ERP Correlates of Action Monitoring in Adolescence

CECILE D. LADOUCEUR, RONALD E. DAHL, AND CAMERON S. CARTER

Department of Psychiatry, School of Medicine, Western Psychiatric Institute and Clinic, University of Pittsburgh, Pittsburgh, Pennsylvania 15213, USA

ABSTRACT: This study examines the development of action monitoring in adolescence by measuring the N200, the ERN (error-related negativity) and the P_E (error positivity), which are event-related potentials (ERPs) that appear to be generated in the anterior cingulate cortex (ACC) and reflect action-monitoring processes. We predicted that amplitude would be significantly greater in late compared to early adolescence. Participants consisted of 11 adolescents that were divided into early (age 9–14) and late (age 14–17) adolescence groups. ERPs were recorded during an 840-trial arrow-flanker task and using 128-channel dense array EEG. Results indicated that there were no differences in P_E amplitude but that N200 and ERN amplitudes were greater in the late adolescence group. According to the conflict monitoring hypothesis, this suggests that the ability to detect error-related conflict, which is involved in the modulation of cognitive control, appears to be fully developed later in adolescence and may be linked to the maturation of the ACC.

KEYWORDS: ERN; P_E; N200; action monitoring; adolescence

Recently, studies in adults have identified event-related potentials (ERPs) that appear to be generated in the anterior cingulate cortex (ACC) and reflect action-monitoring processes.[1–3] Given important changes in the regulation of behavior during adolescence, the goal of this study was to examine the developmental changes in these action-monitoring ERPs (i.e., N200, error-related negativity [ERN], and error positivity [P_E]) in a group of adolescents. The prefrontal cortex undergoes important maturational changes during adolescence.[4] Therefore, we predicted that these action-monitoring ERPs would be significantly greater during late compared to early adolescence. Additional analyses were conducted to examine the relationship between ERN amplitude and post-error slowing in order to investigate the functional role of the ERN in the development of cognitive control. Given the suggested monitoring role of the ERN in cognitive control processes,[5] we hypothesized that ERN amplitude would be associated with trial-to-trial performance adjustments.

Address for correspondence: Cecile D. Ladouceur, Ph.D., Department of Psychiatry, School of Medicine, Western Psychiatric Institute and Clinic, University of Pittsburgh, Pittsburgh, PA 15213. Voice: 412-246-5872; fax: 412-246-5880.
ladouceurcd@upmc.edu

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METHODS

A total of 11 adolescents between 9 and 17 years of age (mean age = 14.18 years, SD = 2.44; 7 girls and 4 boys) participated in this study. Based on the median age, subjects were separated into two groups: early adolescence ($n = 5$, mean age = 12.20 years, SD = 2.17; 2 girls and 3 boys), and late adolescence: ($n = 6$, mean age = 15.83 years, SD = 0.98; 5 girls and 1 boy). Participants were screened for neurological and psychiatric disorders, and for visual accuracy. The University of Pittsburgh Institutional Review Board approved the study. To participate, children and their parents were required to sign assent and informed consent forms, respectively.

PROCEDURE

EEG measures were taken while subjects performed an arrow version of the Eriksen flanker task[6] with congruent (e.g., →→→→→) and incongruent (e.g., ←←→←←) conditions. The probability of each stimulus array was .25. The task comprised a total of 7 blocks with 120 trials each and one practice block of 60 trials. Subjects were asked to respond to the arrow in the center with either their right or left index finger, depending on the direction of the arrow. They also received specific instructions encouraging them to respond as quickly and as accurately as possible. To manipulate motivation further, participants were told at the beginning of the task that they could win a "cash bonus" depending on their performance (all received an extra $5).

EEG Data Acquisition and Analyses

The EEG data were recorded with a 128-channel Geodesic Sensor Net.[7] Impedance measures were below 50 KΩ. The EEG was digitized at 250 samples per second with a 0.1–100-Hz bandpass filter and digitally filtered offline using a 0.3–30-Hz bandpass filter. The EEG was segmented −400 to +800 ms for response-locked trials and −300 to +800 ms for stimulus-locked trials. Channels were interpolated if fast average amplitude was 200 μV, differential amplitude was greater than 100 μV, or if a channel had 0 variance or was bad more than 20% of the time. Segments were eliminated if they contained more than 10 bad channels, or if eye blinks or movements (greater than 70 μV) were detected. A spherical spline interpolation was used to correct bad channels. Channels were referenced to the vertex during data acquisition and re-referenced to the average and using a spherical spline interpolation (PARE Correction). Amplitude and latency were measured at Cz for the ERN, P_E, and N200 and at Pz for the P300; all were measured relative to an early prestimulus (N200, P300) or preresponse baseline (ERN, P_E) (−150 to −50 ms).

RESULTS

Behavioral Data

As expected, reaction times were significantly faster in the late- relative to the early-adolescence group, ($F(1, 9) = 5.79$, $P < .05$, partial $\eta^2 = .39$) (FIG. 1). Across

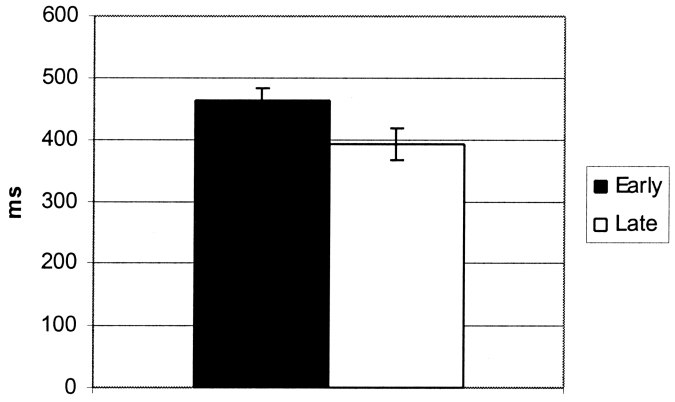

FIGURE 1. Overall reaction times.

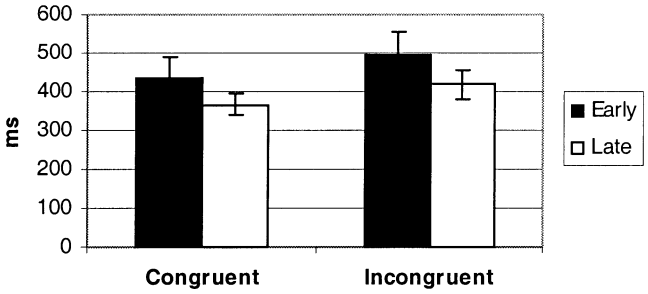

FIGURE 2. Reaction times in congruent and incongruent trials.

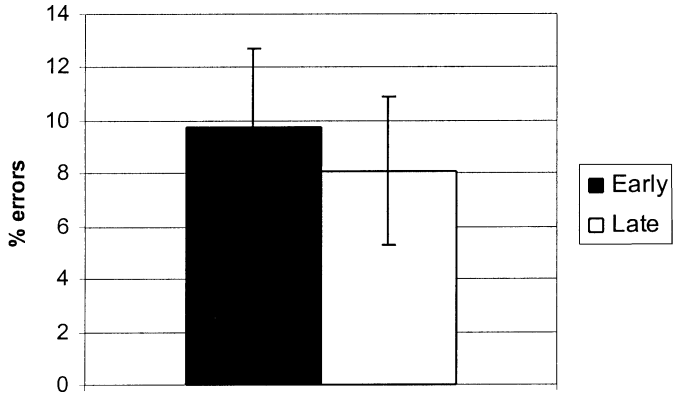

FIGURE 3. Overall percentage of errors.

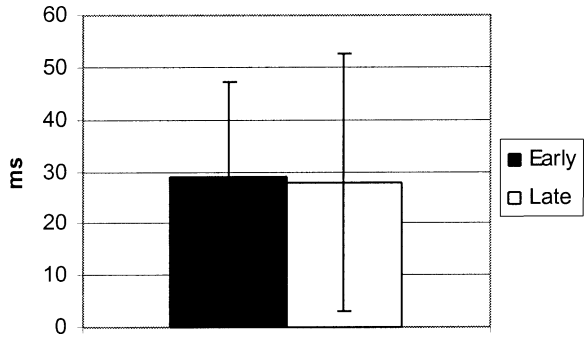

FIGURE 4. Post-error reaction times (Rabbitt effect).

age groups, reaction times were significantly slower in the incongruent relative to the congruent condition, ($F(1, 9) = 197.62$, $P < .001$, partial $\eta^2 = .96$) (FIG. 2) and faster on error ($M = 361$ ms, SD = 60 ms) compared to correct trials ($M = 438$ ms, SD = 64 ms) ($F(1, 9) = 53.36$, $P < .001$, partial $\eta^2 = .86$), which is consistent with results in adult studies.[3] In addition, there were no group differences in the percentage of errors, $t(9) = .98$, n.s.(FIG. 3) and in post-error slowing $t(9) = .98$, n.s. (FIG. 4), which is consistent with previous findings and suggests that performance was similar across age groups.

ERP Data

As shown in FIGURE 5, results revealed significantly greater P_E amplitude ($F(1, 9) = 20.07$, $P < .01$, partial $\eta^2 = .69$) and latency ($F(1, 9) = 30.76$, $P < .001$, partial $\eta^2 = .77$) in error relative to correct trials for both age groups. FIGURE 5 also reveals a significant trial type by age-group interaction ($F(1, 9) = 24.37$, $P < .01$, partial $\eta^2 = .73$) regarding ERN amplitude. Post hoc analyses indicated that ERN amplitude was greater in error compared to correct trials in late-, $t(5) = 5.04$, $P < .01$, but not in early adolescence, $t(4) = -1.96$, n.s.. In addition, as shown in FIGURE 6, N200 amplitude was significantly greater in incongruent relative to congruent trials in late-, $t(5) = 2.95$, $P < .05$, but not in early adolescence, $t(4) = 1.45$, n.s.. The trial type by age-group interaction did not reach statistical significance threshold, most likely owing to a lack of statistical power ($F(1, 9) = 1.42$, $P = .26$, partial $\eta^2 = .26$). No age-related differences were found associated with ERN or N200 latency. Moreover, both groups showed significantly larger P300 amplitude ($F(1, 9) = 15.97$, $P < .01$, partial $\eta^2 = .64$) and latency ($F(1, 9) = 22.31$, $P < .01$, partial $\eta^2 = .71$) in incongruent compared to congruent trials. These results, coupled with the similarity in morphology of the P_E component across groups, rules out the possibility that differences in ERN and N200 amplitude were attributable to overall smaller ERPs in early adolescence. In order to examine the functional role of the ERN in trial-to-trial performance adjustment, we separated error trials as a function of fast and slow reaction times in post-error correct trials within each participant and measured ERN amplitude and latency. After removal of one outlier in the older group, results revealed sig-

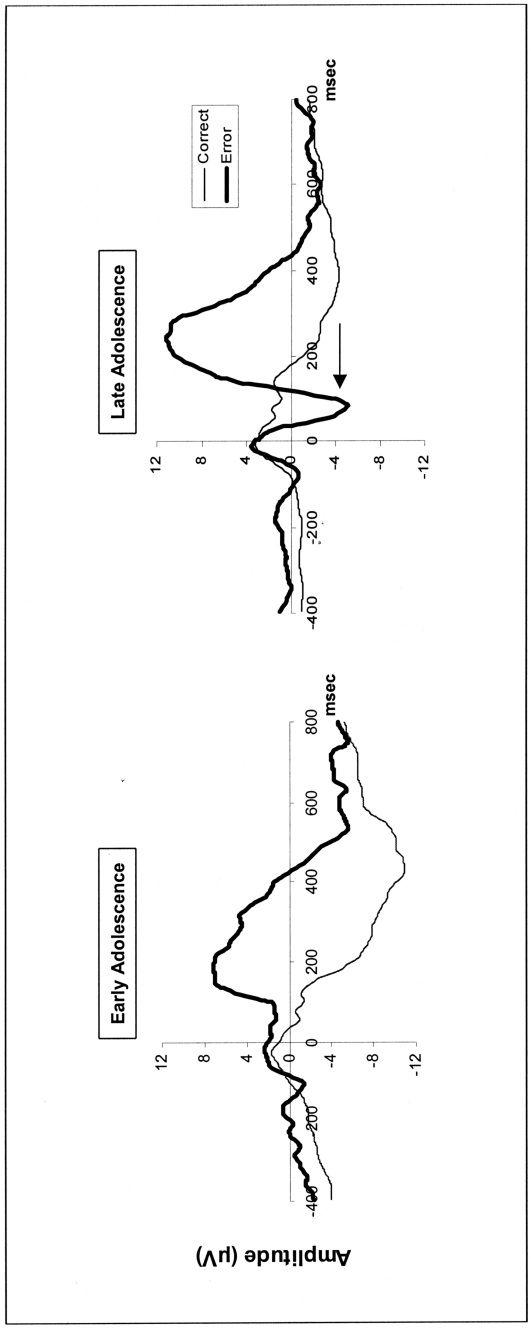

FIGURE 5. Response-locked waveforms.

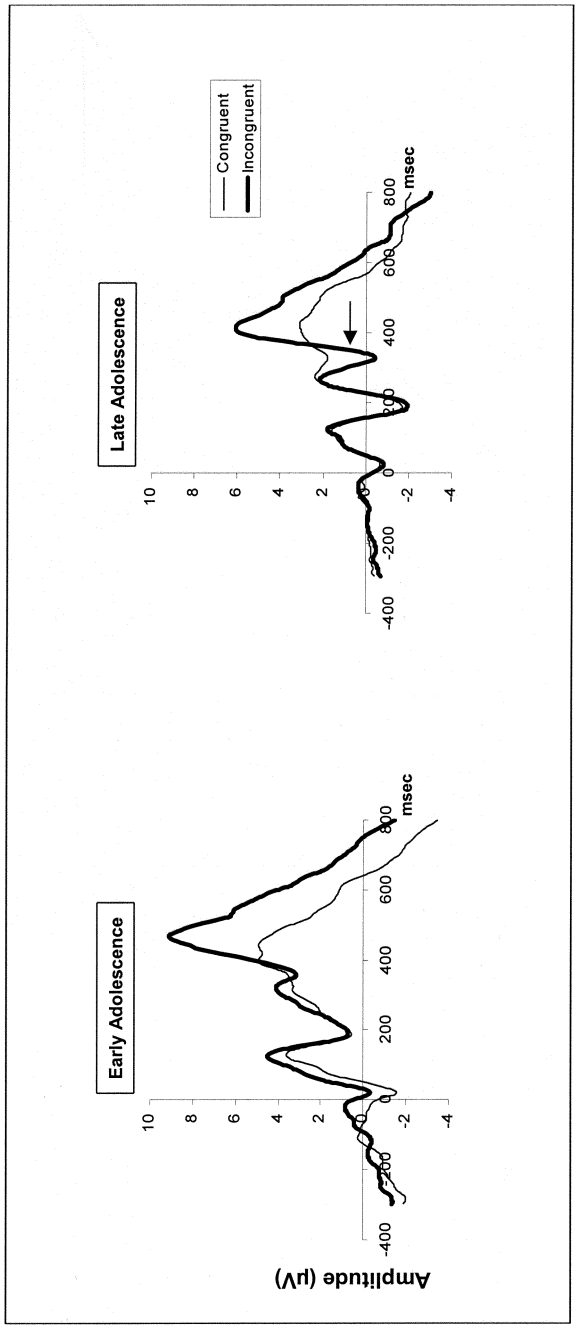

FIGURE 6. Stimulus-locked waveforms.

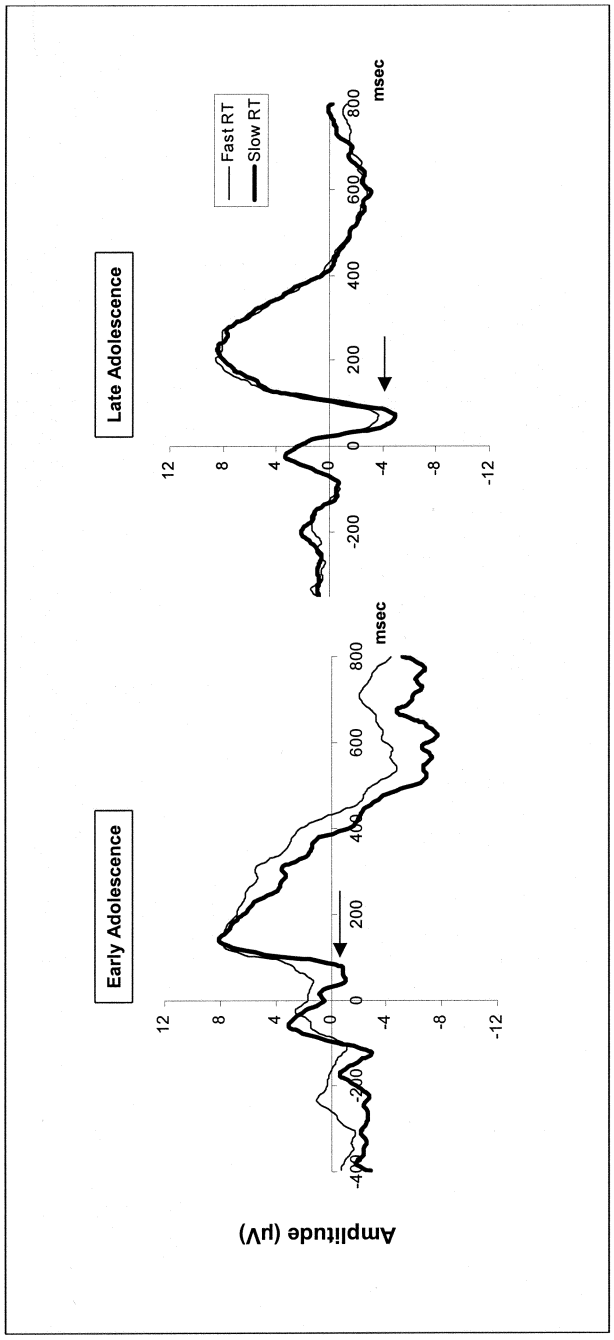

FIGURE 7. Response-locked waveforms as a function of post-error adjustments in performance.

nificantly larger ERN amplitudes in slow compared to fast trials in both age groups ($F(1, 8) = 11.21$, $P < .05$, partial $\eta^2 = .58$).

DISCUSSION

Although P_E was present in early and late adolescence, the N200 and ERN did not seem to appear until late adolescence. According to the conflict-monitoring hypothesis,[3,5] this suggests that although awareness of having committed an error is present early on, the ability of detecting error-related conflict, which is involved in the modulation of cognitive control, develops later in adolescence. Like the older group, the young adolescents did, however, show greater negativity in erroneous trials associated with post-error adjustment. This implies that the presence of ERN in early adolescence may be contingent upon performance adjustment, which would support the role of the ERN in the modulation of cognitive control.[5] Given that the N200 and ERN have been localized in the ACC,[3] these results suggest that the development of action monitoring, which is involved in the regulation of behavior, is linked to the maturation of the ACC during adolescence.[8]

Future research will involve replicating these results with a larger sample of adolescents, estimating the neural generators of these components using source localization analyses, and examining individual differences related to gender, puberty, and mood.

ACKNOWLEDGMENTS

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REFERENCES

1. FALKENSTEIN, M., J. HOHNSBEIN, J. HOORMAN, *et al*. 1991. Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. Electroencephalogr. Clin. Neurophysiol. **78:** 447–455.
2. GEHRING, W.J., B. GOSS, M.G.H. COLES, *et al*. 1993. A neural system for error detection and compensation. Psychol. Sci. **4:** 385–390.
3. VAN VEEN, V. & C.S. CARTER. 2002. The timing of action-monitoring processes in the anterior cingulate cortex. J. Cognit.Neurosci. **14:** 593–602.
4. LUNA, B. & J.A. SWEENEY. 2001. Studies of brain and cognitive maturation through childhood and adolescence: a strategy for testing neurodevelopmental hypotheses. Schizophrenia Bull. **27:** 443–455.
5. BOTVINICK, M.M., C.S. CARTER, T.S. BRAVER, *et al.* 2001. Conflict monitoring and cognitive control. Psychol. Rev. **108:** 624–652.
6. ERIKSEN, C.W. & E.A. ERIKSEN. 1979. Target redundancy in visual search: do repetitions of the target within the display impair processing? Percept. Psychophysiol. **26:** 195–205.
7. TUCKER, D.M. 1993. Spatial sampling of head electrical fields: the geodesic sensor net. Electroencephalogr. Clin. Neurophysiol. **87:** 154–163.
8. CASEY, B.J., R. TRAINOR, J. GIEDD, *et al*. 1997. The role of the anterior cingulate in automatic and controlled processes: a developmental neuroanatomical study. Devel. Psychobiol. **30:** 61–69.