflict detection and monitoring) and have similar neural correlates (i.e., the ACC).

On the behavioral level, we found no group differences in RT and only modest

group differences between the percentages of commission (and not omission) error in the novelty task. After an error, ASD patients did not show accuracy improvement through posterror RT slowing as typical controls did. Normally, performance on these trials is improved as a result of a change in speed–accuracy strategy, which reflects executive control functioning (Burle, Possamai, Vidal, Bonnet, & Hasbroucq, 2002). The worsened posterror performance of ASD children suggests the presence of an executive control deficiency. The impairment of adaptive error-correction behavior may have important consequences in daily life as optimal error-correction is necessary for adequate behavioral responses.

As demonstrated in previous studies (Ridderinkhof et al., 2004), the posterior medial frontal cortex, more specifically the rostral ACC division, is the main brain area responsible for error processing, suggesting that ASD patients have reduced posterior medial frontal cortex functioning. This area is involved when there is a need for adjustments to achieve goals (Ridderinkhof et al., 2004). The current finding that children with ASD have an impaired ability to improve their response accuracy by slowing down the response speed on posterror trials corresponds with this notion. However, it is necessary to take into account that observed significant group differences between ASD and typical controls are manifested not only in the behavioral performance measures on RT tasks (RT, error rate) and associated response monitoring indices (both to erroneous and correct) but also in terms of amplitude and latency characteristics of ERP components preceding motor response (frontal and parietal P100, N100, P200, N200) and those reflecting context update and closure (e.g., P300, N450) in a visual oddball task (Sokhadze et al., 2009). The sum of the group differences across these behavioral and stimulus- and response-averaged ERP indices of the ASD patients' performance is that it reflects global deficits in attentional processes, more specifically deficits in effective differentiation of target and distracter stimuli. This latter interpretation is supported by the significant differences between the ASD patients and typically developing controls in terms of the stimulus-locked ERP amplitudes and latencies, and the correlation between participants' behavioral performance measures and specific ERP components magnitude.

Posterror adaptive correction of responses might be explained by some recent neurobiological findings. There are reports about an excessive preservation of short-distance connections (i.e., local overconnectivity) and relative poor long-distance connections (i.e., distant underconnectivity) in the neocortex of individuals with autism (Casanova, 2005, 2006; Just, Cherkassky, Keller, & Minshew, 2004; Williams & Casanova, 2009). These cortical connectivity abnormalities may explain why persons with autism tend to focus on details rather than perceiving the whole Gestalt. This overfocusing on details may imply an excessively laborious and ineffective way of handling each trial in the cognitive test, and lower availability of resources after an error when effort is needed to react appropriately. This may result in insufficient activation of the ACC (Bogte et al., 2007), and thus error detection and posterror reaction may be hampered (Bauman & Kemper, 2005; Minshew, Sweeney, Bauman, & Webb, 2005). Structural and functional deficiencies of the ACC may contribute to the atypical development of joint attention and social cognition in autism (Mundy, 2003). Our interpretation of the results of this study is consistent with many aspects of theory and research that suggests that ACC-mediated response monitoring may contribute to social-emotional and social-cognitive development in autism (Mundy, 2003). However, although emphasizing the possible role of ACC-related self-monitoring deficits in autism, Mundy (2003) also noted that according to Devinsky and Luciano (1993), these ACC impairment related behavioral deficits emerge only when they are combined with disturbances in other related functional neural networks, for example, dorsolateral prefrontal cortex.

There are several limitations in this study that should be mentioned. We could not rule out medication effects in studying the

neurobiology of autism. Approximately half of the children in our ASD sample were medicated at the time of study, and there was a variability in the type of medication children were taking. Therefore, it was not possible to analyze the associations between specific classes of medications (i.e., stimulants, antidepressants, mood stabilizers) and the ERN/Pe and behavioral measures in this study of only 14 ASD children. Another limitation or methodological issue to consider in this study was the large proportion of participants in both the ASD and control children samples that had relatively low numbers of commission error trials. The number of errors was introducing some additional variability in the amplitude and latency characteristics of individual participant's data. This is a critical issue in all error monitoring research that has to be considered given the large variance of number of error trials on which the ERN and Pe analyses are based. Finally, the dipole source localization in this study was performed only on grand average ERN/Pe waveforms using realistic head model (for adults), and this prevented us from being able to make any statistical analysis to make any definitive statements about the individual differences in the ERN/Pe dipole localizations in our population of children and adolescents. The BESA-based dipole source localization was mostly for demonstration purposes taking into account the extensive literature on the likely ACC source localization of ERN and Pe dipoles (for a review, see Holroyd & Coles, 2002).

CONCLUSIONS

In summary, the present findings reveal that autism is associated with reduced error processing and impaired behavioral correction after an error is made. Because adequate error processing is necessary for optimal behavioral performance, it is plausible that these deficits contribute to the maintenance of the preservative behaviors typical for autism.

Our study reports abnormal response monitoring and correction functions observed in behavioral and electrocortical indices of the ACC in ASD that might be related to the restricted, repetitive behavior typical of this neurodevelopmental disorder. This abnormal function may result from compromised functional and structural connectivity in the neural circuitry subserving response monitoring and error correction. These findings suggest that functional abnormalities of the ACC reflected in lower amplitude and delayed ERN and Pe measures may compromise response monitoring and contribute to behavioral repetition in ASD. Impairments in an ability to correctly and timely evaluate committed error and to learn from errors may lead to behavior that is rigid and repetitive rather than adaptively guided by action outcomes. Deficits in adjustments of erratic behavior during interaction with peers may as well affect social interaction of children with autism. Elucidating the neurobiological basis and clinical significance of response monitoring and correction deficits in ASD represents a promising direction for further quantitative EEG-based research. The ERN and Pe along with behavioral performance measures can be used as functional outcome measures to assess the effectiveness of behavioral interventions (e.g., social skills training) or neurotherapies (e.g., repetitive transcranial magnetic stimulation or neurofeedback) in children with ASD and thus may have important practical implications.

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