DEVELOPMENTAL NEUROPSYCHOLOGY, *25*(3), 355–376

Development of Response-Monitoring ERPs in 7- to 25-Year-Olds

Patricia L. Davies
Department of Occupational Therapy
Colorado State University

Sidney J. Segalowitz
Department of Psychology
Brock University
St. Catharines, Ontario, Canada

William J. Gavin
Department of Speech, Language, and Hearing Sciences
University of Colorado, Boulder

In a target discrimination task, trials with incorrect responses elicit event-related potentials (ERPs) that include an error-related negativity (ERN or Ne) and a later error-positivity (Pe). Substantial evidence points to the anterior cingulate cortex as the source generator of the ERN. We examined the development of ERP component morphology, amplitude and latency to processing of correct and incorrect responses in 124 children, 7 to 18 years of age, and 27 adults, 19 through 25 years of age. The ERN and Pe were recorded during a standard 480-trial visual flanker task. As expected, response times decreased significantly with age. The ERN amplitude in error trials increased with age, although this was qualified by a nonlinear change as well. The Pe amplitude did not change with age. In correct trials, most participants produced a small negativity corresponding to the timing of the ERN in error trials. This correct-response negativity (CRN) amplitude was larger in children than in adults. Results are discussed with respect to continued maturation of the anterior cingulate cortex and prefrontal cortex into young adulthood.

Requests for reprints should be sent to Patricia L. Davies, Department of Occupational Therapy, Colorado State University, 219 Occupational Therapy, Fort Collins, CO 80523. E-mail: pdavies@lamar.colostate.edu

The error-related negativity (ERN or Ne) is a component of the event-related potential (ERP) that is associated with acknowledged incorrect responses that occur in target discrimination tasks (Dehaene, Posner, & Tucker, 1994; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996). The error waveform is time-locked to the behavioral response, as opposed to stimulus onset, and consists of a negative deflection (the ERN) appearing 50 to 150 msec after the execution of the incorrect response, followed by a positive deflection (Pe) peaking 200 to 500 msec after the ERN. The ERN has a fronto-central maximum (Davies, Segalowitz, Dywan, & Pailing, 2001; Dehaene et al., 1994; Eriksen & Eriksen, 1974; Falkenstein et al., 1991; Gehring et al., 1993) and has been associated with the anterior cingulate cortex (ACC) in studies using a variety of technologies: Brain Electric Source Analysis studies point to the ACC as the likely source generator of the ERN (Dehaene, 1996; Dehaene et al., 1994; Dikman & Allen, 2000; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003; Miltner, Braum, & Coles, 1997; Van Veen & Carter, 2002); in functional magnetic resonance imaging studies, the ACC is activated in tasks that elicit error responses (Carter, Braver, Barch, Botvinick, Noll, & Cohen, 1998; Kiehl, Liddle, & Hopfinger, 2002; Menon, Adleman, White, Glover, & Reiss, 2001; Van Veen & Carter, 2002); in addition, damage to the ACC region abolishes the ERN response (Stemmer, Segalowitz, Witzke, & Schönle, 2003).

The ACC is considered one of the largest components of the limbic system, and is a transitional cortex with extensive connections with the prefrontal cortex (Van Hoesen, Morecraft, & Vogt, 1993; Vogt, 1993). The ACC is involved in executive functions and becomes activated during behaviors such as response selection, goal-directed behavior, selective attention, and language generation; and holds an important place in the information throughput to and from virtually all segments of the prefrontal cortex (Devinsky, Morrell, & Vogt, 1995; Van Hoesen et al., 1993). Recent evidence supports anatomical and physiological (Cunningham, Bhattacharyya, & Benes, 2002) maturation of the ACC into early adulthood, as well as increased activation of the ACC from childhood to young adulthood (Adleman et al., 2002; Van Bogaert, Wikler, Damhaut, Szliwowski, & Goldman, 1998). This parallels the development of the prefrontal cortex (PFC) with anatomical (Greenough, Black, & Wallace, 1987), physiological (Rakic, Bourgeois, Eckenhoff, Zecevic, & Goldman-Rakic, 1986), behavioral (Davies & Rose, 1999; Stuss, 1992), clinical (Kolb, Wilson, & Taylor, 1992), and electrophysiological (Segalowitz, Unsal, & Dywan, 1992) evidence for a relatively late maturation of the PFC in general. Both the PFC and ACC mediate executive functions that develop into young adulthood (Devinsky et al., 1995). Furthermore, increased dopaminergic connections and metabolism have been observed in these same brain regions into early adulthood (Benes, Vincent, Molloy, & Khan, 1996; Kalsbeek, Voorn, Buijs, Pool, & Uylings, 1988; Lambe, Krimer, & Goldman-Rakic, 2000; Rosenberg & Lewis, 1995; Verney, Berger, Adrien, Vigny, & Gay, 1982). Holroyd and Coles (2002) recently postulated

that the mesencephalic dopamine system is involved in the generation of the ERN. Given this model and the continued maturation of the ACC, PFC, and domaminergic systems into young adulthood, the production of the ERN may also show evidence of continued maturation through adolescence.

In general, there are various ERP components that have shown age-related changes. The ERP components that have received the most attention in the developmental literature are those elicited by the standard oddball and novelty oddball paradigms, i.e., N1, P2, N2, P3b and the novelty P3 (Courchesne, 1977, 1978, 1983; Cycowicz, Friedman, & Rothstein, 1996; Johnstone, Barry, Anderson, & Coyle, 1996). The contingent negative variation has also been studied in children (Sartory, Besken, & Pothmann, 1997; Segalowitz et al., 1992; Timsit-Berthier & Hausmann, 1972; Warren & Karrer, 1984). However, to our knowledge there have not been any reports on the development of the ERN.

The overall aim of this study was to investigate the development of component morphology, amplitude, and latency to processing of correct and incorrect (error) responses in a standard paradigm used to elicit ERN and Pe. A further aim of the study was to compare age-related changes in the ERPs to behavioral responses of accuracy and reaction times.

METHOD

Participants

One hundred and twenty-four children and adolescents, 7 through 18 years of age, and 27 adults, 19 through 25 years of age (see Table 1), were recruited from the local community as part of a larger normative study. All participants completed two sessions, a session of behavioral testing including neuropsychological measures requiring about 1½ hours (to be reported elsewhere), and a session of EEG data collection that included 5 separate tasks requiring about 2 hours. The majority of the children were contacted through presentations before youth organizations and school classes, during which prospective participants or their parents were invited to write down their name and telephone number if they wished to volunteer. Project staff called to provide additional information about the study and schedule their visits to the lab. Each participant in the study was free from neurological disorders, based on parent report for the children and self-report for the adult participants. Written consent was obtained from the adult participants and from the parents of the children. All children signed assent forms prior to participating in the study.

Electrophysiological Paradigm

The ERN was elicited during a visual flanker task (Eriksen & Eriksen, 1974) that consisted of computer-generated displays, each containing a five-letter array. The

TABLE 1
Number of Participants in Each Age Group
Partitioned by Gender

	Gender		
Age	*Women*	*Men*	*Total*
7	4	8	12
8	8	2	10
9	12	6	18
10	4	6	10
11	7	4	11
12	8	10	18
13	5	3	8
14	6	3	9
15	5	3	8
16	4	2	6
17	3	3	6
18	5	3	8
Adults	18	9	27
Total	89	62	151

paradigm consisted of a total of 480 trials presented in two series of 240 trials each separated by about 15 min, during which time the participant completed a different ERP paradigm. One of four arrays appeared on each trial and the participant's task was to press a key corresponding to the central letter, either an S or an H. There were two congruent arrays, HHHHH and SSSSS (80 trials each) and two incongruent arrays, SSHSS and HHSHH (160 trials each). Faster responses are typically recorded when the letters flanking the center are congruent than when they are incongruent, and incongruent trials are also more likely to elicit errors (Eriksen & Eriksen, 1974). Right and left hand key presses for S and H targets were counter-balanced across participants. Each array remained on the screen for 250 msec, with a 1100 msec stimulus onset asynchrony (SOA) for participants ages 10 through 25 years. A 1500 msec SOA was used for participants ages 7 through 9 years and for older children if they could not perform at the faster presentation. The stimuli subtended 2.4 degrees vertically and 8.1 degrees horizontally.

Electrophysiological Recording

The electroencephalogram (EEG) was recorded with tin electrodes embedded in a Quik-cap (Neuroscan) using a portable QuickTrace system (Neuroscan) from twenty-nine scalp sites according to the 10–20 system: Fz, FCz, Cz, Pz, Oz, Fp1, Fp2, AF3, AF4, F3, F4, F7, F8, FC1, FC2, FC5, FC6, C3, C4, T7, T8, CP1, CP2, P3, P4, P7, P8, PO3, PO4, with AFz as ground. Data from Fz, FCz, Cz, and Pz

were analyzed for the purpose of this study. Thirty-two of the 120 participants did not have FCz site, due a change in the montage at the beginning of the larger normative data collection. EEG was recorded with left earlobe as reference and right earlobe as an active site, and recordings were re-referenced off-line to an average of the two ears.

Data were sampled at a rate of 500 points per s, with a bandpass of .23–100 Hz and filtered off-line with a low pass of 30Hz. Signals were amplified with a hardware gain of 1000. Two bipolar electrooculograms (EOG) were recorded from tin electrodes placed on the left and right outer canthus for horizontal movements and on the left supraorbital and infraorbital region for vertical movements. Impedances for the EEG and EOG channels were maintained below 5 kOhms. Trials with deviations greater than ±100 μV on the EOG channels were eliminated (i.e., eye blink artifact rejection). When there were fewer than 9 trials within a condition or when the waveform was noisy with more than 9 trials, eye movement artifact was removed from the EEG trials using a regression algorithm that applies weighted values to each channel. A trial-by-trial inspection allowed for trials to be eliminated from the average if the regression did not appear to correct the artifact from the EEG channels (i.e., eye blink artifact regression: Segalowitz, 1996). This regression approach was used on the error trials for 36% of the children and adolescents (ages 7 to 18 years) and on correct trials for only 3% of the children. Children ages 7 and 8 years displayed the most eye movement and blink artifacts, with 72% of children in these two age categories requiring the regression approach. Of children and adolescents ages 9 through 18, only 27% required the regression approach, which is similar to the percent of adult participants (26%) requiring the regression approach.

RESULTS

Error Rates and Response Times for Children and Adults

Error rates ranged from 2.5% to 29.3% across subjects ($M = 11.05\%$). Participants made significantly more errors in incongruent trials ($M = 13.9\%$) than congruent trials ($M = 9.2\%$), $t(1,150) = 12.1$, $p < .0005$. Age significantly correlated with error rate, $r = -.32$, $p < .0005$, with children generally having a larger error rate than adults. If just the data for the 7- to 18-year-olds are analyzed, the strength of the correlation decreases, $r = -.24$, $p = .008$. Error rates were not significantly correlated with correct response time (RT; $r = .01$) or error RT ($r = .02$), compatible with an absence of speed-accuracy tradeoff across participants.

As expected, there were age-group differences in RT with strong zero-order correlations between age and correct ($r = -.75$, $p < .0005$) and error ($r = -.61$, $p < .0005$) RTs, with asymptotes around 12 to 13 years (see Figure 1). Consistent with previous findings, subjects were faster on error trials ($M = 400$ msec) than correct

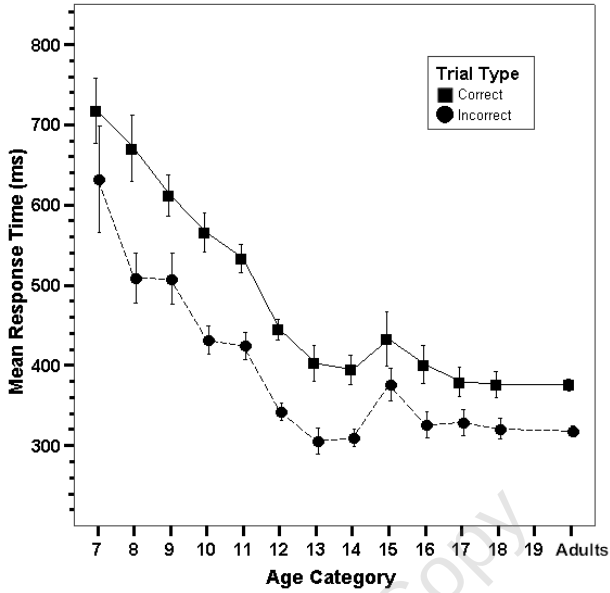

FIGURE 1 Mean response time of each age group for correct and error trials.

trials (M = 489 msec), $F(1, 138) = 331.9$, $p < .0005$. This was true for all age groups, suggesting that the children perform similarly on this task to adults. However, there was a significant interaction between trial type (correct and error trials) and age where the difference between RTs for correct and error trials generally decreased with age, $F(12, 138) = 3.83$, $p < .0005$. This difference between RTs on correct and error trials also decreased with age when we calculated the difference as a residual by regressing the error RTs out from the correct RTs, $r = -.45$, $p < .01$, and therefore the relationship with age was not just a function of the longer response times in the younger children. With respect to post-error slowing, the mean RT for correct trials following error trials (M = 525 msec) was significantly longer than the mean RT for correct trials following correct trials (M = 486 msec), $F(1, 138) = 57.89$, $p < .0005$. This was true for each age group, and the interaction between trial type and age group was not significant. Therefore, even the youngest children slowed on responses immediately following an incorrect response.

Responses were faster for correct trials with congruent flankers (M = 470 msec) than for those with incongruent flankers (M = 500 msec), $F(1, 138) = 156.1$, $p < .0005$, but error trial average RTs for congruent and incongruent trials were similar (402 msec vs. 397 msec, respectively, *ns*). The interaction between congruency and age was not significant in both the correct and error trials suggesting that even the young children exhibited similar congruency effects as the adults. In addition,

the overall waveforms for all errors versus incongruent trial errors were virtually identical. This replicates the finding we reported previously, with no difference in the ERN between incongruent and all errors (Pailing, Segalowitz, Dywan, & Davies, 2002). Thus, for the purpose of ERP component analysis, congruent and incongruent trial errors were combined to maximize the number of trials, and thus maximize the stability of the error waveform.

ERPs for Error Trials in Children and Adults

ERP components for adults were measured relative to an early baseline (600 to 400 msec prior to the response) with the P3 to the stimulus (M = 7.4 μV at Cz) peaking at 17 msec before the response on average, ERN (M = -8.4 μV at Cz) peaking at 81 msec on average following the response, and this being followed by a positive peak, Pe, (M = 12.1 μV at Cz) 288 msec following the response. ERP components for participants 7- to 18-year-olds had a P3 to the stimulus (M = 11.2 μV at Cz) peaking at 31 msec before the response, ERN (M = –0.7 μV at Cz) peaking at 67 msec following the response, and a Pe (M = 17.9 μV at Cz) 248 msec following the response. The grand averages of correct and error trials for adults at 4 midline sites and for each age group at Cz are illustrated in Figures 2 and 3, respectively. Figure

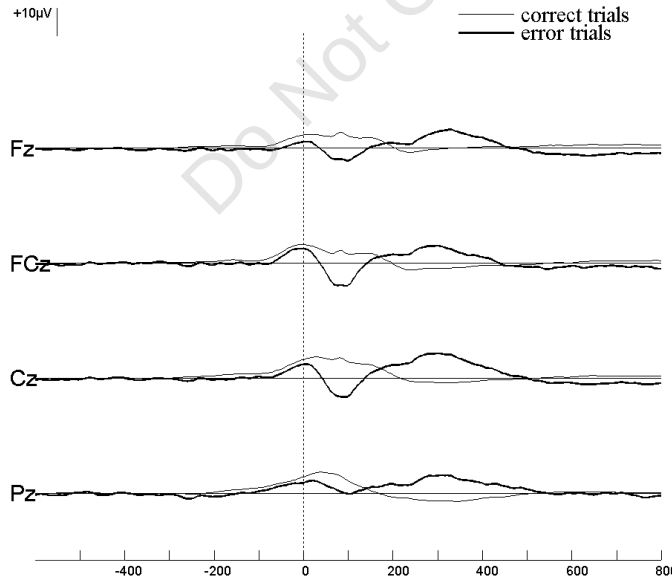

FIGURE 2 Grand average response-locked ERPs for adults (N = 27) at four scalp midline sites for correct trials (thin line) and error trials (thick line). The vertical hashed line represents the timing of the response. ERPs are presented relative to a 200 msec baseline, 600 msec to 400 msec prior to the response.

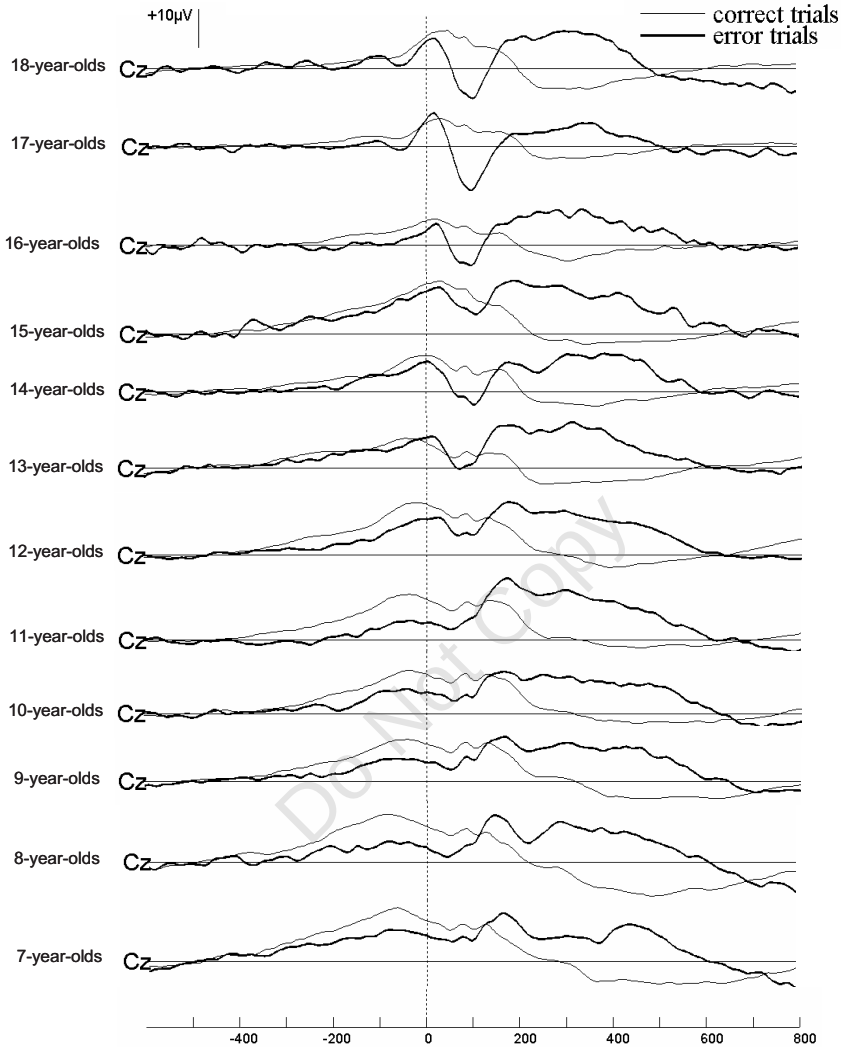

FIGURE 3 Grand average response-locked ERPs for each age group at Cz for correct trials (thin line) and error trials (thick line). The vertical hashed line represents the response. ERPs are presented relative to a 200 msec baseline, 600 msec to 400 msec prior to the response.

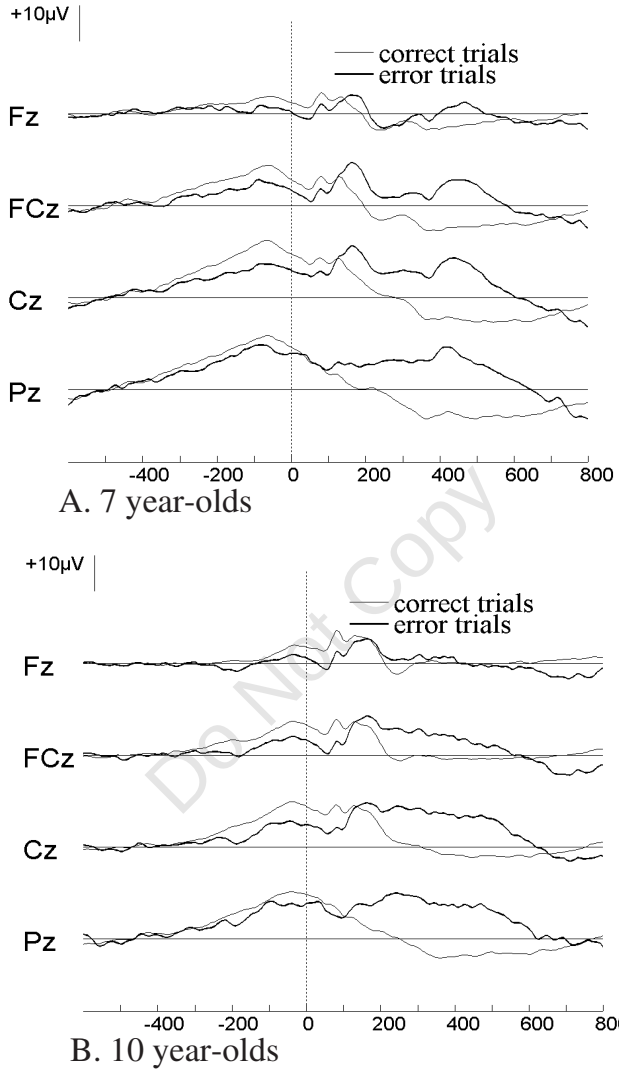

FIGURE 4 Grand average response-locked ERPs at four scalp midline sites for correct trials (thin line) and error trials (thick line) for 7-year-olds ($N = 12$; Plate A), for 10-year-olds ($N = 10$; Plate B), for 13-year-olds ($N = 8$; Plate C), and for 15-year-olds ($N = 8$; Plate D). ERPs are presented relative to a 200 msec baseline, 600 msec to 400 msec prior to the response. The vertical hashed line represents the timing of the response. Note that the peak-to-peak amplitudes of the ERN do not greatly differ at FCz and Cz, especially in the younger children.

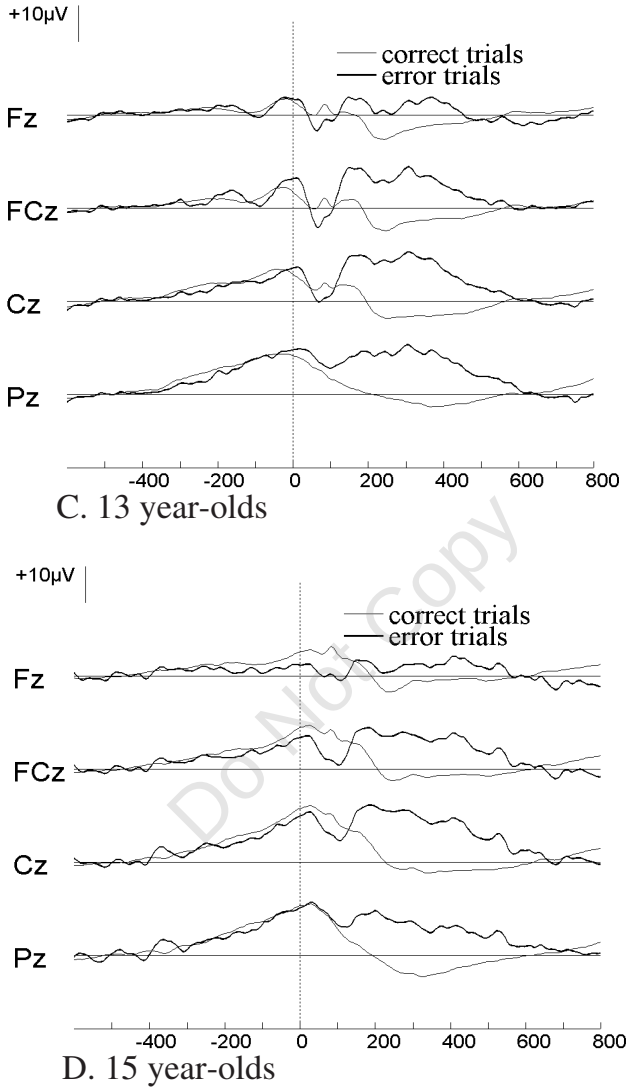

FIGURE 4 *(Continued)*

4 displays four midline sites for age groups 7, 10, 13, and 15, to illustrate the scalp distribution across age groups. The ERN amplitude is larger at FCz (by 1.4 μV peak-to-peak on average for the children) than at Cz on average although the range is large (larger for 68%, smaller for 32%) and the values at the two sites correlated very highly (r = .87). We ran analyses with those subjects for whom we had FCz

amplitudes and confirmed that the results yielded the same pattern of age effects as for the Cz amplitudes (see the following). The adult ERN amplitude values appeared discontinuous with the older male adolescents (see Figure 5), and therefore we analyzed age trends only over the 7 to 18 year range.

Developmental Trends on Error Trials from 7 to 18 Years

The ERN, relative to an early baseline of 600 to 400 msec before the response, shows an increase in amplitude with age over the 7 to 18 years age span, $R^2 = .146$, $F(1,122) = 20.9$, $p < .001$. However, this is modified by a quadratic relationship, R^2 change = .083, $F(1,121) = 13.0$, $p < .001$. Although there is no relationship between the age and the amplitude of the positivity preceding the ERN over the entire distribution ($F<1$ for both linear and quadratic effects), there is considerable variation across the years. Therefore, we also examined the size of ERN amplitude as a difference from this positivity, and further analyses present this peak-to-peak (P3-to-ERN) measure. All results with this peak-to-peak measure replicated in the ERN measure referenced to the early baseline of 600 to 400 msec before the response. The linear and quadratic age effects in the peak-to-peak ERN accounted for 20.4% and 9.5% of the variance in the ERN, respectively, $F(1,122) = 31.2$, $p <$

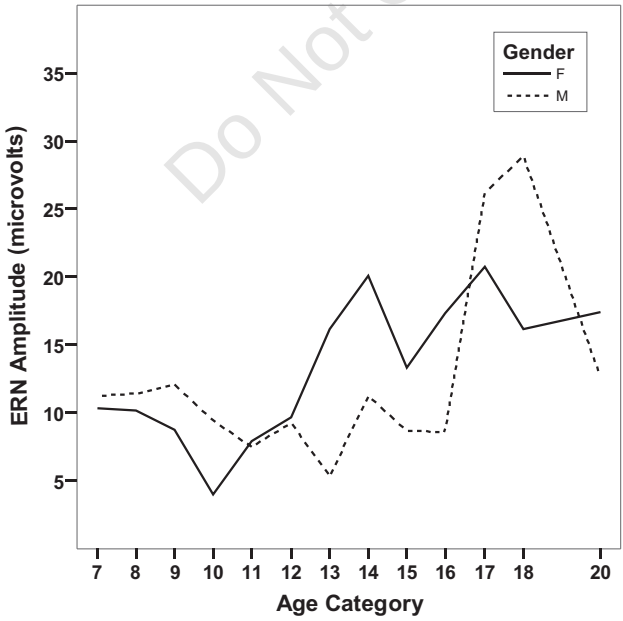

FIGURE 5 Age × Gender interaction in ERN amplitude measured peak-to-peak (P3-to-ERN) in μV.

.001 and $F(1,121) = 16.4$, $p < .001$ (see Figure 5). The clear reduction in ERN amplitude at ages 10 and 13 years and subsequent fluctuations through adolescence are suggestive of pubertal effects, so we examined the interactions with gender. To do this, the data were subjected to a regression analysis with age and age-squared on the first step, gender on the second step, and the interactions with gender (linear-age × gender, age-squared × gender) on the third. Age accounted for 30% of the variance again with the linear and quadratic components, $F(2, 121) = 25.8$, $p < .001$. Gender was not significant ($F < 1$), and the Age × Gender interaction was significant, $F(2,118) = 4.8$, $p = .01$ (see Table 2). The ERN quadratic distribution indicates an initial drop in amplitude, with a subsequent rise through adolescence. The girls have a minimum value at age 10 years; for the boys the lowest value is at age 13 years (see Figure 5). For the boys by themselves, age accounts for 43% of the variance in the ERN, over both a linear and a quadratic effect (linear: $F(1, 51) = 8.9$, $p = .004$, $R^2 = 14.8\%$; quadratic: $F(1, 51) = 25.0$, $p < .001$, R^2 change = 28.4%). For the girls, the linear effect captures the variance ($R^2 = 26.1\%$, $F_{1, 69} = 24.4$, $p < .001$) and the quadratic is not significant ($F < 1$). However, when the age range is restricted to the 8- to 14-year-olds, the girls also show both linear ($R^2 = 21.7\%$, $F(1, 48) = 13.3$, $p = .001$) and quadratic (R^2 change = 21.7%, $F(1, 47) = 18.0$, $p < .001$) effects.

To further validate the developmental trend of the ERN amplitude, several factors also shown to change with age were considered, specifically the number of trials that were included in the ERPs and eye blink artifact removal method. There was a significant difference between age groups in the number of participants whose eye blink artifacts were removed by rejection or by regression, $\chi^2(12, N = 124) = 23.85$, $p = .013$, with the youngest groups having the most participants whose eye blink artifacts were removed by regression. Number of trials included in each participant's ERP also varied, which may influence the amplitude of the ERP components. The ERPs that had the eye blink artifacts removed by regression methods had significantly fewer trials included in the ERP than those ERPs that

TABLE 2
Summary of Regression Analysis Evaluating the Contributions of Age and Gender (Independent Measures) in the Peak-to-Peak ERN Amplitude (Dependent Measure)

					Change Statistics				
Model	*R*	R^2	*Adjusted* R^2	*Standard Error of the Estimate*	R^2 *Change*	*F Change*	*df 1*	*df 2*	*Significant F Change*
1	.547[a]	.299	.287	6.02519	.299	25.795	2	121	.000
2	.547[b]	.299	.281	6.04992	.000	0.013	1	120	.911
3	.593[c]	.352	.324	5.86688	.053	4.802	2	118	.010

[a]Predictors: (Constant), age, age^2. [b]Predictors: (Constant), age, age^2, gender (1 = female, 2 = male). [c]Predictors: (Constant), age, age^2, gender (1 = female, 2 = male), Age × Gender, Age^2 × Gender.

were subjected to eye blink artifact rejection ($R^2 = .08$, $df = 1,122$, $p = .002$). To test the validity of the developmental trend of the ERN amplitude, the data were subjected to a regression analysis with number of trials and eye blink artifact method on the first step and age on the second step. Number of trials and eye blink artifact removal method accounted for 9.2% of the variance of the ERN amplitude, $F(1,121) = 6.1$, $p = .003$, and age still accounted for 17% of the variance in the ERN amplitude, $F(1,120) = 27.9$, $p < .0005$. Thus, the developmental trends in ERN amplitude persist even after removing other developmental correlates.

The Pe amplitude did not change with age, $r = –.08$, *ns.* Thus, unlike the ERN, the Pe does not show systematic changes with age from 7 to 18 years. See Table 3 for the mean amplitude of the P3, ERN, P3-to-ERN, and Pe for all age groups.

Developmental Trends on Correct Trials

Consistent with previous results reported in the literature, on correct trials the adults produced a positive peak about the time of the response, followed by a negative drift reaching baseline about 200 msec later (see Figure 2). However, the cor-

TABLE 3
Mean Amplitude (μV) of each ERP Component (and Standard Deviation) in the Error Trials Time Locked to Response for Each Age Group Measured With –600 to –400 Baseline at Cz

		ERP Components							
		P3		*ERN*		*P3-ERN Peak-to-Peak*		*Pe*	
Age	*n*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
7	12	12.7	7.4	1.8	8.1	10.9	2.4	17.8	12.6
8	10	10.2	8.0	–0.2	7.2	10.4	3.9	17.9	7.1
9	18	10.4	7.6	0.5	6.6	9.9	5.5	17.9	7.3
10	10	9.7	4.0	2.5	4.3	7.2	6.0	16.8	6.5
11	11	8.6	5.0	0.9	3.9	7.7	3.5	20.8	6.0
12	18	13.0	7.3	3.6	7.5	9.4	5.2	19.0	5.8
13	8	11.4	9.2	–0.7	6.2	12.1	7.4	17.6	9.5
14	9	12.0	7.4	–5.1	7.9	17.1	8.4	16.3	3.2
15	8	15.7	6.8	4.1	5.3	11.6	5.9	21.6	5.1
16	6	7.8	6.5	–6.6	6.3	14.4	7.9	16.1	5.4
17	6	10.5	5.2	–13.0	8.0	23.5	7.4	11.7	5.0
18	8	10.8	8.2	–10.1	9.0	20.9	8.5	16.7	10.0
Adults		7.4	4.9	–8.4	6.8	15.8	7.0	12.1	6.1
Grand mean		10.5	6.8	–2.1	8.4	12.6	7.2	16.8	7.5

Note. ERP = event-related potential; P3 = positive component around 300 msec following the stimulus; ERN = event-related negativity; Pe = largest positivity following the ERN.

rect trials in children revealed a negative-then-positive shift at about the same time as the ERN in the error trials (see Figure 3). This correct-trials ERN has been labeled the correct-response negativity (CRN: Coles, Scheffers, & Holroyd, 2001). All participants had measurable CRNs except for 11 (five adults, one 18-year-old, one 17-year-old, two 15-year-olds, one 9-year-old, and one 8-year-old). In order for the CRN to be measurable, the negativity must be followed by another positive deflection before coming back to baseline. To determine the magnitude of the CRN, we used a peak-to-peak value (from the CRN to the late positive deflection peak). To evaluate the age effect of the amplitude of the CRN, the data were subjected to a regression analysis with age on the first step, age-squared on the second step, and age-cubed on the third (to capture an apparent cubic relationship).

The CRN reduces in amplitude with age from 7 to 18 years, R^2 = .069, $F(1,116) = 8.6$, p = .004. Although the quadratic function was not significant, the linear function was modified by a cubic relationship, R^2 change = .069, $F(1,114) = 5.3$, p = .023). Figure 6 shows this cubic function with 3 noticeable outliers marked with arrows. The linear age effect becomes stronger when these 3 outliers are removed from the data set, R^2 = .129, $F(1,113) = 16.7$, $p < .0005$,

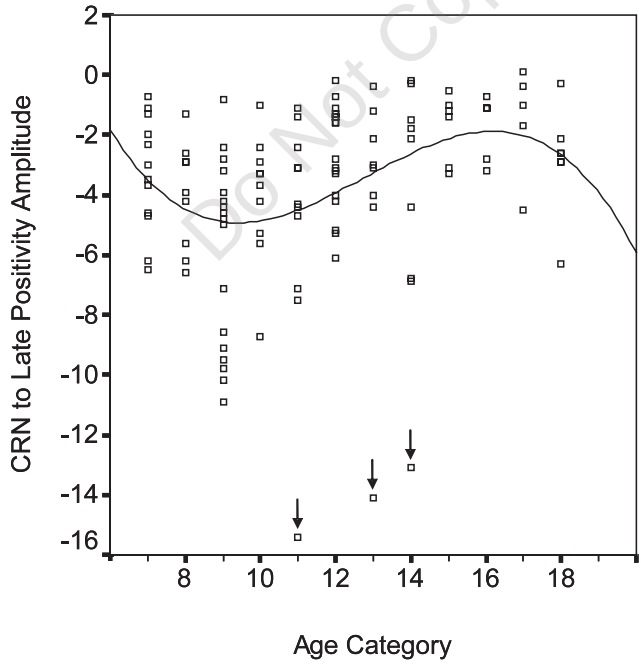

FIGURE 6 Age differences in CRN amplitude in μV. Both linear and cubic effects are significant, and are made more reliable by the removal of the three outliers marked with arrows.

as does the cubic relationship, R^2 change = .087, $F(1,111) = 12.3$, $p = .001$. The cubic distribution indicates an initial increase in CRN amplitude peaking around 9 to 10 years of age, with a subsequent reduction through adolescence peaking around 16 years of age, followed by another increase in the late teen years. A subsequent regression with gender and Gender × Age interactions indicated a lack of gender effects. CRN amplitude does not correlate with the mean RT on correct trials ($r = .07$, $p = .459$) when controlling for age. The variability in RT (as reflected by the *SD* of RT) did not correlate with CRN amplitude on correct trials when controlling for age ($r = -.13$, $p = .174$).

DISCUSSION

The results of this study support a developmental model of the ERN. Given that the likely generator of the ERN is the dorsal and/or rostral ACC (Luu et al., 2003), our results support the notion of a late maturation of the ACC. A recent biochemical model of the error processing system implicates the mesencephalic dopamine system as a major player in the production of the ERN (Holroyd & Coles, 2002). The authors of this model propose that when a participant commits an error, the mesencephalic dopamine system conveys a negative reinforcement learning signal to the frontal cortex, where it generates the ERN in the ACC. Interestingly, Lambe et al. (2000) reported a large developmental increase in the dopaminergic innervation of pyramidal cells in the PFC in primates reaching its highest level during puberty, suggesting that the biochemical system that may produce the ERN is not fully mature until young adulthood. Furthermore, a rapid decline in potential sites of dopamine release occurs towards the end of adolescence, reaching stable adult levels in early adulthood (Anderson, Classey, Conde, Lund, & Lewis, 1995; Lewis, 1997). Lewis suggests that the neuromodulation effects of dopamine may have a robust influence on information processing around the adolescent years. These changes in dopamine neuroactivity may account for some of the fluctuations seen in the ERN at the beginning of adolescence and then the discontinuity seen from late teen to early adult years (see Figure 5). So, in addition to the anatomical, physiological, behavioral, clinical, and electrophysiological evidence for a relatively late maturation of the PFC and the ACC mentioned previously, it appears that there may be a biochemical system specific to the generation of the ERN that also matures during adolescence. This late-developing mesencephalic dopaminergic system may be one reason for the absence of a strong ERN in young children in this study. Thus, whether due to developmental biochemical or structural changes in the ACC, our data appear to support a model of late maturation of this brain region as reflected in ERPs related to error monitoring.

However, before concluding this, we must consider other potential sources for the age difference in the ERN amplitude. The results of the adult and older adoles-

cents' ERPs to error trials reported here are similar in morphology to previous studies using the same paradigm (Davies et al., 2001; Pailing et al., 2002) with all displaying a measurable ERN and Pe. However, the ERN was very small in most young children, ages 7 to 12 years of age. In fact, the average amplitude for the peak negativity—between 20 ms before the response to 100 ms after the response for most of the children—was above the baseline. Only the age groups of 8, 13, 14, and 16 through 18 years had an average amplitude that dipped below the baseline. See Figure 7 for selected ERPs from children at various ages illustrating the range obtained. This raises the question of whether the children who showed a lack of a strong ERN recognized that they are making an error response (i.e., whether they registered that they had made an error). Our data suggest that the children did recognize their errors. First, the children made all the same gestures and grimaces as adults when they made errors, suggesting that they realized that they had pressed the wrong button for a given trial. Second, even in the absence of the ERN, they all produced a robust Pe, as did the adults. This is consistent with the findings of Nieuwenhuis and colleagues, showing that for recognized errors adults produced a

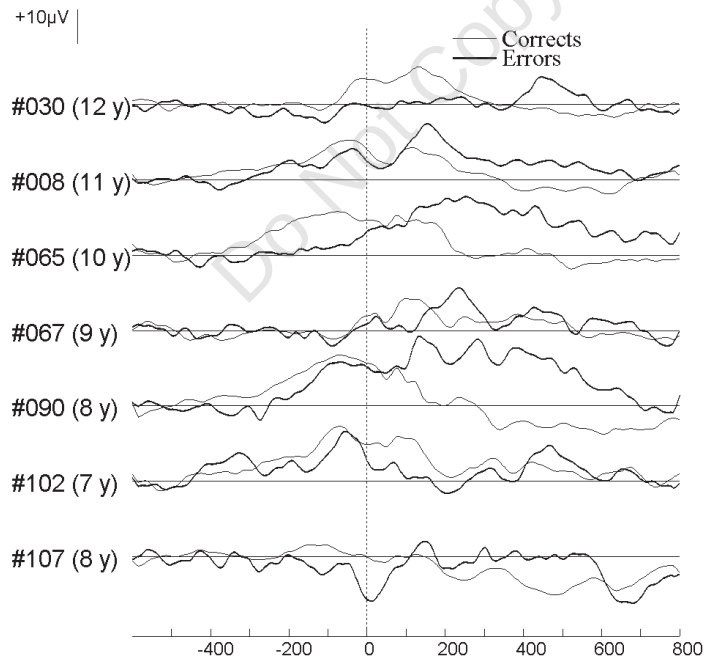

FIGURE 7 Selected waveforms from individual children (ages 7–12) at Cz. The vertical hashed line represents the timing of the response. ERPs are presented relative to a 200 msec baseline, 600 msec to 400 msec prior to the response. Younger children rarely exhibit a strong ERN, but always a Pe. A rare strong ERN is shown last in this figure.

Pe, but for unperceived errors a Pe was not generated (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001; Ridderinkhof et al., 2002). Third, the young children slowed their response time on trials immediately following error trials, as did the adults, when compared to the response time following correct trials. The post-error slowing is considered a controlled process (Rabbitt, 1966) and indicates recognition of the error (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). These three findings support the notion that there is a dissociation between the processing of the error and production of an ERN in children. This dissociation has also been shown in patients with lesions of the ACC (Stemmer et al., 2003). However in the intact adult brain, the error processing and production of an ERN may be tightly linked (e.g., Ridderinkhof et al., 2002).

Falkenstein and colleagues suggest that the Pe is a specific error component, separate from the ERN (Falkenstein et al., 2000). They suggest that this late processing following errors, as reflected in the Pe, is functionally different from error detection and response checking, but relates to error processing. Therefore, if young children produce a Pe, they must be registering that they made an error. If children are producing a Pe but not an ERN, one could speculate that one reason for this could be that the Pe is generated by brain regions different from those regions associated with the ERN. This is supported by Van Veen and Carter's (2002) dipole model indicating that the late Pe (about 280 to 320 msec following the response), which was maximal at Pz, indicated the rostral ACC and the superior parietal cortex as sources (Van Veen & Carter, 2002). In a previous study on individual differences, we found that the Pe in adults correlated significantly with the P3 of the stimulus-locked waveforms, suggesting that the Pe was similar to the P3, and was presumably an evaluation of the internally generated error stimulus (Davies et al., 2001). If the Pe can be at least partially generated from more posterior brain regions, this may account for why young children display a robust Pe and not the more frontally generated ERN. Further dipole source generator modeling with children will be required to address whether different cortical regions generate the Pe and ERN.

If future data support that in children the Pe is primarily generated by posterior brain regions, then the ERP paradigms that produce an ERN and Pe can be used to dissociate the functions of different brain regions. The results of this study indicate that although children are processing the error information, in that their behaviors are similar to adults, they are not using the frontal brain regions as adults are to process the same error information.

We also do not think that the reason children have smaller ERNs is due to them having smaller ERPs in general, because the amplitude of the Pe component was actually as large in young children as in adults. In fact, another possible explanation for why young children do not produce as large an ERN as adults is that children have a larger P3 response to the stimulus (occurring in the ERP about the time of the response; see Figures 2 and 3). This stronger P3 response would move the

waveform positive compared to the baseline, reducing the degree of negativity of the ERN. To test this hypothesis we measured peak-to-peak (P3-to-ERN) amplitude, which was also larger in adults. Thus, our data rule out the ERN difference being due to simply a larger P3 in children.

Another factor to take into consideration is that increased time-pressure can reduce the amplitude of the ERN. Falkenstein and colleagues found that under severe time pressure, the ERN was reduced, compared to a lesser time pressure (Falkenstein et al., 2000). However, in our study, the children ages 7 to 10 were given a stimulus set with longer SOA (children had 1.5 sec SOA, and older children and adults had a 1.1 SOA), which was selected based on some pilot data collected in young children. Given that there was no age trend with respect to the number of errors, older children did not attend to accuracy more than did younger children.

Falkenstein and colleagues hypothesize that the small CRN negativity in correct trials is actually due to a few correct trials where the participant guesses on the response, gets it correct, but due to the feeling that it may be an error, could produce an ERN (Falkenstein et al., 2000). When averaged with other correct trials, this would produce a small negativity in correct trials (Falkenstein et al., 2000). Coles and colleagues similarly suggest that the negativity seen in correct trials could be due to the participant being unsure of his or her response due to factors such as the stimulus being degraded or due to fatigue (Coles et al., 2001). Cole and colleagues also suggest that artifacts may cause the presence of the CRN such as variable response times or slower response times. However, our developmental data do not show any relationship of the CRN amplitude with response time or with the variability of response time. Interestingly, the CRN was more evident in young children than in adults in our study, which of course could reflect less certainty about their responses, although this possibility is mitigated by their having similar post-error slowing. If true, however, and if this certainty factor is reliant on a well-functioning dorsolateral prefrontal cortex (DLPFC), then immaturity in this region could account for the results: Gehring and Knight (2000) found that patients with DLPFC lesions produced correct ERPs with large CRNs. Also interesting in our data is the fact that there is a discontinuity in the developmental trends of both the ERN in incorrect trials and the CRN in correct trials around age 9 and 10 years. However these data should be interpreted cautiously until more developmental investigations explore possible confounds that are inherent in ERP studies involving children.

Related to the ERN and CRN, one issue that needs to be further explored is whether the ERP waveform of the correct response is the same as the error ERPs, especially in children younger than 12 years of age (see Figure 3). In other words, do children show different electrocortical responses to error and correct trials? To test this we compared the peak-to-peak CRN amplitude (P3-to-CRN in correct trials) to the peak-to-peak ERN amplitude (P3-to-ERN in error trials) in children 12

years and younger, $F(1, 71) = 17.7$, $p < .0005$, indicating that the children process the error trials differently from the correct trials.

Essential to understanding the developmental pattern of the ERN is the exploration of the question of why some young children produce strong ERNs, although most do not (see Figure 7). A recent study shows that children with obsessive-compulsive tendencies and those with greater socialization scores have larger ERNs (Segalowitz, Davies, Santesso, Gavin, & Schmidt, 2004). Further studies are needed to determine the extent to which executive functions and personality influence the production of ERNs.

In summary, the data presented here support a continued physiological maturation of the ACC and its connections with the PFC through adolescence. The response-monitoring paradigm may be a useful one with which to study the physiological development of the ACC and PFC, given that the ERN is generated in the ACC and develops into adolescence, not reaching adult levels until late teen years. This contrasts with the development of the Pe component of response monitoring, found to be very robust even in the young children.

ACKNOWLEDGMENTS

This research was funded, in part, by Grant 1 K01 HD01201–01A1 from the National Institute of Child Health and Human Development to Patricia L. Davies; and, in part, by Grant 122222–98 from the Natural Sciences and Engineering Research Council of Canada to Sidney J. Segalowitz.

REFERENCES

Adleman, N. E., Menon, V., Blasey, C. M., White, C. D., Warsofsky, I. S., Glover, G. H., & Reiss, A. L. (2002). A developmental fMRI study of the Stroop Color-Word Task. *Neuroimage, 16*(1), 61–75.

Anderson, S. A., Classey, J. D., Conde, F., Lund, J. S., & Lewis, D. A. (1995). Synchronous development of pyramidal neuron dendritic spines and parvalbumin-immunoreactive chandelier neuron axon terminals in layer III of monkey prefrontal cortex. *Neuroscience, 67*(1), 7–22.

Benes, F. M., Vincent, S. L., Molloy, R., & Khan, Y. (1996). Increased interaction of dopamine-immunoreactive varicosities with GABA neurons of rat medial prefrontal cortex occurs during the postweanling period. *Synapse, 23*(4), 237–245.

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science, 280*(5364), 747–749.

Coles, M. G., Scheffers, M. K., & Holroyd, C. B. (2001). Why is there an ERN/Ne on correct trials? Response representations, stimulus-related components, and the theory of error-processing. *Biological Psychology, 56*(3), 173–189.

Courchesne, E. (1977). Event-related brain potentials: Comparison between children and adults. *Science, 197*(4303), 589–592.

Courchesne, E. (1978). Neurophysiological correlates of cognitive development: Changes in long-latency event-related potentials from childhood to adulthood. *Electroencephalography and Clinical Neurophysiology, 45*(4), 468–482.

Courchesne, E. (1983). Cognitive components of the event-related potential: changes associated with development. In W. Ritter (Ed.), *Tutorials in event related potential research: endogenous components* (pp. 329–344). Amsterdam: North-Holland Publishing Company.

Cunningham, M. G., Bhattacharyya, S., & Benes, F. M. (2002). Amygdalo-cortical sprouting continues into early adulthood: Implications for the development of normal and abnormal function during adolescence. *Journal of Comparative Neurology, 453*(2), 116–130.

Cycowicz, Y., Friedman, D., & Rothstein, M. (1996). An ERP developmental study of repetition priming by auditory novel stimuli. *Psychophysiology, 33*(6), 680–690.

Davies, P. L., & Rose, J. D. (1999). Assessment of cognitive development in adolescents by means of neuropsychological tasks. *Developmental Neuropsychology, 15*(2), 227–248.

Davies, P. L., Segalowitz, S. J., Dywan, J., & Pailing, P. E. (2001). Error-negativity and positivity as they relate to other ERP indices of attentional control and stimulus processing. *Biological Psychology, 56*(3), 191–206.

Dehaene, S. (1996). The organization of brain activations in number comparison: Event-related potentials and the additive-factors method. *Journal of Cognitive Neuroscience, 8*, 47–68.

Dehaene, S., Posner, M. I., & Tucker, D. M. (1994). Localization of a neural system for error detection and compensation. *Psychological Science, 5*, 303–305.

Devinsky, O., Morrell, M. J., & Vogt, B. A. (1995). Contributions of anterior cingulate cortex to behaviour. *Brain, 118*(1), 279–306.

Dikman, Z. V., & Allen, J. J. (2000). Error monitoring during reward and avoidance learning in high- and low-socialized individuals. *Psychophysiology, 37*(1), 43–54.

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception & Psychophysics, 16*(1), 143–149.

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. *Electroencephalography & Clinical Neurophysiology, 78*(6), 447–455.

Falkenstein, M., Hoormann, J., Christ, S., & Hohnsbein, J. (2000). ERP components on reaction errors and their functional significance: A tutorial. *Biological Psychology, 51*(2–3), 87–107.

Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science, 4*(6), 385–390.

Gehring, W. J., & Knight, R. T. (2000). Prefrontal-cingulate interactions in action monitoring. *Nature Neuroscience, 3*(5), 516–520.

Greenough, W. T., Black, J. E., & Wallace, C. S. (1987). Experience and brain development. *Child Development, 58*(3), 539–559.

Holroyd, C. B., & Coles, M. G. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review, 109*(4), 679–709.

Johnstone, S. J., Barry, R. J., Anderson, J. W., & Coyle, S. F. (1996). Age-related changes in child and adolescent event-related potential component morphology, amplitude and latency to standard and target stimuli in an auditory oddball task. *International Journal of Psychophysiology, 24*(3), 223–238.

Kalsbeek, A., Voorn, P., Buijs, R. M., Pool, C. W., & Uylings, H. B. (1988). Development of the dopaminergic innervation in the prefrontal cortex of the rat. *Journal of Comparative Neurology, 269*(1), 58–72.

Kiehl, K. A., Liddle, P. F., & Hopfinger, J. B. (2002). Error processing and the rostral anterior cingulate: An event-related fMRI study. *Psychophysiology, 37*(2), 216–223.

Kolb, B., Wilson, B., & Taylor, L. (1992). Developmental changes in the recognition and comprehension of facial expression: Implications for frontal lobe function. *Brain & Cognition, 20*(1), 74–84.

Lambe, E. K., Krimer, L. S., & Goldman-Rakic, P. S. (2000). Differential postnatal development of catecholamine and serotonin inputs to identified neurons in prefrontal cortex of rhesus monkey. *Journal of Neuroscience, 20*(23), 8780–8787.

Lewis, D. A. (1997). Development of the prefrontal cortex during adolescence: Insights into vulnerable neural circuits in schizophrenia. *Neuropsychopharmacology, 16*(6), 385–398.

Luu, P., Tucker, D. M., Derryberry, D., Reed, M., & Poulsen, C. (2003). Electrophysiological responses to errors and feedback in the process of action regulation. *Psychological Science, 14*(1), 47–53.

Menon, V., Adleman, N. E., White, C. D., Glover, G. H., & Reiss, A. L. (2001). Error-related brain activation during a Go/NoGo response inhibition task. *Human Brain Mapping, 12*(3), 131–143.

Miltner, W. H. R., Braum, C. H., & Coles, M. G. H. (1997). Event-related brain potentials following incorrect feedback in a time-estimation task: Evidence for a "generic" neural system for error detection. *Journal of Cognitive Neuroscience, 9*, 788–798.

Nieuwenhuis, S., Ridderinkhof, K. R., Blom, J., Band, G. P., & Kok, A. (2001). Error-related brain potentials are differentially related to awareness of response errors: Evidence from an antisaccade task. *Psychophysiology, 38*(5), 752–760.

Pailing, P. E., Segalowitz, S. J., Dywan, J., & Davies, P. L. (2002). Error negativity and response control. *Psychophysiology, 39*(2), 198–206.

Rabbitt, P. M. (1966). Errors and error correction in choice-response tasks. *Journal of Experimental Psychology, 71*(2), 264–272.

Rakic, P., Bourgeois, J. P., Eckenhoff, M. F., Zecevic, N., & Goldman-Rakic, P. S. (1986). Concurrent overproduction of synapses in diverse regions of the primate cerebral cortex. *Science, 232*(4747), 232–235.

Ridderinkhof, K. R., de Vlugt, Y., Bramlage, A., Spaan, M., Elton, M., Snel, J., & Band, G. P. (2002). Alcohol consumption impairs detection of performance errors in mediofrontal cortex. *Science, 298*(5601), 2209–2211.

Rosenberg, D. R., & Lewis, D. A. (1995). Postnatal maturation of the dopaminergic innervation of monkey prefrontal and motor cortices: A tyrosine hydroxylase immunohistochemical analysis. *Journal of Comparative Neurology, 358*(3), 383–400.

Segalowitz, S. J., Davies, P. L., Santesso, D., Gavin, W. J., & Schmidt, L. A. (2004). The development of the error negativity in children and adolescents. In M. Ullsperger & M. Falkenstein (Eds.), *Errors, conflicts, and the brain: Current opinions on performance monitoring* (pp. 177–184). Leipzig: Max Planck Institute for Cognition and Neuroscience.

Sartory, G., Besken, E., & Pothmann, R. (1997). Contingent negative variation in childhood migraine. *Journal of Psychophysiology, 11*(2), 138–146.

Scheffers, M. K., Coles, M. G., Bernstein, P., Gehring, W. J., & Donchin, E. (1996). Event-related brain potentials and error-related processing: An analysis of incorrect responses to go and no-go stimuli. *Psychophysiology, 33*(1), 42–53.

Segalowitz, S. J. (1996). EYEREG.EXE program for epoch-based eye-channel correction of ERPs. St. Catharines, Canada: Brock University.

Segalowitz, S. J., Unsal, A., & Dywan, J. (1992). Cleverness and wisdom in 12-year-olds: Electrophysiological evidence for late maturation of the frontal lobe. *Developmental Neuropsychology, 8*(2–3), 279–298.

Stemmer, B., Segalowitz, S. J., Witzke, W., & Schönle, P. W. (2003). Error detection in patients with lesions to the medial prefrontal cortex: An ERP study. *Neuropsychologia, 42,* 118–130.

Stuss, D. T. (1992). Biological and psychological development of executive functions. *Brain & Cognition, 20*(1), 8–23.

Timsit-Berthier, M., & Hausmann, J. (1972). Étude de la VCN et du phénomène de préparation motrice chez des enfants de 5 à 15 ans [Study of the CNV and of the phenomenon of motor preparation in 5–15-year-old children]. *Revue d'Electroencephalographie et de Neurophysiologie Clinique, 2*(2), 141–146.

Van Bogaert, P., Wikler, D., Damhaut, P., Szliwowski, H. B., & Goldman, S. (1998). Regional changes in glucose metabolism during brain development from the age of 6 years. *Neuroimage, 8*(1), 62–68.

Van Hoesen, G. W., Morecraft, R. J., & Vogt, B. A. (1993). Connections of the monkey cingulate cortex. In M. Gabriel (Ed.), *Neruobiology of cingulate cortex and limbic thalamus: A comprehensive handbook* (pp. 249–284). Boston: Birkhauser.

Van Veen, V., & Carter, C. S. (2002). The timing of action-monitoring processes in the anterior cingulate cortex. *Journal of Cognitive Neuroscience, 14*(4), 593–602.

Verney, C., Berger, B., Adrien, J., Vigny, A., & Gay, M. (1982). Development of the dopaminergic innervation of the rat cerebral cortex. A light microscopic immunocytochemical study using anti-tyrosine hydroxylase antibodies. *Brain Research, 281*(1), 41–52.

Vogt, B. A. (1993). Structural organization of cingulate cortex: Areas, neruons, and somatodendritic transmitter receptors. In M. Gabriel (Ed.), *Neruobiology of cingulate cortex and limbic thalamus: A comprehensive handbook* (pp. 19–70). Boston: Birkhauser.

Warren, C. A., & Karrer, R. (1984). Movement-related potentials during development: A replication and extension of relationships to age, motor control, mental status and IQ. *International Journal of Neuroscience, 24*(2), 81–96.