Published in final edited form as:

Clin Neurophysiol. 2014 May ; 125(5): 962–970. doi:10.1016/j.clinph.2013.10.012.

Dissociation of preparatory attention and response monitoring maturation during adolescence

M. L. Padilla¹, A. Pfefferbaum^{1,2}, E. V. Sullivan², F. C. Baker^{3,4}, and I M. Colrain^{3,5}

¹Neuroscience Program, Center for Health Sciences, SRI International, Menlo Park, CA

²Department of Psychiatry & Behavioral Sciences, Stanford University School of Medicine, Stanford, CA, USA

³Human Sleep Research Program, Center for Health Sciences, SRI International, Menlo Park, CA, USA

⁴Brain Function Research Group, School of Physiology, University of the Witwatersrand, Johannesburg, South Africa

⁵School of Psychological Sciences, University of Melbourne, Parkville, Victoria, Australia

Abstract

Objective—Substantial brain development occurs during adolescence providing the foundation for functional advancement from stimulus-bound “bottom-up” to more mature executive-driven “top-down” processing strategies. The objective was to assess development of EEG markers of these strategies and their role in both preparatory attention (contingent negative variation, CNV) and response monitoring (Error Related Negativity, ERN, and Correct Related Negativity, CRN).

Methods—CNV, ERN and CRN were assessed in 38 adolescents (18 girls), age 11 to 18 years, using a variation of a letter discrimination task.

Results—Accuracy increased with age and developmental stage. Younger adolescents used a posterior attention network involved in inhibiting irrelevant information. Activity in this juvenile network, as indexed by a posteriorly-biased CNV and CRN decreased with age and advancing pubertal development. Although enhanced frontal CNV, known to be predictive of accuracy in adults, was not detected even in the older adolescents, top-down medial frontal response monitoring processes (ERN) showed evidence of development within the age-range studied.

Conclusions—The data revealed a dissociation of developmental progress, marked by relatively delayed onset of frontal preparatory attention relative to error monitoring.

Significance—This dissociation may render adolescents vulnerable to excessive risk-taking and disinhibited behavior imposed by asynchronous development of component cognitive control processes.

© 2013 International Federation of Clinical Neurophysiology. Published by Elsevier Ireland Ltd. All rights reserved.

*Correspondence: Edith V. Sullivan, Ph.D., Department of Psychiatry and Behavioral Sciences, Stanford University School of Medicine (MC5723), 401 Quarry Road, Stanford, CA 94305-5723, USA, Tel: +1 650 859-2880, FAX: +1 650 859-2743, edie@stanford.edu.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Keywords

ERP; cognitive control; development; adolescence; brain; maturation; error monitoring

Introduction

The adolescent brain undergoes dramatic maturational changes (Bava, Tapert, 2010; Giedd et al., 2010; Paus, 2010; Schmithorst, Yuan, 2010), with a curvilinear developmental trajectory of brain growth in childhood, followed by a rapid decline in adolescence (Giedd, 2004; Jernigan et al., 1991; Sowell et al., 2002; Steen et al., 1997) (for review, Stiles, Jernigan, 2010). Longitudinal studies reveal regionally specific nonlinear prepubertal increases, followed by post-pubertal decreases in cortical gray matter volume starting in more dorsal parietal cortices, spreading rostrally over the frontal cortex, and ending with the dorsolateral prefrontal cortex (Brain Development Cooperative Group, 2012; Giedd et al., 1999a; Lenroot et al., 2007; Raznahan et al., 2011a; Raznahan et al., 2011b; Shaw et al., 2008; Sowell et al., 2004; Sullivan et al., 2011). Adolescence is also associated with ongoing myelination of neuronal axons, progressing from inferior to superior and from posterior to anterior brain regions (Yakovlev, Lecours, 1967), with a consequent increase in white matter volume determining ultimate brain size (Courchesne et al., 2000; Giedd et al., 1999b; Pfefferbaum et al., 1994; Reiss et al., 1996; Sowell et al., 2004). Thus, brain structures follow different courses of maturation, thereby contributing to different maturational time courses of brain functions. Complex processes that rely on synergistic functions that do not mature in lock-step might help define the “awkward teenager,” embodying cognitive mature, maturing, and immature processes. Behavioral challenges incorporating complex attentional tasks measured with electrophysiological probes could serve to identify dissociable functions, index their maturational levels, and provide insight into limitations imposed by asynchronous development of component cognitive processes.

Functional correlates of brain developmental changes are the maturation of cognitive processes in adolescence and a notable shift in strategy from a posterior bottom-up stimulus-driven orienting network of brain regions (Posner, Petersen, 1990) characteristic of immature attentional processes to engagement of an anterior executive top-down attentional network (Corbetta, Shulman, 2002; Posner, Petersen, 1990). Several studies have provided evidence that with advancing adolescence, top-down processing predominates in several domains, including visual search (Acik et al., 2010; Donnelly et al., 2007), phonological processing (Bitan et al., 2009), dichotic listening (Andersson et al., 2008; Takio et al., 2009), and dual processing of verbal and visual information (Karatekin, 2004). Evidence from studies evaluating the effect of working memory load on Stroop interference (Spronk, Jonkman, 2012) and voluntary control of reflex responses (antisacade task) (Luna et al., 2001) show evidence of effective top-down modulation as not occurring until adulthood. Later development of frontal top-down mechanisms relative to other brain-behavior associations has been hypothesized to underlie the particular vulnerability of adolescents to engage in risky behavior (Casey, Jones, 2010).

Efficient cognitive processing involves both preparatory attention and the continuous monitoring of performance. These control processes permit high levels of functioning and enable flexible actions in response to moment-to-moment changes in the environment. In adults, these processes depend on the integrity of anterior cingulate cortex (ACC) and related prefrontal neural circuitry (Gehring, Knight, 2000; Rosahl, Knight, 1995). Preparatory attention can involve both bottom-up and top-down processing and can be indexed using the EEG waveform, contingent negative variation (CNV). The late phase of the CNV indexes cortical activity specific to preparatory processes involved in the analysis

of an imperative stimulus and selection of the appropriate response (Hillyard, 1969; Hillyard et al., 1973). Motor preparation is also related to the CNV. One index of motor preparation that may be superposed onto the CNV is the pre-response Lateralized Readiness Potential (LRP) that is modulated by response conflict (Mathalon et al., 2002). The CNV is most likely the result of activation of a network of prefrontal and parietal association cortices active in judgment and decision making process (Drake et al., 1997; Lai et al., 1997). The dorsolateral prefrontal cortex (DLPFC) is also implicated in CNV generation (Rosahl, Knight, 1995), given that neuronal populations in DLPFC (associated with top-down processing) form recurrent circuits with posterior polymodal association cortices (related to short-term memory and sensory attention bottom-up processes) and premotor areas (Fuster, 2000, 2002). In adults, accuracy in a complex task is predicted by fronto-central CNV amplitude, with correct trials associated with larger, more negative, CNV amplitudes than to error trials (Padilla et al., 2006). Likewise, larger LRP amplitudes may reflect a strategic emphasis on accuracy (versus speed) through an enhancement of response criteria (Wild-Wall et al., 2008).

Evidence for the maturational timeline of the CNV is inconsistent. Early reports indicated that although the amplitude of the CNV was greater in children than adults (Low et al., 1965), age-related changes may cease by age 12 (Low et al., 1966), the age at which Bender et al. (Bender et al., 2005) reported stabilization of late CNV topography. A second group, which studied children 6 to 18 years of age, reported increasing CNV amplitudes at the vertex until age 15 (Tecce, 1971). Studies specifically evaluating the late CNV have reported a linear increase in late CNV with age when comparing 6–7 year-olds, 9–10 year-olds, and 19–23 year-olds (Jonkman, 2006), consistent with another study reporting increasing late CNV in 7 to 17 year-olds (Segalowitz, Davies, 2004).

Performance monitoring also involves bottom-up and top-down processing. Top-down processing is associated with the Error Related Negativity (ERN), a fronto-central maximal electrophysiological marker observed following an incorrect response (Falkenstein et al., 1991; Gehring, 1993; Gehring, Fencsik, 2001; Gehring, Taylor, 2004; Gehring, Willoughby, 2002). Larger ERNs occur with fast-acting systems that inhibit and correct an error as it occurs and with slower acting systems that prolong reaction time (RT) on trials following errors (Gehring, 1993). A second component occurring with much the same latency as ERN is observed during performance monitoring of correct trials that have high levels of response conflict (Vidal et al., 2000). This Correct Related Negativity (CRN) (Carter et al., 1998; Gehring, Fencsik, 2001) reflects a more basic element of conflict following responses than the ERN. Together, the ERN and CRN comprise markers reflecting different constellations of component processes (Bartholow et al., 2005; Luu et al., 2000; Taylor et al., 2007; van Veen, Carter, 2006). Given that greater accuracy in a complex task has been related to less negative CRN amplitudes in healthy adults (Padilla et al., 2006), it is possible that the CRN is more reflective of bottom-up processing, than the top-down ERN (Kenemans, Kahkonen, 2011).

ERN and CRN appear also to have different developmental trajectories. Several studies report greater ERN amplitude with older age across adolescence (Davies et al., 2004; Ladouceur et al., 2004, 2007; Santesso, Segalowitz, 2008; Segalowitz, Davies, 2004; Wiersma et al., 2007). By contrast, CRN amplitudes have been shown to decrease (i.e., less negative) from 7 to 18 years (Davies et al., 2004).

Thus, developmental trajectories have been estimated for both the preparatory attention-related CNV and the performance monitoring ERP components, the ERN and CRN. Both also show evidence of a shift from bottom-up to top-down processing during adolescence and into adulthood but may mature at different rates. To date, however, there have been no

studies evaluating both processes in the same subjects that would permit an evaluation of the relative timing of the development of each set of processes and how they might inter-relate to support efficient cognitive performance. In this cross-sectional study, we examined differences in preparatory attention and response monitoring across adolescence, from age 11 to 18 years, by using a letter discrimination variation of the Eriksen flanker task (Eriksen, Eriksen, 1974; Gehring, Knight, 2000; Padilla et al., 2006). Fronto-central CNV associated with enhanced performance was observed in adults (mean age = 24 years) (Padilla et al., 2006). Based on this observation we tested the hypothesis that larger CNV amplitudes would occur with older adolescence over fronto-central scalp areas and smaller amplitudes over posterior scalp areas. To confirm previous developmental findings, we also tested the hypothesis that larger ERN and smaller CRN amplitude would occur with older age. An additional goal of this study was to examine the relation between response monitoring and preparatory attention potentials. Accordingly, we tested the hypothesis that evidence for more matured response monitoring and preparatory attention mechanisms would be associated with enhanced coordination between these processes in older and more physiologically mature adolescents than in younger and less mature adolescents.

Methods

Participants

The study participants were 38 adolescents (18 girls) recruited from the local community through flyers, school newsletters, word of mouth, and classroom presentations. Subjects were 11 – 18 years (mean $\pm$ SD = 13.89 $\pm$ 2.26). All subjects were given the self-administered rating scale for pubertal development yielding Tanner stages (Carskadon, Acebo, 1993). Pubertal category scores for boys were based on body hair growth, voice change, and facial hair growth. Scores for girls were based on body hair growth, breast development, and menarche. Tanner stages ranged from 1 to 5 (Figure 1), where Tanner 1 = pre-pubertal, Tanner 2 = early pubertal, Tanner 3 = mid-pubertal, Tanner 4 = late pubertal, and Tanner 5 = post-pubertal (Carskadon, Acebo, 1993). Scaled Intelligence Quotient (IQ) from the Peabody Picture Vocabulary Test (Dunn, Dunn, 1997) ranged from 89 to 138 (mean 119.47 $\pm$ 10.56); Socioeconomic Status (SES), provided by parents (Hollingshead, 1975), ranged from 11 to 70 (mean 21.69 $\pm$ 11.21), where lower scores reflected higher SES.

The SRI International Institutional Review Board approved the study protocol. Parents gave written informed consent for their children to participate in the study, and the adolescents gave assent. A structured interview (Kiddie Schedule for Affective Disorders, K-SADS) (Kaufman et al., 1997) was administered to parents and their children by a psychologist or trained researcher to assess current and lifetime history of psychiatric disorders. None of the adolescents met criteria for any DSM-IV disorder. In addition, none of the parents reported diseases of the central nervous system or history of loss of consciousness > 30 minutes in their children. All subjects were right-handed. One child was adopted, and no other child, based on a parental report, endorsed a family history of alcoholism.

Behavioral task

The experimental paradigm, a letter discrimination task (Gehring, Knight, 2000; Padilla et al., 2006), was presented with E-Prime 2.0 software on a 16-inch Dell Ultra Sharp monitor via an NVIDIA GeForce FX 5200 video card. Responses were collected on a Psychology Software Tools (PST) serial response box. Subjects were instructed to respond as quickly and accurately as possible and not to dwell on their mistakes, but rather maintain pace with the experiment. After achieving a performance criterion of above 60% accuracy, subjects completed 5 blocks of 96 trials each with rest periods in between the blocks. A random intertrial interval between 400 and 800 ms was used to increase the likelihood of errors. A

trial was composed of a cue, fixation stimulus, imperative stimulus, and response (Figure 2). The cue, either the word “GREEN” or “RED” printed in black on white background, was presented at time zero for 200 ms. A centrally located fixation cross was presented after the cue for a random length of time between 400 and 800 ms. The imperative stimuli were composed of a centrally presented fixation stimulus and two letters, one letter on each side of the fixation, one colored red and the other green (H + S, S + H, S + S, H + H). The target letter was identified by the color cue. Subjects had as long as 1000 ms to identify the target letter and respond. Subjects responded by pressing a button with their left index finger when the target letter was an H or with their right index finger when the target letter was an S. Response conflict was low when the target letter was on the same side as the response and high when the target letter was presented on the opposite side of the response.

EEG data collection

The EEG and evoked potential data were recorded from a Neuroscan 68-Channel Quick-Cap (El Paso, TX, USA). All electrodes were placed according to the International 10–20 electrode placement standard (Society., 1994). Two external electrooculogram (EOG) electrodes were used to monitor vertical and horizontal eye movements. Raw data were acquired with Neuroscan Synamps 2 amplifiers at a sampling rate of 1 kHz, with a recording bandwidth of DC to 200Hz.

Data analysis

Behavioral data were analyzed using E-Data Aid and Microsoft Excel. Accuracy, defined as the mean proportion correct, was calculated across all blocks. The reaction time (RT) analysis involved calculating the average RT separately for correct and error trials across all blocks. No analysis of low versus high response conflict was presented here due to the limited number of response errors produced in the behavioral task.

EEG data were analyzed offline with Neuroscan 4.5 Scan software in the following order. All EEG channels were re-referenced to linked mastoids. Data were band pass filtered using a zero phase shift Finite Impulse Response (FIR) filter between 0.3 and 30 Hz. Vertical and horizontal ocular artifacts were reduced using the Independent Components Analysis-based Ocular Artifact Reduction algorithms implemented in Neuroscan 4.5. When present, the EKG Noise Reduction algorithm from Neuroscan 4.5 was used to reduce these artifacts. All data were then visually inspected, and any epochs with remaining artifact (muscle, movement, etc.) were manually rejected.

Stimulus locked (imperative stimulus) ERPs (CNV) were evaluated over 1200 ms spanning 400 ms before to 800 ms following the stimulus. Response locked ERP epochs spanned from 200 ms before to 600 ms following the response. All epoched waveforms were then band pass filtered using a zero phase shift FIR filter between 1 and 12 Hz. This filter range was selected because it produced maximal differences between correct and incorrect conditions in both the stimulus and response locked potentials. The first half of the pre-stimulus interval served as baseline for the stimulus locked potentials (–400 to –200 ms) while the response locked potentials were baseline corrected using the first half of the pre-response interval (–200 to –100 ms). Baselines were selected based on the shortest possible interstimulus interval (ISI), to minimize baseline contamination by responses to the previous trial. CRN and ERN (response-locked) were assessed between 0 and 100 ms (Padilla et al., 2011; Riesel et al., 2013) at electrodes Fz, FCz, CPz and Pz and the mean voltages over the interval recorded. Three subjects were excluded from the ERN analysis because they generated consistent eye artifacts following their responses that were not adequately removed by the ocular artifact reduction algorithms and too few error trials remained for analysis after eliminating these epochs. Mean CNV amplitudes for correct and error trials

were averaged between 0 and 100 ms following the stimulus presentation. Because we focused on identifying developmental differences between frontal and more posterior sites, we evaluated mean voltages at 5 midline electrode sites for all CNV conditions: Fz, FCz, CPz, Pz, and POz. Site selection was based on previously published work for each component, and the absence of any lateralization hypotheses.

The CNV is a superposition of slow waves representing both motor preparation and stimulus anticipation. When activity related to motor preparation — the readiness potential — is removed from the CNV, either via mathematical or experimental manipulations, the remaining waveform, the stimulus preceding negativity, can be observed (Brunia, 1988; Brunia, Damen, 1988; Damen, Brunia, 1987). Because we did not segregate activity related to motor preparation, we used the term “CNV” to identify the slow wave during preparatory attention intervals.

The CNV is traditionally measured during a long time window between the cue and the imperative stimulus; however, in this version of the letter discrimination task, this epoch was shortened considerably (random ISI of 400–800 ms) to increase the number of errors for our ERN analysis. Thus, our stimulus locked epochs began 400 ms before the presentation of the stimulus. Further, we assessed the time epoch immediately following the stimulus and immediately preceding the peak of the potential (0 to 100 ms following the presentation of the stimulus) because previous work indicated that this time window shows the largest amplitude differences between preparatory attention conditions in this task (Padilla et al., 2006). We also reasoned that this epoch of preparatory attention would be especially sensitive to the influence of response monitoring processes because early attention effects have been observed within this time window (Hillyard et al., 1995).

Statistical analysis

The statistical analysis of the ERP components was conducted on the mean ERP amplitudes (i.e., mean voltage over each defined window). The mean amplitudes were calculated for each subject using the same electrode location/s and time window across all subjects, and these are specified in the Evoked Potentials section of the Results.

Relations between accuracy, RTs, CNV values preceding correct and error trials at electrodes Fz, FCz, CPz, Pz, and POz and age were assessed with Pearson correlation analyses; relations with Tanner Stage were assessed with Spearman’s rank order correlations. The same analyses were applied to CRN data in the response-locked averages to correct responses and ERN data in the response-locked averages to error responses. For the correlations between age or Tanner score and the amplitudes of the negative ERP components, positive correlations would indicate the components are smaller (i.e., less negative) with older age or more advanced Tanner stage. Probabilities were corrected for multiple comparisons using a false discovery rate procedure (<http://sdmproject.com/utilities/?show=FDR>).

For CNV amplitudes, analyses of variance (ANOVAs) were conducted with performance (error vs. correct) and electrode site as within-subjects repeated-measures. To assess differences of the age regression slopes between different electrode sites, ANOVAs were conducted for the correct CNV, error CNV, CRN and ERN amplitudes with site as a repeated measure and age as a covariate. Slopes were considered different if the age x site interaction term was significant. All p-values were Huynh–Feldt corrected where appropriate, and presented with corrected degrees of freedom.

Results

Behavior

Accuracy and error type count—The overall accuracy for all subjects was 0.83 ± 0.07 . Accuracy was significantly and positively correlated with age [$r = .425$, $p = .008$] and Tanner score [$\rho = .404$, $p = .012$]. Subjects made more errors of commission (50.5 ± 32.1) than omission (30.7 ± 24.8) [$t(37) = 2.67$, $p = .011$]. Across the whole data set there were an average of 398.8 ± 34.7 correct responses and 50.5 ± 32.2 errors.

Reaction Time (RT)—RTs for errors (629.7 ± 97.2 ms) were longer than for correct responses (606.6 ± 79.4 ms) [$t(37) = 4.06$, $p < .001$]. Neither age nor Tanner stage correlated with error RT or correct RT.

Post-error slowing—RT for correct trials following an error (641.1 ± 88.0 ms) was slower than for correct trials following a correct response (603.8 ± 79.3 ms) [$t(37) = 7.24$, $p < .001$]. Neither age nor Tanner stage correlated with post-error RT.

Evoked Potentials

Preparatory attention: CNV prior to correct responses and prior to errors—

There was no difference in CNV amplitude prior to correct and error responses [$p = .843$] and no site by performance (error, correct) interaction [$p = .913$]. There was a significant effect of electrode site [$F(1.43, 54.45) = 20.72$, $p < .001$] with the component larger at more posterior sites. For CNV amplitudes prior to correct responses the slopes of the age regression varied with electrode site as indicated by a significant age-by-site interaction [$F(1.5, 53.1) = 4.66$, $p = .020$], with older age associated with smaller posterior CNV amplitudes (Figure 3A) [CPz, $r = .321$, $p = .042$; Pz, $r = .509$, $p = .005$; POz, $r = .422$, $p = .01$]. Higher Tanner stage was also associated with smaller posterior CNV amplitudes prior to correct responses [CPz, $\rho = .379$, $p = .019$; Pz, $\rho = .516$, $p = .001$; POz, $\rho = .482$, $p = .002$]. Figure 3B displays the prominent posterior scalp topography of the CNV prior to correct responses for the 20 younger subjects (age 11 to 13 years) not present in the 18 older subjects (age 14 to 18 years).

For CNV amplitude prior to errors, the age $\times$ site interaction was not significant [$p = .179$]; however, amplitude, particularly at posterior sites, did again display significant correlations with age [Pz, $r = .441$, $p = .015$; POz, $r = .366$, $p = .03$], and Tanner stage [FCz, $\rho = .332$, $p = .041$; CPz, $\rho = .397$, $p = .014$; Pz, $\rho = .493$, $p = .002$; POz, $\rho = .416$, $p = .009$]. Figure 3B displays the prominent posterior scalp topography of the CNV prior to correct responses for the 20 younger subjects (age 11 to 13 years) not present in the 18 older subjects (age 14 to 18 years).

Response monitoring: ERN and CRN—ERN amplitude did not display a significant effect of site [$p = .194$] but did show a significant site $\times$ age interaction [$F(1.8, 66.3) = 4.12$, $p = .023$]. Older age was significantly correlated with larger ERN amplitude at Fz [$r = -.398$, $p = .028$] (see Figure 4A) but not at the more posterior sites. Higher Tanner stage did not predict ERN amplitude at any site. Figure 4B displays the greater amplitude and broader frontal scalp topography of the ERN for the older subjects in the cohort compared with the younger subjects.

CRN amplitude did not display significant electrode site [$p = .134$] or age-by-site [$p = .102$] effects. Older age was correlated with smaller (more positive) CRN amplitude at FCz [$r = .329$, $p = .043$] (Figure 5A), CPz [$r = .356$, $p = .043$] and Pz [$r = .304$, $p = .043$]. Higher

Tanner stage did not predict CRN amplitude at any site. Figure 5B displays the lesser frontal CRN in the older subjects compared with the younger subjects.

Relations between preparatory attention and response monitoring ERPs and behavior

Correlations with accuracy and RT were evaluated with ERP amplitudes at sites at which the maximal age-relations were displayed: CNV at Pz, ERN at Fz, and CRN at FCz. Greater accuracy correlated with larger (more negative) ERN amplitudes at Fz [$r = -.298$, $p = .034$] and smaller (more positive) CNV amplitudes prior to correct responses at Pz [$r = .383$, $p = .009$]. Faster correct RTs correlated with smaller CRN amplitudes at FCz [$r = -.359$, $p = .013$]. Faster error RTs correlated with more negative ERN amplitudes at Fz [$r = .480$, $p = .001$].

Less negative CRN amplitudes were associated with less negative CNV amplitudes prior to correct responses [$r = .490$, $p = .001$] and prior to errors [$r = .505$, $p = .006$]. By contrast, the ERN was not significantly correlated with CNV amplitude at any site for either trial type.

The posterior CNV and frontal ERN amplitudes displayed an opposite pattern with age. While the frontal ERN amplitude was larger with older age (Figure 6A), the posterior CNV amplitude was smaller with older age (Figure 6B).

Discussion

The three working hypotheses for this study were largely supported. CNV amplitudes over posterior scalp were smaller with advancing age and Tanner stage; however the hypothesized increase in CNV over frontal scalp with older age was not observed. This pattern was seen for CNV prior to both correct and error responses. The hypothesis that ERN would become larger and CRN smaller with advancing age was supported; however, the ERN effect was limited to frontal scalp. Finally, mature performance, indicated by greater accuracy with older age, was associated with both larger frontal ERN and smaller posterior CNV. That the fronto-central CNV associated with enhanced performance in adults (Padilla et al., 2006) did not demonstrate development across the age range studied suggests room for further cognitive development in the age and Tanner range studied. These data support the position that the neural mechanisms underlying the relation between enhanced CNV and better performance in adults are only partially developed by late adolescence.

ERN and CRN components are thought to be generated in the anterior (Donamayor et al., 2012; Keil et al., 2010; Miltner et al., 2003) or posterior (Dhar et al., 2011) regions of the cingulate cortex, although a network of other areas (Donamayor et al., 2012) has also been implicated in their generation. The ERN reflects well-developed error monitoring circuitry (Wiersema et al., 2007), evidenced even in the younger adolescents in the present sample, despite other reports indicating a later development (Ladouceur et al., 2004, 2007; Santesso, Segalowitz, 2008; Segalowitz, Davies, 2004). The difference between amplitudes of the CRN and ERN provides an index of the functional integrity of the error-monitoring system (Falkenstein, 2004). The ERN being larger and the CRN being smaller in older adolescents would thus seem to index a progression of brain development to permit more refined error monitoring (Endrass et al., 2012). The developmental trajectory we infer from the age distribution of the response monitoring potentials suggests that frontal processes associated with monitoring errors mature earlier than those associated with preparatory attention. This pattern is consistent with earlier development of the anterior cingulate cortex, required for error monitoring relative to the dorsolateral prefrontal cortex (Giedd, 2004), which in turn is required for preparatory attentional allocation. However, it should be recognized that there

are inherent limitations in being able to infer regional brain differences from scalp-recorded EEG.

The test design permitted an evaluation of the effect of age on the relations among CRN, ERN and CNV amplitudes at frontal and posterior scalp sites. Age-related differences in CRN amplitudes at posterior sites were associated with age-related differences in CNV amplitudes also at posterior sites. Thus, both posterior-oriented components, showed consistent age differences, indicative of maturation. The lack of an association between frontal CNV and frontal ERN amplitudes with age (Davies et al., 2001; Stemmer et al., 2004) is further evidence of the developmental dissociation of these processes. With age and maturation, better performance was associated with inhibition of posterior CRN (faster RT), inhibition of posterior CNV (improved accuracy), and enhancement of frontal ERN (improved accuracy), with a movement to top-down processing resulting in better performance.

There was a trend for faster correct reaction times with older age. Although two studies have reported faster reaction times in older adolescence (Ladouceur et al., 2004, 2007; Segalowitz, Davies, 2004) the change in RT with age displayed an asymptote at about age 12–13 years. Consistent with previous studies of children and adolescents (Ladouceur et al., 2004, 2007; Santesso, Segalowitz, 2008; Segalowitz, Davies, 2004; Wiersma et al., 2007), our data show post-error RT slowing with no differences in magnitude of the slowing across the age-range that we studied. Whereas adults make more errors of omission (missed responses) than commission (incorrect responses) (Padilla et al., 2011), our cohort of adolescents made more errors of commission than omission, indicating that our adolescents were poorly able to inhibit prepotent responses. Although it is typically the case that error trials have a faster reaction time than correct responses, the opposite pattern was reported in the present study. However this apparent anomaly may relate to the specific task used, as other studies using the same task in adults found the same result of faster responses to correct responses than to errors. (Padilla et al., 2011; Padilla et al., 2006).

The study is limited in terms of the limited range in age and Tanner stage, sample size and the cross-sectional nature of the study. Ideally, more subjects would have been studied at Tanner stage 1 to obtain a clearer estimate of pre-pubertal performance and neurophysiological correlates of processing prior to the onset of adolescence. Likewise, the addition of a group of post-adolescent young adults would have provided a comparison point reflective of complete development. Verification of cross-sectional findings of developmental differences requires longitudinal study to derive true developmental trajectories.

The findings suggest that development of preparatory attention and response monitoring follows a posterior-anterior scalp gradient. It is possible that the anterior, top-down attention network that enhances task relevant events does not come online until late adolescence, and that youth compensate by engaging a posterior, bottom-up attention network, which improves task performance by inhibiting task irrelevant events (Padilla et al., 2006). If this is the case then it would appear that this posterior network appears to continue maturing into late adolescence and, until mature, is less efficient, resulting in a higher proportion of errors of commission than omission, which is opposite to the adult pattern (Padilla et al., 2011). Not until early adulthood, when both the processes involved in inhibiting irrelevant information (posterior) and monitoring responses (medial frontal) reach greater maturity and presumably are better coordinated, can enhanced preparatory attention effects be observed at anterior sites in conditions that require greater control. The apparent dissociation of maturation of preparatory attention and response monitoring is likely to produce less effective attentional regulation during adolescence than is possible later in adulthood.

Acknowledgments

The authors wish to acknowledge the research personnel who assisted with data collection for this study: Lindsay Hoffman, Benjamin Mayer, Amanda Wagstaff, and Sharon Turlington. Research was supported by NIH grants AA005965; AA017168; AA017320; AA020565; AA012388. The funding sources had no direct involvement in any stage of the study.

References

- Acik A, Sarwary A, Schultze-Kraft R, Onat S, Konig P. Developmental Changes in Natural Viewing Behavior: Bottom-Up and Top-Down Differences between Children, Young Adults and Older Adults. *Front Psych*. 2010; 1:207.
- Andersson M, Llera JE, Rimol LM, Hugdahl K. Using dichotic listening to study bottom-up and top-down processing in children and adults. *Child Neuropsych*. 2008; 14:470–479.
- Bartholow BD, Pearson MA, Dickter CL, Sher KJ, Fabiani M, Gratton G. Strategic control and medial frontal negativity: beyond errors and response conflict. *Psychophys*. 2005; 42:33–42.
- Bava S, Tapert SF. Adolescent brain development and the risk for alcohol and other drug problems. *Neuropsych Rev*. 2010; 20:398–413.
- Bender S, Weisbrod M, Bornfleth H, Resch F, Oelkers-Ax R. How do children prepare to react? Imaging maturation of motor preparation and stimulus anticipation by late contingent negative variation. *Neuro Image*. 2005; 27:737–752. [PubMed: 16027009]
- Bitan T, Cheon J, Lu D, Burman DD, Booth JR. Developmental increase in top-down and bottom-up processing in a phonological task: an effective connectivity, fMRI study. *J Cog Neurosci*. 2009; 21:1135–1145.
- Brain Development Cooperative Group. Total regional brain volumes in a population-based normative sample from 4 to 18 years: the NIH MRI Study of Normal Brain Development. *Cereb Cortex*. 2012; 22:1–12. [PubMed: 21613470]
- Brunia CH. Movement and stimulus preceding negativity. *Biol Psych*. 1988; 26:165–178.
- Brunia CH, Damen EJ. Distribution of slow brain potentials related to motor preparation and stimulus anticipation in a time estimation task. *Electroenceph Clin Neurophys*. 1988; 69:234–243.
- Carskadon MA, Acebo C. A self-administered rating scale for pubertal development. *J Adol Health*. 1993; 14:190–195.
- Carter CS, Braver TS, Barch DM, Botvinick MM, Noll D, Cohen JD. Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*. 1998; 280:747–749. [PubMed: 9563953]
- Casey BJ, Jones RM. Neurobiology of the adolescent brain and behavior: implications for substance use disorders. *J Am Acad Child Adol Psych*. 2010; 49:1189–1201. quiz 1285.
- Corbetta M, Shulman GL. Control of goal-directed and stimulus-driven attention in the brain. *Nat Rev Neurosci*. 2002; 3:201–215. [PubMed: 11994752]
- Courchesne E, Chisum HJ, Townsend J, Cowles A, Covington J, Egaas B, et al. Normal brain development and aging: quantitative analysis at in vivo MR imaging in healthy volunteers. *Radiol*. 2000; 216:672–682.
- Damen EJ, Brunia CH. Precentral potential shifts related to motor preparation and stimulus anticipation: a replication. *Electroenceph Clin Neurophys (Supplement)*. 1987; 40:13–16.
- Davies PL, Segalowitz SJ, Dywan J, Pailing PE. Error-negativity and positivity as they relate to other ERP indices of attentional control and stimulus processing. *Biol Psychol*. 2001; 56:191–206. [PubMed: 11399350]
- Davies PL, Segalowitz SJ, Gavin WJ. Development of error-monitoring event-related potentials in adolescents. *Ann NY Acad Sci*. 2004; 1021:324–328. [PubMed: 15251904]
- Dhar M, Wiersma JR, Pourtois G. Cascade of neural events leading from error commission to subsequent awareness revealed using EEG source imaging. *PloS One*. 2011; 6:e19578. [PubMed: 21573173]
- Donamayor N, Heilbronner U, Munte TF. Coupling electrophysiological and hemodynamic responses to errors. *Hum Brain Map*. 2012; 33:1621–1633.

- Donnelly N, Cave K, Greenway R, Hadwin JA, Stevenson J, Sonuga-Barke E. Visual search in children and adults: top-down and bottom-up mechanisms. *Quart J Exper Psych.* 2007; 60:120–136.
- Drake ME Jr, Weate SJ, Newell SA. Contingent negative variation in epilepsy. *Seizure.* 1997; 6:297–301. [PubMed: 9304721]
- Dunn, LM.; Dunn, ES. Peabody Picture Vocabulary Test. 3. American Guidance Service; Circle Pines, MN: 1997.
- Endrass T, Klawohn J, Gruetzmann R, Ischebeck M, Kathmann N. Response-related negativities following correct and incorrect responses: evidence from a temporospatial principal component analysis. *Psychophys.* 2012; 49:733–743.
- Eriksen BA, Eriksen CW. Effects of noise letters upon the identification of target letter in a nonsearch task. *Percept & Psychophys.* 1974; 16:143–149.
- Falkenstein, M. ERP correlates of erroneous performance. In: Ullsperger, M.; Falkenstein, M., editors. *Errors, conflicts and the brain. Current opinions on performance monitoring.* Leipzig: Max Plank Institut for Kog und Neurowiss; 2004. p. 5-14.
- Falkenstein M, Hohnsbein J, Hoormann J, Blanke L. Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. *Electroenceph Clin Neurophys.* 1991; 78:447–455.
- Fuster JM. Executive frontal functions. *Experimental Brain Research.* 2000; 133:66–70. [PubMed: 10933211]
- Fuster JM. Frontal lobe and cognitive development. *J Neurocyt.* 2002; 31:373–385.
- Gehring, WJ. The error-related negativity: Evidence for a neural mechanism for error-related processing. University of Illinois; Champaign: 1993. p. 296
- Gehring WJ, Fencsik DE. Functions of the medial frontal cortex in the processing of conflict and errors. *J Neurosci.* 2001; 21:9430–9437. [PubMed: 11717376]
- Gehring WJ, Knight RT. Prefrontal-cingulate interactions in action monitoring. *Nature Neurosci.* 2000; 3:516–520. [PubMed: 10769394]
- Gehring WJ, Taylor SF. When the going gets tough, the cingulate gets going. *Nature Neurosci.* 2004; 7:1285–1287. [PubMed: 15643434]
- Gehring WJ, Willoughby AR. The medial frontal cortex and the rapid processing of monetary gains and losses. *Science.* 2002; 295:2279–2282. [PubMed: 11910116]
- Giedd JN. Structural magnetic resonance imaging of the adolescent brain. *Ann NY Acad Sci.* 2004; 1021:77–85. [PubMed: 15251877]
- Giedd JN, Blumenthal J, Jeffries NO, Castellanos FX, Liu H, Zijdenbos A, et al. Brain development during childhood and adolescence: a longitudinal MRI study. *Nat Neurosci.* 1999a; 2:861–863. [PubMed: 10491603]
- Giedd JN, Blumenthal J, Jeffries NO, Rajapakse JC, Vaituzis AC, Liu H, et al. Development of the human corpus callosum during childhood and adolescence: a longitudinal MRI study. *Prog Neuropsychopharm Biol Psych.* 1999b; 23:571–588.
- Giedd JN, Stockman M, Weddle C, Liverpool M, Alexander-Bloch A, Wallace GL, et al. Anatomic magnetic resonance imaging of the developing child and adolescent brain and effects of genetic variation. *Neuropsych Rev.* 2010; 20:349–361.
- Hillyard SA. Relationships between the contingent negative variation (CNV) and reaction time. *Physiol & Behav.* 1969; 4:351–357.
- Hillyard SA, Hink RF, Schwent VL, Picton TW. Electrical signs of selective attention in the human brain. *Science.* 1973; 182:177–180. [PubMed: 4730062]
- Hillyard, SA.; Mangun, GR.; Woldorff, MG.; Luck, SJ. Neural systems mediating selective attention. In: Gazzaniga, MS., editor. *The Cognitive Neurosciences.* Cambridge MA: MIT Press; 1995. p. 665-681.
- Hollingshead, A. Four-Factor Index of Social Status. Yale University, Department of Sociology; New Haven, CT: 1975.
- Jernigan TL, Trauner DA, Hesselink JR, Tallal PA. Maturation of human cerebrum observed in vivo during adolescence. *Brain.* 1991; 114:2037–2049. [PubMed: 1933232]

- Jonkman LM. The development of preparation, conflict monitoring and inhibition from early childhood to young adulthood: a Go/Nogo ERP study. *Brain Res.* 2006; 1097:181–193. [PubMed: 16729977]
- Karatekin C. Development of attentional allocation in the dual task paradigm. *International J Psychophys.* 2004; 52:7–21.
- Kaufman J, Birmaher B, Brent D, Rao U, Flynn C, Moreci P, et al. Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL): initial reliability and validity data. *J Am Acad Child Adol Psych.* 1997; 36:980–988.
- Keil J, Weisz N, Paul-Jordanov I, Wienbruch C. Localization of the magnetic equivalent of the ERN and induced oscillatory brain activity. *Neuro Image.* 2010; 51:404–411. [PubMed: 20149884]
- Kenemans JL, Kahkonen S. How human electrophysiology informs psychopharmacology: from bottom-up driven processing to top-down control. *Neuropsychopharmacol.* 2011; 36:26–51.
- Ladouceur CD, Dahl RE, Carter CS. ERP correlates of action monitoring in adolescence. *Ann NY Acad Sci.* 2004; 1021:329–336. [PubMed: 15251905]
- Ladouceur CD, Dahl RE, Carter CS. Development of action monitoring through adolescence into adulthood: ERP and source localization. *Develop Sci.* 2007; 10:874–891.
- Lai C, Ikeda A, Terada K, Nagamine T, Honda M, Xu X, et al. Event-related potentials associated with judgment: comparison of S1- and S2-choice conditions in a contingent negative variation (CNV) paradigm. *J Clin Neurophys.* 1997; 14:394–405.
- Lenroot RK, Gogtay N, Greenstein DK, Wells EM, Wallace GL, Clasen LS, et al. Sexual dimorphism of brain developmental trajectories during childhood and adolescence. *Neuro Image.* 2007; 36:1065–1073. [PubMed: 17513132]
- Low MD, Borda RP, Frost JD, Kellaway P. Surface negative, slow potential shift associated with conditioning in man. *Neurol.* 1966; 16:771.
- Low MD, Borda RP, Frost JD, Kellaway P, Gol A. General scientific session in conjunction with section on Neurology of Behavior. *Neurol.* 1965; 15:279.
- Luna B, Thulborn KR, Munoz DP, Merriam EP, Garver KE, Minshew NJ, et al. Maturation of widely distributed brain function subserves cognitive development. *Neuro Image.* 2001; 13:786–793. [PubMed: 11304075]
- Luu P, Collins P, Tucker DM. Mood, personality, and self-monitoring: negative affect and emotionality in relation to frontal lobe mechanisms of error monitoring. *J Exp Psych: Genl.* 2000; 129:43–60.
- Mathalon DH, Fedor M, Faustman WO, Gray M, Askari N, Ford JM. Response-monitoring dysfunction in schizophrenia: an event-related brain potential study. *J Abnorm Psychol.* 2002; 111:22–41. [PubMed: 11866176]
- Miltner WH, Lemke U, Weiss T, Holroyd C, Scheffers MK, Coles MG. Implementation of error-processing in the human anterior cingulate cortex: a source analysis of the magnetic equivalent of the error-related negativity. *Biol Psych.* 2003; 64:157–166.
- Padilla ML, Colrain IM, Sullivan EV, Mayer BZ, Turlington SR, Hoffman LR, et al. Electrophysiological evidence of enhanced performance monitoring in recently abstinent alcoholic men. *Psychopharm.* 2011; 213:81–91.
- Padilla ML, Wood RA, Hale LA, Knight RT. Lapses in a prefrontal-extrastriate preparatory attention network predict mistakes. *J Cog Neurosci.* 2006; 18:1477–1487.
- Paus T. Growth of white matter in the adolescent brain: myelin or axon? *Brain & Cog.* 2010; 72:26–35.
- Pfefferbaum A, Mathalon DH, Sullivan EV, Rawles JM, Zipursky RB, Lim KO. A quantitative magnetic resonance imaging study of changes in brain morphology from infancy to late adulthood. *Arch Neurol.* 1994; 51:874–887. [PubMed: 8080387]
- Posner MI, Petersen SE. The attention system of the human brain. *Annl Rev Neurosci.* 1990; 13:25–42. [PubMed: 2183676]
- Raznahan A, Lerch JP, Lee N, Greenstein D, Wallace GL, Stockman M, et al. Patterns of coordinated anatomical change in human cortical development: a longitudinal neuroimaging study of maturational coupling. *Neuron.* 2011a; 72:873–884. [PubMed: 22153381]

- Raznahan A, Shaw P, Lalonde F, Stockman M, Wallace GL, Greenstein D, et al. How does your cortex grow? *J Neurosci*. 2011b; 31:7174–7177. [PubMed: 21562281]
- Reiss AL, Abrams MT, Singer HS, Ross JL, Denckla MB. Brain development, gender and IQ in children. A volumetric imaging study. *Brain*. 1996; 119:1763–1774. [PubMed: 8931596]
- Riesel A, Weinberg A, Endrass T, Meyer A, Hajcak G. The ERN is the ERN is the ERN? Convergent validity of error-related brain activity across different tasks. *Biol Psychol*. 2013; 93:377–385. [PubMed: 23607999]
- Rosahl SK, Knight RT. Role of prefrontal cortex in generation of the contingent negative variation. *Cereb Cort*. 1995; 5:123–134.
- Santesso DL, Segalowitz SJ. Developmental differences in error-related ERPs in middle- to late-adolescent males. *Devel Psych*. 2008; 44:205–217.
- Schmithorst VJ, Yuan W. White matter development during adolescence as shown by diffusion MRI. *Brain & Cog*. 2010; 72:16–25.
- Segalowitz SJ, Davies PL. Charting the maturation of the frontal lobe: an electrophysiological strategy. *Brain & Cog*. 2004; 55:116–133.
- Shaw P, Kabani NJ, Lerch JP, Eckstrand K, Lenroot R, Gogtay N, et al. Neurodevelopmental trajectories of the human cerebral cortex. *J Neurosci*. 2008; 28:3586–3594. [PubMed: 18385317]
- Society AE. Guidelines for standard electrode position nomenclature. *J Clin Neurophys*. 1994; 11:111–113.
- Sowell ER, Thompson PM, Toga AW. Mapping changes in the human cortex throughout the span of life. *Neuroscientist*. 2004; 10:372–392. [PubMed: 15271264]
- Sowell ER, Trauner DA, Gamst A, Jernigan TL. Development of cortical and subcortical brain structures in childhood and adolescence: a structural MRI study. *Devel Med & Child Neurol*. 2002; 44:4–16. [PubMed: 11811649]
- Spronk M, Jonkman LM. Electrophysiological evidence for different effects of working memory load on interference control in adolescents than adults. *Int J Psychophys*. 2012; 83:24–35.
- Steen RG, Ogg RJ, Reddick WE, Kingsley PB. Age-related changes in the pediatric brain: quantitative MR evidence of maturational changes during adolescence. *AmJ Neuroradiol-AJNR*. 1997; 18:819–828.
- Stemmer B, Segalowitz SJ, Witzke W, Schonle PW. Error detection in patients with lesions to the medial prefrontal cortex: an ERP study. *Neuropsychologia*. 2004; 42:118–130. [PubMed: 14615082]
- Stiles J, Jernigan TL. The basics of brain development. *Neuropsychol Rev*. 2010; 20:327–348. [PubMed: 21042938]
- Sullivan EV, Pfefferbaum A, Rohlfing T, Baker FC, Padilla ML, Colrain IM. Developmental change in regional brain structure over 7 months in early adolescence: comparison of approaches for longitudinal atlas-based parcellation. *Neuro Image*. 2011; 57:214–224. [PubMed: 21511039]
- Takio F, Koivisto M, Jokiranta L, Rashid F, Kallio J, Tuominen T, et al. The effect of age on attentional modulation in Dichotic listening. *Devel Neuropsych*. 2009; 34:225–239.
- Taylor SF, Stern ER, Gehring WJ. Neural systems for error monitoring: recent findings and theoretical perspectives. *Neuroscientist*. 2007; 13:160–172. [PubMed: 17404376]
- Tecce JJ. Contingent negative variation and individual differences. A new approach in brain research. *Arch Gen Psychiatry*. 1971; 24:1–16. [PubMed: 4923214]
- van Veen V, Carter CS. Error detection, correction, and prevention in the brain: a brief review of data and theories. *Clinical EEG and Neurosci*. 2006; 37:330–335.
- Vidal F, Hasbroucq T, Grapperon J, Bonnet M. Is the ‘error negativity’ specific to errors? *Biol Psych*. 2000; 51:109–128.
- Wiersema JR, van der Meere JJ, Roeyers H. Developmental changes in error monitoring: an event-related potential study. *Neuropsychologia*. 2007; 45:1649–1657. [PubMed: 17303199]
- Wild-Wall N, Falkenstein M, Hohnsbein J. Flanker interference in young and older participants as reflected in event-related potentials. *Brain Res*. 2008; 1211:72–84. [PubMed: 18433737]
- Yakovlev, P.; Lecours, AR. The myelogenetic cycles of regional maturation in the brain. Oxford: Blackwell; 1967.

Highlights

- EEG markers of preparatory attention reflect development, but anterior scalp markers seen in adults are not present even in the older youth
- EEG markers of response monitoring reflect development
- Age-related changes in EEG markers of preparatory attention and response monitoring are correlated with age-related changes in performance accuracy.

Figure 1. Age and Tanner Stages

Distribution of subject ages by Tanner stage. Boys in blue diamonds, and girls in solid pink diamonds.

Figure 2. Task description

Outline of the sequence of events for our modified version of the letter discrimination task.

Figure 3. CNV waveforms

A: Stimulus-locked correct and incorrect error waveforms are plotted at electrode CPz. The adolescents were divided into younger (11–13 years) and older (14–18 years) groups for illustrative purposes. Correct waveforms are plotted in solid green for younger adolescents (mean±SD number of correct responses: 386.85 ± 34.36) and in dotted green for older adolescents (mean±SD number of correct responses: 412.00 ± 30.90). Error waveforms are plotted in solid red for younger adolescents (mean±SD number of error responses: 56.80 ± 36.52) and in dotted red for older adolescents (mean±SD number of error responses: 43.50 ± 25.65). Vertical line marks measurement onset of the imperative stimulus. B: Scalp topography of the CNV in the young (left panel) and older (right panel) subjects. The color scale in each map reflects mean voltage in the grand average between 1.25 (red) and –2.5 (dark blue) microvolts.

Figure 4. ERN waveforms

A: Response-locked incorrect error waveforms are plotted at electrode Fz. The adolescents were divided into younger (11–13 years) and older (14–18 years) groups for illustrative purposes. Waveforms are plotted in solid red for younger adolescents (mean±SD number of error responses: 56.80 ± 36.52) and in dotted red for older adolescents (mean±SD number of error responses: 43.50 ± 25.65). B: Scalp topography of the ERN in the young (left panel) and older (right panel) subjects. The color scale in each map reflects mean voltage in the grand average between 1.0 (red) and –3.0 (dark blue) microvolts.

Figure 5. CRN waveforms

A: Response-locked correct waveforms are plotted at electrode FCz. The adolescents were divided into younger (11–13 years) and older (14–18 years) groups for illustrative purposes. Waveforms are plotted in solid green for younger adolescents and in dotted green for older adolescents. B: Scalp topography of the CRN in the young (left panel) and older (right panel) subjects. The color scale in each map reflects mean voltage in the grand average between 2.0 (red) and –3.0 (dark blue) microvolts.

Figure 6. CNV and ERN amplitudes and age

A. ERN (red circles) and CNV (green squares) amplitudes at Fz plotted against age in years. B. ERN (red circles) and CNV (green squares) amplitudes at Pz plotted against age in years. The solid lines represent the linear regressions age and component amplitude. Linear regressions are significant ($p < .05$) for ERN amplitude at Fz over age and for CNV amplitude at Pz over age.