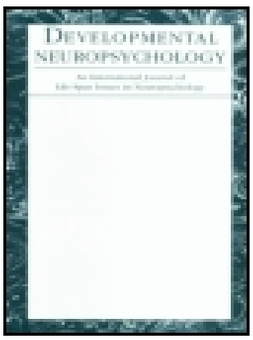

Developmental Neuropsychology

ISSN: 8756-5641 (Print) 1532-6942 (Online) Journal homepage: http://www.tandfonline.com/loi/hdvn20

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To cite this article: Dana C. Torpey , Greg Hajcak & Daniel N. Klein (2009) An Examination of Error-Related Brain Activity and Its Modulation by Error Value in Young Children, Developmental Neuropsychology, 34:6, 749-761, DOI: 10.1080/87565640903265103

To link to this article: http://dx.doi.org/10.1080/87565640903265103

DEVELOPMENTAL NEUROPSYCHOLOGY, *34*(6), 749–761

ISSN: 8756-5641 print / 1532-6942 online
DOI: 10.1080/87565640903265103

Psychology Press
Taylor & Francis Group

An Examination of Error-Related Brain Activity and Its Modulation by Error Value in Young Children

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The error-related negativity (ERN) is an event-related brain potential observed in adults when errors are committed, and which appears to be sensitive to error value. Recent work suggests that the ERN can also be elicited in relatively young children using simple tasks and that ERN amplitude might be sensitive to error value. The current study employed a Go No-Go paradigm in which 5–7-year-old children ($N = 18$) earned low or high points for correct responses. Results indicated that errors were associated with an ERN; however, the size was not reliably moderated by error value.

Response monitoring involves the ability to detect errors and subsequently adjust behavior (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). Studies that have examined the response-locked event-related potential (ERP) have identified a component now referred to as the error-related negativity (ERN; Gehring, Coles, Meyer, & Donchin, 1990) or negativity error (NE; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991) associated with error detection. The ERN is a negative deflection with a fronto-central maximum that peaks approximately 50 msec following an erroneous response (Falkenstein et al., 1991; Falkenstein et al., 2000; Gehring et al., 1990; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Holroyd & Coles, 2002). ERP (Dehaene, Posner, & Tucker, 1994; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003; Mathewson, Dywan, & Segalowitz, 2005; van Veen & Carter, 2002), functional magnetic resonance imaging (fMRI) (Kiehl, Liddle, & Hopfinger, 2000; Mathalon, Whitfield, & Ford, 2003; Menon, Adleman, White, Glover, & Reiss, 2001), and intracerebral (Brázdil, Roman, Daniel, & Rektor, 2005; Brázdil et al., 2002) studies suggest that the ERN is generated in the medial frontal cortex, specifically the anterior cingulate cortex (ACC).

Although original conceptualizations of the ERN emphasized its role in conflict and error detection, findings that this component is moderated by affective and motivational influences suggest a more complex function. Specifically, the ERN may reflect a more affective or motivationally salient evaluation of errors (Gehring, & Willoughby, 2002; Hajcak, McDonald, & Simons, 2004; Luu, Collins, & Tucker, 2000; Luu & Tucker, 2004; Luu et al., 2003; Pailing & Segalowitz, 2004). Several studies have reported an enhanced ERN in populations in which error commission would be likely associated with increased negative evaluation of performance com-

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Funding for this study was provided by NIMH Grant RO1 MH 069942 and NIH GCRC Grant #MO1RR10710.

pared to controls. For example, the amplitude of the ERN is larger in adults with both subclinical levels of obsessive-compulsive symptoms (Hajcak & Simons, 2002) and those with obsessive-compulsive disorder (Gehring, Himle, & Nisenson, 2000; Johannes et al., 2001; Ruchsow et al., 2005; although see Nieuwenhuis, Nielen, Mol, Hajcak, & Veltman, 2005). Additionally, the ERN is larger in adults who endorse high levels of worry (Hajcak, McDonald, & Simons, 2003), negative affect (Hajcak, McDonald et al., 2004; Luu et al., 2000), and in individuals with major depressive disorder (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008).

In addition to this work demonstrating that the ERN is moderated by individual differences in the negative affectivity associated with error commission, support for the hypothesis that the ERN plays a role in the affective or motivationally salient evaluation of an erroneous response can be found in studies that have manipulated the value of the error itself. For example, the ERN is larger when errors are made in conditions that emphasize accuracy rather than speed (Gehring et al., 1993). Pailing and Segalowitz (2004) manipulated the size of the monetary reward and found that, although there was no main effect for error value, there were significant interactions between error value and personality variables. Specifically, the amplitude of the ERN in individuals who were either low in conscientiousness or high in negative affectivity was greater for errors that were more motivationally salient (i.e., more valuable). Hajcak, Moser, Yeung, and Simons (2005) extended these findings by using two simpler manipulations of error value: in Study 1, some errors were more valuable than others; in Study 2, some errors were committed in the presence of an experimenter evaluating the participant's performance. In both studies, the ERN was modulated by motivational salience, providing further evidence that the ERN is sensitive to the significance of the error committed (cf., Hajcak & Foti, 2008).

Although there has been a significant amount of research examining the ERN in adults, far fewer studies have examined the ERN in young children, and the results of that work have been variable. One of the first studies to examine the ERN in children found that it is not as reliably elicited before age 12 (Davies, Segalowitz, & Gavin, 2004). However, Santesso, Segalowitz, and Schmidt (2006) reported an ERN in 10-year-old children, and Wiersema, van der Meere, and Roeyers (2007) showed that the ERN could be reliably elicited in children as young as 7–8 years old, although the amplitude was smaller for children compared to adults in both studies. Further, Kim, Iwaki, Imashioya, Uno, and Fugita (2007) demonstrated that although the amplitude of the ERN elicited in 7–8-year-olds was smaller than that found in 9–11- year-old children, neither group significantly differed from young adults on ERN amplitude.

The notion that the ERN in younger participants might be reduced has been thought to reflect the structural and functional developmental trajectory of the ACC itself. The ACC and its connections to the prefrontal cortex continue to develop through adolescence (Cunningham, Bharracharyya, & Benes, 2002). Additionally, activation of the ACC increases from childhood into young adulthood (Adleman et al., 2002; Van Bogaert, Wikler, Damhaut, Szliwowski, & Goldman, 1998). It is possible that the later maturation of the ACC may be associated with developmental differences in the ERN.

Alternatively, the disparate findings in children could be related to the difficulty of the paradigms used across studies. After all, even very young children are often well-aware when they make mistakes. Results obtained by Hogan, Vargha-Khadem, Kirkham, and Baldeweg (2005) suggest that task complexity influences the amplitude of the ERN in youth: they found that adolescents, but not adults, demonstrated a smaller ERN when the task was more complex. In fact, the ERNs elicited during the complex version of their task (Hogan et al., 2005) are similar to those

shown for 7-year-olds in the Davies et al. paper (2004). Importantly, Davies et al. (2004), who demonstrated that there was substantial variability in the ERN elicited by children under 12 years old, used a flanker paradigm, whereas Wiersema et al. (2007) and Kim et al. (2007), who obtained reliable ERNs in 7–8-year-olds, used simpler Go No-Go designs. Relatedly, Santesso and Segalowitz (2008) found differences between 15- and 18-year-old adolescents at both the behavioral and neural level under conditions of increased task demands, whereas differences were only evident on neural measures for simple response tasks.

As in adults, there is evidence that the ERN in children is moderated by the motivational significance of errors. Specifically, the amplitude of the ERN has been found to be larger in a non-clinical population of children with high levels of obsessive-compulsive symptoms (Santesso et al., 2006) and in clinically anxious children (Hajcak, Franklin, Foa, & Simons, 2008; Ladouceur, Dahl, Birmaher, Axelson, & Ryan, 2006). However, fewer studies have been conducted to examine the impact of error value on the ERN in children. Kim, Iwaki, Imashioya, Uno, and Fujita (2005) reported larger ERNs in children when they were being observed by a friend compared to when they were completing the task alone, providing preliminary evidence that the ERN in children is moderated by motivational influences. Because of its association with psychopathology, the ability to measure and manipulate the ERN in young children might provide a window onto risk factors that precede the development of psychiatric disorders.

Response-locked ERP studies have isolated a second component associated with response monitoring: a large positivity known as the Error Positivity (Pe), that appears within 200–500 msec following an erroneous response (Falkenstein et al., 2000; Santesso et al., 2006). A review by Overbeek, Nieuwenhuis, and Ridderinkhof (2005) concluded that although the exact function of the Pe is uncertain, its purpose appears to be independent of the ERN. Specifically, some source localization studies have demonstrated that the Pe is localized in more posterior regions than the ERN (Burgio-Murphy et al., 2007; Ullsperger & von Cramon, 2006) and there is evidence that it is differentially affected by a number of factors, such as awareness of error commission (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001), task demands (Mathewson et al., 2005), degree of response conflict, and affective salience of stimuli (Simon-Thomas & Knight, 2005).

There is also evidence that the Pe does not change over development at more posterior sites (Davies et al., 2004; Wiersema et al., 2007); however, Santesso et al. (2006) found that adults demonstrated a larger Pe than children at anterior sites. The Pe in children does not appear to be influenced by the same factors as the ERN. For example, Ladouceur et al. (2006) and Hajcak et al. (2008) found no difference in the amplitude of the Pe in anxious and non-anxious children. There is mixed evidence regarding whether or not the Pe is specifically affected by the motivational significance of errors. Santesso et al. (2006) found that children high in obsessive-compulsive behaviors had a larger Pe compared to children low in these behaviors; however, children high and low in socialization did not demonstrate differences in this component (Santesso, Segalowitz, & Schmidt, 2005).

THE PRESENT STUDY

The results from these studies suggest the need for a thorough examination of ERPs related to response monitoring in younger children. Specifically, the findings by Kim et al. (2007) and Wiersema et al. (2007) indicate that if the paradigm used is simple enough, an ERN might be dem-

onstrated in children even younger than 7 years old. One of the primary goals of the present study was to determine whether an ERN could be elicited in 5–7-year-old children using a simple Go No-Go paradigm. Additionally, a second goal of the current work is to examine the effect of motivational salience on the ERN and Pe in this young population by manipulating the value of errors.

METHODS

Participants

Eighteen children between the ages of 5 and 7 years (mean age = 6.31, *SD* = .60) were recruited for this study. Six of the 18 participants were siblings of children who were participating in a separate study examining child temperament. Those children had been recruited through a commercial mailing list and were initially contacted by the Stony Brook University Center for Survey Research. The remaining participants were recruited via advertisements and word-of-mouth. None of the children had significant medical conditions or developmental disabilities, as indicated by parent report. Fifteen of the children were Caucasian (83.33%), one was African American (5.56%), one was Asian (5.56%), and one was both Caucasian and Hispanic (5.56%). Eleven of the participants were female (61.11%). The parents of all participants were paid $20.00 and both the parents and the children were told that the child could earn an additional $5.00 based on their performance. All children ultimately received this bonus. Data from one participant were excluded due to near-perfect task performance in the high-value condition. Data from a second subject were excluded due to technical difficulties; thus the analyses include the data from 16 children.

Task

A Go No-Go paradigm adapted from that described in Kim et al. (2007) was administered using Presentation software (Neurobehavioral Systems, Inc.). The stimuli were green equilateral triangles in four different orientations presented at a 1.54º visual angle. There were a total of 480 trials, which were divided into 8 blocks of 60 trials each. In each block, 60% of the triangles were vertically aligned and pointed up, 20% were vertically aligned and pointed down, 10% were tilted slightly to the left, and 10% were tilted slightly to the right. All stimuli were presented on a black background.

Each trial started with the presentation of one of the four triangles for 1,200 msec in the middle of the monitor. Following this, a small white fixation cross was displayed in the middle of the monitor for 300–800 msec before the next trial commenced with the presentation of a new triangle. At the end of each block, the number of points won by the participant was displayed in white numbers.

Procedure

A series of practice blocks were administered to ensure that the participant understood the various aspects of the task. First, each of the stimuli was presented on a card to the child. Participants were instructed to press a button with their thumb only when the vertically aligned upward-pointing tri-

angle was displayed (Go stimulus) and not to respond when the other three types of triangles (No-Go stimuli) or the fixation cross were presented. Participants were then presented with 8 triangles, (2 Go stimuli, 6 No-Go stimuli), and were given as much time as necessary to decide whether or not to press the button.

The next practice block contained 20 trials. In addition to the triangles and fixation cross, participants also received feedback after each trial consisting of a "thumbs-up" or "thumbs-down" stimulus (1.5 cm^2) presented in the middle of the monitor, indicating whether their performance was correct or incorrect on the preceding trial. In addition to helping the participants learn to differentially respond to the stimuli, the feedback stimulus also emphasized the importance of speedy response: if participants did not respond to Go stimuli within 1,300 msec, the thumbs-down feedback was presented.

The final practice block of 30 trials was identical to the task, as described earlier; however, there was no feedback to indicate whether the participant's responses had been correct or incorrect on a trial-by-trial basis. Following completion of this practice block, the children were told that the actual game was going to begin and that there would be some blocks for which they would earn one point (low-value) and some blocks for which they would earn ten points (high-value) for correct responses on Go trials and for withholding responses on No-Go trials. They were told that if they earned enough points, they would win up to $5.00. These blocks were alternated, such that each participant started with a low-value block and ended with a high- value block. Both speed and accuracy of response was emphasized to the children. Between each block, the experimenter told the participants how many points they had earned, reminded the children of the task instructions, and informed the children of the value of the next block. Additionally, the importance of response speed and accuracy were re-emphasized before each block commenced. Following completion of the task, all children were told they won the maximum number of points and given $5.00.

Psychophysiological Recording

Data were acquired using the Active Two system (Biosemi, Amsterdam, Netherlands). A stretch Lycra cap was placed on the child's head and 32 Ag/AgCl-tipped electrodes arranged according to the 10/20 international labeling system (American Electroencephalographic Society, 1994) were attached to the cap. A small amount of electrolyte (Signa Gel; Bio-Medical Instruments Inc., Warren, Michigan) was applied to the child's scalp at each electrode position. Electrode offsets were between ±20 units; when necessary, the scalp was slightly abraded using a plastic syringe tip to reduce impedance. Additionally, flat electrodes were placed at supra and infra orbital sites of the right eye to monitor vertical eye movements and on the outer canthi of the left and right eyes to monitor horizontal eye movements; an electrode was also placed on the tip of the nose. All data were sampled at 512 Hz. Per BioSemi's design, the ground electrode during acquisition was formed by the common mode sense active electrode and the driven right leg passive electrode.

Offline, all data processing was performed with Brain Vision Analyzer (Brain Products, Gilching, Germany). EEG data were re-referenced to the nose, and high- and low-pass filtered at 2 Hz and 20 Hz, respectively. From the continuous EEG, 1,200 msec segments were extracted beginning 500 msec prior to correct and erroneous responses. ERP data were corrected for blinks and eye-movements using the method developed by Gratton, Coles, and Donchin (1983). Additional artifacts were rejected when any of the following criteria were met: a voltage step of more than 50 μV between data points, a

voltage difference of 300 μV within a single trial, or a voltage difference of less than .5 μV within 100-msec intervals. This last criterion refers to the rejection of a channel if there was no activity in it. ERP averages were then created separately for each trial type (low-value correct, high-value correct, low-value error, and high-value error) and were baseline corrected by subtracting from each data point the average activity in a 500- to 300-msec window prior to the response. Each trial was also examined for artifacts manually following the semi-automated procedure. Trials were not included in ERP averages if the reaction time occurred outside of a 200–1,300 msec window.

The ERN was defined as the average voltage in the window from 100 msec before to 100 msec after errors, and the Pe was evaluated as the average voltage in the window 200 msec to 500 msec following errors; both the ERN and Pe were compared to correct trial activity in the same windows (i.e., –100 to 100 msec relative to the response for the ERN, and 200 to 500 msec following the response for the Pe). To examine possible stimulus-locked differences between high- and low-value trials, the P300 was also measured on correct Go trials, and was defined as the average voltage in the window 400 msec to 600 msec following stimulus onset. All ERP components were evaluated along the midline (i.e., Fz, Cz, and Pz). Behavioral measures were analyzed using repeated measures analysis of variance (ANOVA) and all ERP components were statistically evaluated using repeated measures ANOVA with the Greenhouse-Geisser epsilon correction (Jennings & Wood, 1976) applied to *p* values to counteract heterogeneity of variance-covariance matrices associated with repeated measures.

RESULTS

Behavioral Measures

Table 1 presents reaction times (RT) and accuracy data for high- and low-value trials. Both the number of errors and percentage of correct trials are presented because they provide different information. The number of errors refers only to errors of commission, whereas the percentage of correct trials accounts for both correct responses to Go stimuli and correct rejections of No-Go stimuli. The children did not differ in the total number of low- and high-value errors, $F(1, 15) < 1, p > .96$, nor did they differ in overall accuracy, $F(1, 15) = .14, p > .71$. All participants made at least 10 errors overall across the value conditions; however two subjects made only 4 errors in either the high- or low-value condition. The data were analyzed both including and excluding these two subjects and results were identical; therefore, all analyses described include the data from these two participants.

A 2 (Trial Type: Correct and Error) × 2 (Trial Value: High and Low) ANOVA conducted to examine the RT data indicated that participants responded faster on error trials than on correct trials, $F(1, 15) = 20.08, p < .001$. There was no main effect of Trial Value, $F(1 ,15) = .45, p > .51$, thus reaction times were not faster on high-value trials compared to low-value trials. The interaction between Trial Type and Trial Value also was not significant, $F(1, 15) = .18, p > .67$, suggesting that reactions times on correct and erroneous trials were not differentially impacted by trial value.

Response-Locked ERPs

Figure 1 presents the response-locked ERPs at Fz, Cz, and Pz following error and correct responses on both high- and low-value trials. The difference waveform (i.e., error minus correct ERPs) is also displayed—and the ERN is evident at all midline recording sites around the time of response as a negative deflection on error relative to correct trials. The average ERP values for

TABLE 1
Mean Reaction Times and Accuracy Measures (and Standard Deviations)

	Low Value Trials	*High Value Trials*
Number of errors	11.38 (5.43)	11.44 (4.75)
Accuracy (% correct)	88.80 (.07)	89.04 (.07)
Error Reaction Time (ms)	524.33 (124.95)	511.48 (110.18)
Correct Reaction Time (ms)	621.40 (77.04)	618.33 (77.64)
Number of errors included in ERN	11.38 (5.43)	11.44 (4.75)
Number of correct trials included in Correct trials average	131.75 (15.55)	132.38 (18.76)

ERN = error-related negativity.

correct and error trials at the Fz, Cz, and Pz sites for both low- and high-value trials are presented in Table 2. Consistent with the impression from these data, a 3 (Electrode Site: Fz, Cz, and Pz) × 2 (Trial Type) × 2 (Trial Value) ANOVA confirmed that both error and correct trial averages were more negative at the anterior site, $F(2, 30) = 9.33$, $p < .01$. Consistent with the presence of the ERN on error trials, errors were associated with a greater negativity than correct trials, $F(1, 15) = 101.16$, $p < .001$. These data are consistent with the impression from Figure 2, which depicts the scalp distribution of error-related brain activity in the time-range of the ERN, collapsing across high- and low-value trials. Collectively, these data confirm the presence of an anterior maximal ERN that did not vary as a function of error value. Moreover, consistent with the impression from Figure 1, the difference between error and correct trials varied as a function of Electrode Site, $F(2, 30) = 14.79$, $p < .001$. Post-hoc paired sample *t*-tests indicated that the ERN was more negative than the correct trial average at all three electrode sites ($t(1,15) = 10.77$, $p < .001$ at Fz; $t(1,15) = 10.19$, $p < .001$ at Cz; $t(1,15) = 8.20$, $p < .001$ at Pz), although the difference between the error and correct trials was larger at both Pz and Cz compared to Fz, ($t(1,15) = –4.18$, $p = .001$, $t(1,15) = –5.65$, $p < .001$, respectively), but was not significantly larger at Pz compared to Cz ($t(1,15) = –1.35$, $p > .20$). Important to the purpose of the present study, trial value did not have an overall effect on error and correct trials ERP amplitudes, $F(1,15) = .31$, $p > .59$, and other 2- and 3-way interactions did not reach significance (all p values > .14). The data were also analyzed using the peak detection method, rather than the area –100 to +100 msec around the response. Results were identical, except that there was no longer a significant main effect of Electrode Site.

The Pe is evident in Figure 1 as a positive deflection that is maximal approximately 400 msec following errors at central and parietal recording sites. The average Pe values at the Fz, Cz, and Pz sites for low- and high-value correct and error trials are presented in Table 2. Consistent with the impression from Table 2, a 3 (Electrode Site) × 2 (Trial Type) × 2 (Trial Value) ANOVA for error trials confirmed that the Pe was more positive on error than correct trials, $F(1, 15) = 19.22$, $p = .01$, and was more positive at central and posterior sites, $F(2, 30) = 33.46$, $p < .01$. The scalp distribution of error-related brain activity in the time window of the Pe, collapsing across trial value, is presented in Figure 2. There was a trend toward a significant interaction between electrode site and trial type, $F(2, 30) = 3.29$, $p > .10$, indicating that the difference between the Pe and the correct trial positivity tended to vary across electrode sites. Importantly, trial value did not affect the amplitude of the Pe, $F(1, 15) = .01$, $p > .90$, nor were there any 2- or 3-way interactions involving trial value that reached significance (all p values > .18). These results suggest that the amplitude of the Pe, like the ERN, is not affected by the motivational significance of errors in young children.

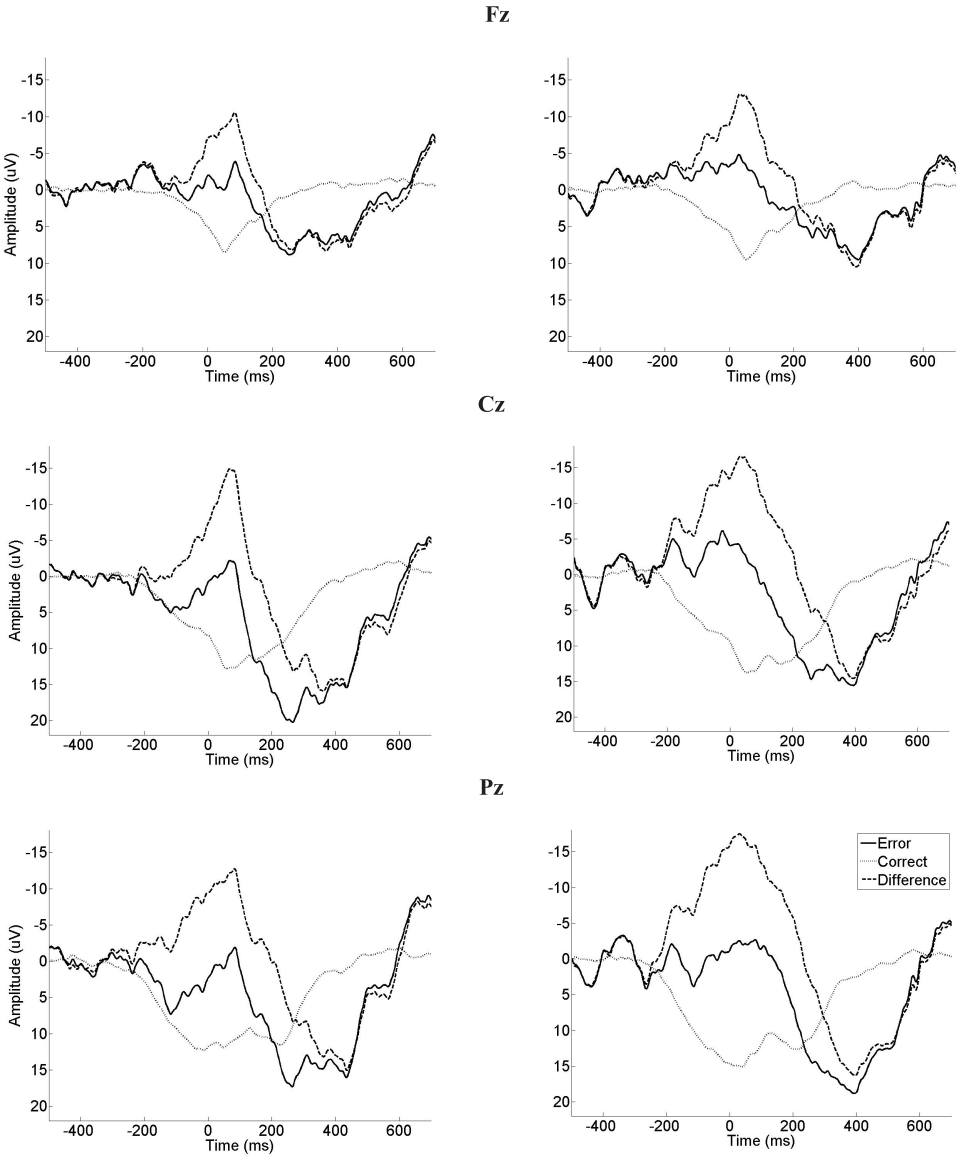

FIGURE 1 Response-locked event related potentials (ERPs) for error (solid line), correct (dotted line), and error minus correct (dashed line) low- (left) and high- (right) value trials at Fz (top), Cz (middle), and Pz (bottom) sites.

Stimulus-Locked ERPs

An evaluation of the stimulus-locked P300 for both high- and low-value trials was conducted to examine whether value influenced stimulus-locked ERPs. A 3 (Electrode Site) × 2 (Trial Value) ANOVA confirmed that the P300 was larger at more parietal sites ($F(2, 30) = 29.64$, $p < .001$).

TABLE 2
Mean (*SD*) of ERN and Pe Amplitude (μV) in Error Trials and Amplitude (μV) in Correct Trials at Midline Sites

		Trial Value	
ERP Component	*Electrode Site*	*Low*	*High*
ERN	Fz	–.68 (6.97)	–2.68 (7.42)
	Cz	.87 (8.82)	–3.52 (8.63)
	Pz	1.25 (7.28)	–1.74 (8.62)
Correct Trials Averaged	Fz	4.62 (3.88)	5.91 (4.73)
	Cz	8.38 (5.69)	9.50 (5.74)
	Pz	10.17 (5.96)	12.10 (6.02)
Pe (Error Trials)	Fz	6.44 (1.28)	6.00 (2.41)
	Cz	15.01 (1.89)	11.73 (2.40)
	Pz	13.54 (1.78)	14.00 (3.05)
Correct Trial Positivity	Fz	–.44 (1.12)	.43 (1.19)
	Cz	2.99 (1.11)	3.80 (1.57)
	Pz	3.85 (1.51)	4.94 (1.85)
P300 (Correct Go-Trials)	Fz	2.41 (3.09)	2.85 (4.78)
	Cz	4.93 (4.22)	5.06 (5.36)
	Pz	7.90 (4.79)	8.46 (5.77)

ERN = error-related negativity; ERP = event related potentials.

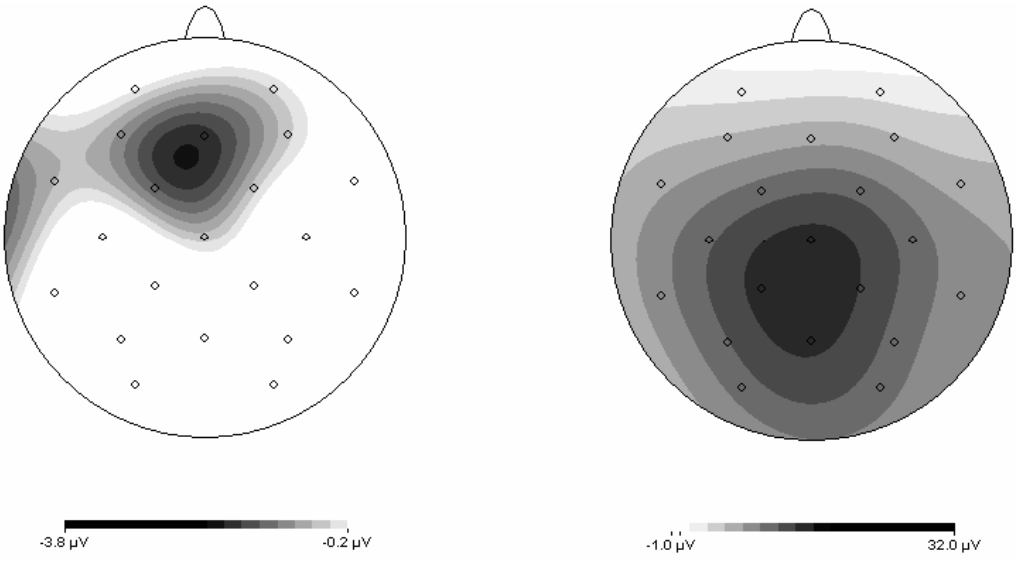

FIGURE 2 Scalp distribution of error-related brain activity in the time window of the error-related negativity (ERN) (left) and Pe (right). Note that darker colors indicate more negativity in the left part of the figure depicting the ERN scalp distribution, but indicate more positivity in the right part of the figure depicting the Pe scalp distribution.

Post-hoc paired *t*-tests revealed that the P300 was larger at Pz than at Cz and Fz ($F(1, 15) = 5.97$, $p < .001$ and $F(1,15) = 5.86$, $p < .001$, respectively) and was larger at Cz than at Fz ($F(1, 15) = 3.81$, $p < .01$). There was no effect of Trial Value on the P300, $F(1, 15) = .13$, $p > .70$, nor was there an interaction between Electrode Site and Trial Value, $F(2, 30) = .54$, $p > .52$.

DISCUSSION

This study demonstrated that an ERN can be reliably elicited in children as young as 5–7 years of age. The present study also found that, although the ERN was numerically larger on high- than low-value trials, this difference did not approach significance. Thus, errors of greater value were not associated with a relatively more negative ERN than errors of lesser value. Further, the error value manipulation was not associated with any systematic effects on the subjects' performance. Specifically, participants were equally accurate on both high- and low-value errors and the reaction times did not differ between high- and low-value trials for either correct or error trials. Although the behavioral results are identical to those obtained in adults using a comparable trial-by-trial manipulation of error value (Hajcak et al., 2005), the finding that ERN amplitude was not associated with motivational significance contrasts with that demonstrated in adults by Hajcak and colleagues (2005), in which more valuable errors were associated with a relatively more negative ERN in adults.

The results of the current study are in contrast to those found by Kim et al. (2005) in which the ERN was enhanced when a friend was present. Although the children in that study were slightly older than the participants in this sample, the Kim et al. data may suggest that the social evaluation of performance might be a more salient value manipulation in young children. This discrepancy could be due to the abstract manipulation of error value in the present study: associating errors worth more or less points may not have been a sufficient motivator to elicit changes in the ERN. Alternatively, children may have been extremely motivated on both high- and low-value trials. This situation would be similar to those results found by Pailing and Segalowitz (2004) in which they reported that those high on conscientiousness may have been motivated to do well in each condition. Future work is necessary to clarify this issue.

The present results suggest that moderators of the ERN may change over the course of development; thus, it is crucial to examine in younger children other variables for which associations have been found with ERN amplitude in older children and in adults. Specifically, there are a number of personality and temperament variables, such as degree of socialization (Dikman & Allen, 2000; Santesso et al., 2005), conscientiousness (Pailing & Segalowitz, 2004), and impulsiveness (Ruchsow, Spitzer, Grön, Grothe, & Kiefer, 2005, although see Luu, Collins, & Tucker, 2000) for which such associations have been demonstrated. Unfortunately, personality and temperament variables were not assessed in the current study. However, this should be explored in future studies.

Like the ERN, the amplitude of the Pe was not significantly different for high- and low-value trials; however, the implications of the current results are unclear and require further examination. By comparing these results from young children with other studies that have examined adults, this work suggests that the ERN and Pe may be relatively similar topographically over the course of development. Specifically, the ERN was maximal over the frontal electrode whereas the Pe was maximal over central-parietal sites—both consistent with many of the findings in the adult

(Burgio-Murphy et al., 2007; Ullsperger & von Cramon, 2006) and developmental literatures (Davies, et al., 2004; Wiersema et al., 2007). Also, the latencies of the ERN and Pe in this group of young children were similar to what has been reported in adults. Specifically, the ERN peaked just after response execution and the Pe was maximal approximately 300 to 400 msec later. The ERN latency is consistent with many of the findings that have been demonstrated in adults (Falkenstein et al., 1991; Falkenstein et al., 2000; Gehring et al., 1990; Gehring et al., 1993) and children (Davies et al., 2004; Santesso & Segalowitz, 2008; Wiersema et al.,2007). The Pe latency in the current sample of young children is identical to what has been reported in both adults (Falkenstein et al., 2000) and older children (Santesso et al., 2006).

In conclusion, this study demonstrated that both an ERN and Pe can be elicited from 5–7-year-old children and that both the scalp distribution and latency of the ERN and the Pe are similar to what has been found in adults. These data suggest that the ERN can be reliably observed at a younger age than has been reported in previous studies (Davies et al., 2004; Kim et al., 2007; Wiersema et al., 2007). Further, the current work indicates that the amplitude of the ERN is not moderated by error value. Although there is evidence that the ERN is enhanced when error value is manipulated in adults (Hajcak et al., 2005), this type of value manipulation was not associated with ERN amplitude in this sample of young children, suggesting potential changes over development in the factors to which the ERN is sensitive. Additional work focused on examining other types of error manipulations and personality variables is necessary in order to clarify the nature of these changes.

ACKNOWLEDGMENTS

Thanks to Jiyon Kim and Flannery Murphy for their assistance in data collection.

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