The Development of the Error Negativity in Children and Adolescents

Sidney J. Segalowitz[1], Patricia L. Davies[2], Diane Santesso[1], William J. Gavin[3], and Louis A. Schmidt[4]
[1] *Brock University, St. Catharines, Canada,* [2] *Colorado State University, Fort Collins, U.S.A.*
[3] *University of Colorado, Boulder, U.S.A.,* [4] *McMaster University, Hamilton, Canada*

We present data from two studies with children showing that the Ne increases in amplitude with development through adolescence while the Pe is relatively stable. In the first study with 10-year-olds, the Ne amplitude was related to obsessive-compulsive behaviors and to socialization. In the second study with 7- to 18-year-olds, the Ne amplitude increased with age but showed a pattern consistent with pubertal effects. The results are discussed in the context of the Ne being associated with the anterior cingulate cortex and the dopaminergic system, and the implications of these results for child development.

Introduction

The error-monitoring ERP components Ne (or ERN) and Pe seem sensitive in special populations to personality or cognitive factors that affect the individual's ability or desire to closely monitor his or her performance (Dywan et al, this volume; Gehring et al., 2000; Mathalon et al., 2002; Pailing et al., 2002). Similarly, specific motivating instructions influence these components (Pailing and Segalowitz, in press). One may similarly speculate that the development of brain systems which are associated with successful performance monitoring would be reflected in the developmental study of the Ne/Pe paradigm. Error and performance monitoring involve attentional control and working memory mechanisms associated with executive functions, and therefore implicate regions of prefrontal cortex and the anterior cingulate cortex (ACC), a transitional region with major tracts connecting the limbic system with the prefrontal cortex (Van Hoesen et al., 1993; Vogt, 1993). The ACC becomes activated during 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 et al., 1995). There is much evidence for late maturation of executive functions during childhood (and their lack in the case of developmental disorders), and their association with functions of the prefrontal cortex (PFC), the ACC and the dopaminergic system. The Ne has been specifically linked to ACC and dopamine systems (and the Pe less clearly so), and therefore these components may be an important window on this development.

Recent reports present evidence of anatomical and physiological maturation of the ACC into early adulthood (Cunningham et al., 2002), as well as increased activation of the ACC from childhood to young adulthood (Adleman et al., 2002; Van Bogaert et al., 1998). This parallels the relatively late maturation of the PFC, shown with evidence from anatomical (Greenough et al., 1987), physiological (Rakic et al., 1986), behavioral (Davies and Rose, 1999; Stuss, 1992), clinical (Kolb et al., 1992), and electrophysiological (Segalowitz et al., 1992) studies. Both the PFC and ACC mediate executive functions that develop into young adulthood (Devinsky et al., 1995). Furthermore, increases into early adulthood in dopamine metabolism and dopaminergic connections, which are profuse in the PFC and especially in the ACC, have been observed (Benes

et al., 1996; Kalsbeek et al., 1988; Lambe et al., 2000; Rosenberg and Lewis, 1995; Verney et al., 1982). Holroyd and Coles (2002) recently postulated that it is the mesencephalic dopamine system that specifically is involved in the generation of the Ne. Given this model and the continued maturation of the ACC, PFC, and domaminergic systems into young adulthood, the production of the Ne may also show evidence of continued maturation through adolescence.

Goals of the studies presented in this chapter

Our initial studies on the Ne and Pe in children were designed to answer two primary questions: (1) What is the developmental course of error-monitoring ERP components? and (2) what implications does this have for developmental psychology? In this paper we will present a summary of the data from our initial studies in this regard. In both studies, the Ne and Pe were recorded during a standard letter-string 480-trial visual flanker task (80 trials each of stimuli SSSSS and HHHHH, and 160 trials each of SSHSS and HHSHH; Eriksen and Eriksen, 1974). Baseline was always taken at 400 to 600 ms before the response, thus permitting measurement of the Ne and Pe independent of the positivity preceding the Ne. When early and late Pe were present, results reflect the late Pe.

Comparison of children's and adults' Ne and Pe

In our first study, we compared the Ne and Pe at four midline scalp locations (Fz, FCz, Cz, Pz) in a sample of thirty-nine 10-year-old children and twenty-seven young adults aged 18-30 years (Santesso et al., 2003b). As expected, for both age groups, response times were faster for errors compared with correct trials [$F(1, 61) = 61.3$, $p < .01$] and for incongruent compared with congruent error trials [$F(1, 61) = 6.8$, $p = .01$]. Both age groups made more errors during incongruent compared with congruent trials [$F(1, 61) = 71.0$, $p < .01$]. The children consistently produced a measurable Ne and its amplitude was maximal at FCz and Cz for both children and adults. However, the children's Ne was significantly smaller in amplitude at Cz compared with adults as measured by either the peak-to-peak or baseline-to-peak amplitude from the preceding positivity to the Ne (Cz: $t(63) = 2.3$, $p = .02$) while peak-to-peak amplitude comparisons provided a trend in the same direction at FCz ($t(62) = 1.8$, $p = .07$; see Figure 1). The Pe was maximal at FCz for the children with only slightly lower values at Fz and Cz but considerably lower at Pz. In the adults the Pe was maximal at Cz with only slight reductions at FCz and Pz. The Pe was significantly greater for the children than adults at Fz, while the adults had greater Pe at Cz and Pz.

Relation to Ne and Personality Traits in children 10-years-old

As previously discussed, the ACC is involved in modulating attention, response selection and motor control. This region also contributes to emotional regulation and guiding and/or constraining complex social behavior (Devinsky et al., 1995; Pujol et al., 2002). Not surprisingly, activity of the ACC has been associated with affective disorders and motivational level. For example, heightened activity of the ACC is associated with negative-affective states, anxiety-based disorders, depression, social phobia, and obsessive-compulsiveness (Baxter, 1991; Veit et al., 2001). Hypoactivity of the ACC is associated with apathy and with antisocial tendencies in adults (Kiehl et al., 2001; Veit et al., 2001). Taken together, activity of the ACC reflects increased or decreased motivation and affective responses towards positive or negative events.

Using it as an indirect measure of ACC activity, the Ne amplitude has been shown to correlate with obsessive-compulsiveness and socialization in adults. For example, Gehring and colleagues found that patients with a primary diagnosis of OCD had more pronounced and prolonged Ne's compared with controls and the Ne amplitude was related to symptom severity (Gehring et al., 2000). In another study, Hajcak and Simons (2002) demonstrated that obsessive-compulsive characteristics in non-clinical young adults were related to a larger Ne in response to both correct and error trials. Thus, the Ne may be a marker for error signals that are hyperactive in OCD patients and which, in turn, contribute to anxiety and compulsive behaviors.

Just as hyperactivity of the ACC and a pronounced Ne may reflect excessive performance monitoring and concern over the outcome of an event, hypoactivity of the ACC and a diminished Ne may reflect a lack of concern. Low-socialized undergraduate participants generate smaller Ne during incorrect avoidance learning trials than high-socialized participants (Dikman and Allen, 2000). The authors concluded that low-socialized participants found errors to be less salient or were less concerned about the consequences of having erred.

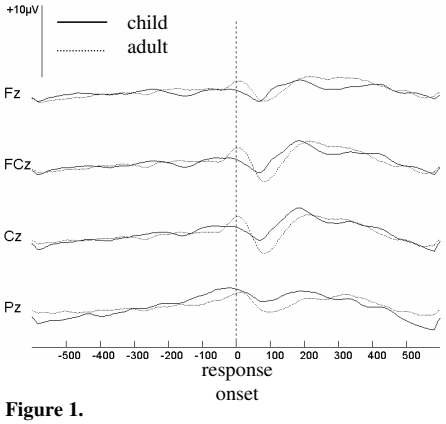

Figure 1.
Response-locked ERP waveforms for incongruent, error trials for children (heavy line) and adults (light line).

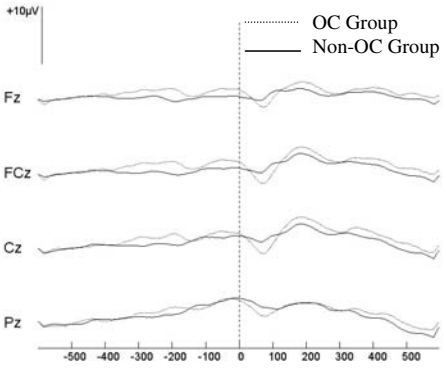

Figure 2.
Response-locked ERP waveforms for children with no obsessive-compulsive behaviors (according their mothers) versus those who have at least one (light line)

Results in children with respect to obsessive-compulsive behaviors

In order to examine some of these issues, we also collected measures on various personality scales with the parent-report Child Behavior Checklist (Achenbach, 1991). We used an alternative scoring system developed by Lengua, Sadowski, Friedrich, and Fisher (2001) because these scales address behaviors more closely associated with clinical disorders. Of the subscales, only the one for obsessive-compulsive (OC) behaviors related to the Ne and Pe. We found that children with more reported obsessive-compulsive behaviors produced larger Ne (r = -.35, p = .04) and late Pe amplitudes (r = .43, p < .01). A multiple regression analysis revealed that together, Ne and Pe components accounted for 27% of the variance in predicting OC behavior and that both components accounted for a significant amount of unique variance in OC behaviors (9% and 15%, respectively; see Figure 2).

Results in children with respect to socialization

We also administered the self-report psychoticism scale of the Junior Eysenck Personality Questionnaire-Revised (JEPQR-S; Corulla, 1990), and it correlated significantly with the Ne of the children (Santesso et al., 2003a). An item analysis showed that it was only the non-aggressive items relating to socialization from this scale that significantly correlated, and a socialization aggregate subscale correlated with the Ne (r = .49, p < .005, see Figure 3), while the aggressiveness questions subscale was not related (r = .21, ns). The late Pe did not relate to either the social or the aggressiveness questions on the JEPQR-S). Participants reporting some poor social behaviors had a smaller Ne compared with participants with no reported poor social behaviors. We also examined the Lie Scale of the JEPQR-S, and it also correlated with the Ne such that those with high Lie scores, presumably reflecting sensitivity to socialization demands, had larger Ne amplitudes. However, regression indicated that this variance was the same as the variance in Ne accounted for by the socialization scale.

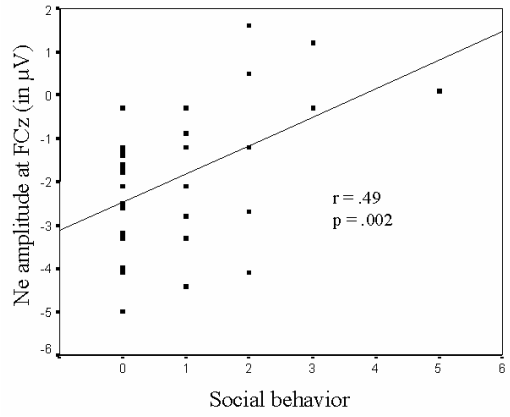

Figure 3.
Scatterplot showing the significant relation between the error negativity and scores on the socialization questions of the Psychoticism scale of the Junior Eysenck Personality Questionnaire-Revised.

Given that the OC and socialization behaviors are quite different from each other, we examined whether they account for separate variance in the Ne. A multiple regression analysis with OC and social behavior entered simultaneously into the equation accounted for 35% of the variance in predicting Ne amplitude. Of this, the OC behaviors accounted for 11% of unique variance and social behavior accounted for 24% of the variance, with virtually no overlap.

Development of Ne and Pe from 7 to 25 years of age

In the second study, we examined the development of Ne and Pe in 124 children (7 to 18 years of age), and 27 adults (19 to 25 years of age). Children were slower in responding in both correct (r = -.75, $p < .0005$) and error trials (r = -.61, $p < .0005$) than adults. Consistent with previous findings, RTs were faster on error trials (M = 400 ms) than correct trials (M = 489 ms), $F(1, 138) = 331.9$, $p < .0005$, with this difference generally decreasing with age, $F(12, 138) = 3.83$, $p < .0005$. Post-error slowing – RT for correct trials following error trials (M = 525 ms) *versus* following correct trials (M = 486 ms) – significantly decreased with age, $F(1, 138) = 57.89$, $p < .0005$. Thus, both the adults' and children's errors were impulsive (in that it was fast responses that contributed to errors) and both adults and children slowed after making an error, a standard strategy when monitoring one's performance. Thus, we believe that the children were performing the task in the same way as adults.

The adult subjects produced a standard Ne with great regularity, and it developed gradually with age, $R^2 = .204$, $F(1,122) = 31.2$, $p < .001$. The error ERP only started to be consistently shaped like that of adults by teenage years (see Figure 4). However, this developmental trend may be described as being non-linear as it was qualified by a quadratic relationship, R^2 change = .095, $F(1,121) = 16.4$, $p < .001$. There was a clear reduction in Ne amplitude at age 10 years and subsequent fluctuations through adolescence were suggestive of pubertal effects, so we examined the interactions with gender (see Figure 5). The Ne quadratic distribution indicates an initial drop in amplitude with a subsequent rise through adolescence. The girls have a minimum value at age 10 years while for the boys the lowest value is at age 13 years. For a more complete description of the age by gender effects see Davies, Segalowitz, and Gavin (2003).

The Pe amplitude did not change with age (r = -.08, n.s.). Therefore the reduction in Ne is not due to smaller ERP peaks and troughs in general. While the younger children rarely showed a strong error negativity, they almost always had a strong Pe. However, of particular interest in these data is that there were a few children who did produce a strong Ne component despite their young age. Are these children more sensitive to their slips, or to the task demands? Or perhaps these children are activating their dopaminergic system more than their peers, reflecting a more mature Ne. One might speculate that these children are more likely to be able to engage adaptive functions

associated with the prefrontal and AC cortex, perhaps associated with social resilience (i.e., being able to adapt socially in difficult situations because their self-monitoring is superior).

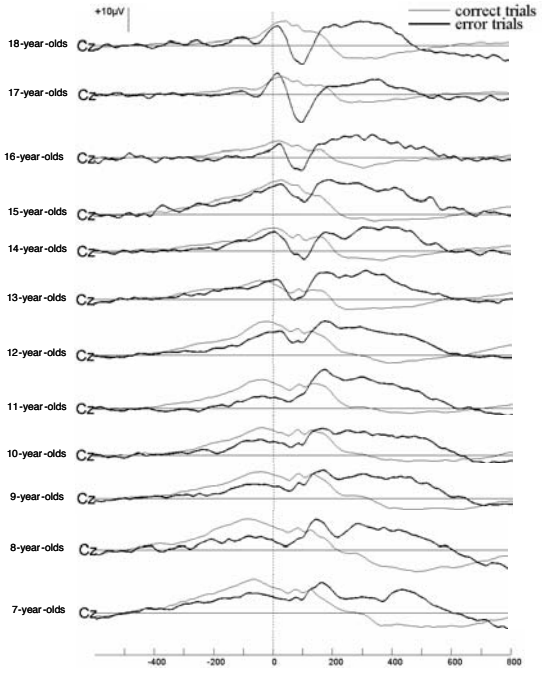

Figure 4 (left).
Averaged response-locked waveforms for correct (light lines) and error (heavy lines) trials for each age group, showing a slow maturation of the Ne. Note that the early Pe at least is apparent from the youngest ages.

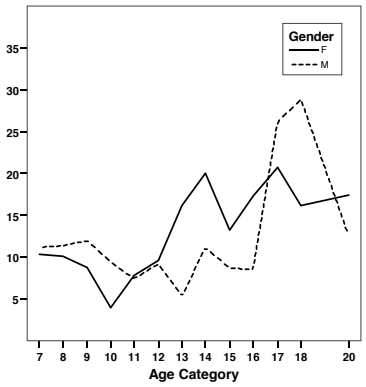

Figure 5 (above).
Error negativity amplitudes for girls (solid line) and boys (hashed line) showing the quadratic relation with age for each.

Discussion

The data presented in the first study clearly shows a difference in Ne amplitude from age 10 years to adulthood, while the second study shows that the Ne develops slowly with age. This relationship is not linear and may be related to puberty. Given that the likely generator of the Ne is the ACC, our results appear to support a model of late maturation of the ACC and possibly the associated PFC and dopamine system. These results raise the question of whether the children who showed a lack of a strong Ne recognized that they are making an error response. Our data suggest that the children did recognize their errors. Even in the absence of the Ne, all children produced a robust Pe, as did the adults. Falkenstein and colleagues 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 and conscious error recognition (Falkenstein et al., 2000). The young children also slowed their responses following error trials which indicates recognition of the error. This robust Pe in the children also indicates that the reduction in Ne amplitude could not be due simply to an attenuation of all ERP components, nor that the lack is simply due to increased latency jitter, although the generally wider envelope of the Pe compared to the Ne does not eliminate the possibility of a jitter difference. However, we do not think any such jitter is due to the increased variance in response time which the children show, because when we statistically control for this, the results do not change.

The results reported in the study with 10-year-olds examining the relation between the Ne, Pe and affective traits replicate previous findings with adults and extend these findings to non-clinical children. Results also suggest that the size of the Ne may index the hyper- or hypo-activity of the ACC and, consequently, the individual's concern with performance during cognitive tasks and the extent to which they engage in successful response monitoring. This is particularly important when

considering individual differences in response to punishing stimuli. That is, some individuals may fail to respond appropriately because of diminished responsiveness to punitive stimuli or hyper-responsivity to these cues. Researchers argue that these differences in children interact with parenting skill and affect the success of socialization efforts and the establishment of behavioral inhibitions (Center and Kemp, 2002; Eysenck, 1997).

The Ne, late maturation of the brain, and implications for developmental psychology

The results from our initial studies of the Ne and Pe in children suggest that the error monitoring paradigm as reflected in ERPs may be a useful tool for the study of child development through adolescence because of several issues. First, the ACC is clearly still developing into early adulthood and activation levels of the ACC increase into the late teens, as reflected in PET activation (Van Bogaert et al., 1998). In addition, the development of ACC size has implications for personality development, where the right ACC size relates to the individual's predisposition to worry about events (Pujol et al., 2002). However, the linkage is bidirectional: early emotional experiences affect the connectivity of the ACC (Helmeke et al., 2001).

Second, the dopamine system is still maturing with some major changes around puberty. In their animal model, Andersen et al. (2000) report a growth and elimination of dopaminergic receptors around puberty. Similarly, dopamine production as reflected in the quantity of tyrosine hydroxylase, an enzyme critical in its synthesis, continues to mature into adulthood in rhesus monkeys which contrasts with the development of another neurotransmitter, serotonin (Lambe et al., 2000). Other neurotransmitters GABA and glutamate are reported to rise dramatically during childhood and drop in level by adolescence, while dopamine rises slowly at first and then dramatically rises only during adolescence, dropping in early adulthood to a level similar to that of early childhood (Anderson et al., 1995). Thus, the error negativity may give us an important window on the development of the ACC and the dopamine system, being dependent on both of these. Both of these systems are intimately involved in what we call executive functions, the key psychological development during childhood adolescence, and this speaks directly to the possible importance of the error monitoring paradigm in reflecting the growth of these structures.

A third physiological component to be considered is the hypothalamic-pituitary-adrenal (HPA) axis and its reactivity to environmental stimulation. The stress response controlled by the HPA axis is vital for normal interactions with the world and, most likely, for learning from it. However, there are large variations in this function, reflected in some of the personality variables discussed in this chapter. As well, we now know that individual differences in social stress responses as early as infancy have significant effect in adulthood on the reactivity of the amydalae to novel faces (Schwartz et al., 2003). However, as mentioned above, the ACC is sensitive during its development to stress levels, and in fact, HPA reactivity itself is also affected by early stressors to that system. We also know that the dopamine system is influenced by early stress levels. Thus, these three systems are intimately intertwined, and an interpretation of the error negativity response may need to be associated as well with moderating variables of HPA reactivity.

These developmental changes of the brain in adolescents may predispose the adolescent to behave in particular ways such as participating in risk-taking behaviors and these changes in the adolescent brain may also relate to the onset of psychopathology such as depression and schizophrenia seen in adolescence (Spear, 2000a, b). Further investigations of the Ne and Pe, especially as they relate to traits such as gender and socialization, may provide insights into adolescence, a vulnerable period of development in the transition from childhood to adulthood.

Acknowledgments

Preparation of this manuscript was supported in part by grant # 122222-98 from the Natural Sciences and Engineering Research Council of Canada (NSERC) to SJS, by grant # 1 K01 HD01201-01A1 from National Institute of Child Health and Human Development (NICHD) to PLD, and by #410-2002-1663 from the Social Science and Humanities Research Council of Canada to LAS.

References

Achenbach TM (1991). Manual for the Child Behavior Checklist/4-18 and 1991 Profile. Burlington: University of Vermont, Department of Psychiatry.

Adleman NE, Menon V, Blasey CM, White CD, Warsofsky IS, Glover GH, et al. (2002). A Developmental fMRI Study of the Stroop Color-Word Task. Neuroimage 16: 61-75.

Andersen SL, Thompson AT, Rutstein M, Hostetter JC, Teicher MH (2000). Dopamine receptor pruning in prefrontal cortex during the periadolescent period in rats. Synapse 37: 167-169.

Anderson SA, Classey JD, Conde F, Lund JS, Lewis DA (1995). Synchronous development of pyramidal neuron dendritic spines and parvalbumin-immunoreactive chandelier neuron axon terminals in layer III of monkey prefrontal cortex. Neuroscience 67: 7-22.

Baxter LR (1991). PET studies of cerebral function in major depression and obsessive-compulsive disorder: Emerging prefrontal cortex consensus. Annals of Clinical Psychiatry 3: 103-109.

Benes FM, Vincent SL, 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: 237-45.

Center DB, Kemp DE (2002). Antisocial behaviour in children and Eysenck's theory of personality: An evaluation. International Journal of Disability, Development and Education 49: 353-366.

Corulla WJ (1990). A revised version of the psychoticism scale for children. Personality and Individual Differences 11: 65-76.

Cunningham MG, Bhattacharyya S, Benes FM (2002). Amygdalo-cortical sprouting continues into early adulthood: implications for the development of normal and abnormal function during adolescence. The Journal of Comparative Neurology 453: 116-30.

Davies PL, Rose JD (1999). Assessment of cognitive development in adolescents by means of neuropsychological tasks. Developmental Neuropsychology 15: 227-248.

Davies PL, Segalowitz SJ, Gavin WJ (2003). Development of error monitoring ERPs in participants aged 7 to 25 years. Submitted for publication.

Devinsky O, Morrell MJ, Vogt BA (1995). Contributions of anterior cingulate cortex to behaviour. Brain 118: 279-306.

Dikman ZV, Allen JJB (2000). Error monitoring during reward and avoidance learning in high- and low-socialized individuals. Psychophysiology 37: 43-54.

Eriksen BA, Eriksen CW (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. Perception & Psychophysics 16: 143-149.

Eysenck HJ (1997) Personality and the biosocial model of anti-social and criminal behavior. In: Biosocial bases of violence (A. Raine PB, D. Farrington, & S. Mednick, ed),pp21-37. New York: Plenum.

Falkenstein M, Hoormann J, Christ S, Hohnsbein J (2000). ERP components on reaction errors and their functional significance: a tutorial. Biological Psychology 51: 87-107.

Gehring WJ, Himle J, Nisenson LG (2000). Action-monitoring dysfunction in obsessive-compulsive disorder. Psychological Science 11: 1-6.

Greenough WT, Black JE, Wallace CS (1987). Experience and brain development. Child Development 58: 539-559.

Hajcak G, Simons RF (2002). Error-related brain activity in obsessive-compulsive undergraduates. Psychiatry Research 110: 63-72.

Helmeke C, Ovtscharoff WJ, Poegge G, Braun K (2001). Juvenile emotional experience alters synaptic inputs on pyramidal neurons in the anterior cingulate cortex. Cerebral Cortex 11: 717-727.

Holroyd CB, Coles MG (2002). The neural basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. Psychol Rev 109: 679-709.

Kalsbeek A, Voorn P, Buijs RM, Pool CW, Uylings HB (1988). Development of the dopaminergic innervation in the prefrontal cortex of the rat. J Comp Neurol 269: 58-72.

Kiehl KA, Smith AM, Hare RD, Mendrek A, Forster BB, Brink J, et al. (2001). Limbic abnormalities in affective processing by criminal psychopaths as revealed by functional magnetic resonance imaging. Biological Psychiatry 50: 677-684.

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: 74-84.

Lambe EK, Krimer LS, Goldman-Rakic PS (2000). Differential postnatal development of catecholamine and serotonin inputs to identified neurons in prefrontal cortex of rhesus monkey. J Neurosci 20: 8780-7.

Lengua LJ, Sadowski CA, Friedrich WN, Fisher J (2001). Rationally and empirically derived dimensions of children's symptomology: expert ratings and confirmatory factor analyses of the CBCL. Journal of Consulting and Clinical Psychology 69: 683-698.

Mathalon DH, Fedor M, Faustman WO, Gray M, Askari N, Ford JM (2002). Response-monitoring dysfunction in schizophrenia: an event-related brain potential study. J Abnorm Psychol 111: 22-41.

Pailing PE, Segalowitz SJ (in press). The error-related negativity (ERN/Ne) as a state and trait measure: motivation, personality and ERPs in response to errors. Psychophysiology.

Pailing PE, Segalowitz SJ, Dywan J, Davies PL (2002). Error negativity and response control. Psychophysiology 39: 198-206.

Pujol J, Lopez A, Deus J, Cardoner N, Vallejo J, Capdevila A, et al. (2002). Anatomical variability of the anterior cingulate gyrus and basic dimensions of human personality. Neuroimage 15: 847-55.

Rakic P, Bourgeois JP, Eckenhoff MF, Zecevic N, Goldman-Rakic PS (1986). Concurrent overproduction of synapses in diverse regions of the primate cerebral cortex. Science 232: 232-235.

Rosenberg DR, Lewis DA (1995). Postnatal maturation of the dopaminergic innervation of monkey prefrontal and motor cortices: a tyrosine hydroxylase immunohistochemical analysis. J Comp Neurol 358: 383-400.

Santesso DL, Segalowitz SJ, Schmidt LA (2003a). ERP correlates of error monitoring in 10 year olds are related to socialization. Submitted for publication.
Santesso DL, Segalowitz SJ, Schmidt LA (2003b). Error-related electrocortical responses are enhanced in children with obsessive-compulsive behaviors. Submitted for publication.
Schwartz CE, Wright CI, Shin LM, Kagan J, Rauch SL (2003). Inhibited and uninhibited infants "grown up": adult amygdalar response to novelty. Science 300: 1952-3.
Segalowitz SJ, Unsal A, Dywan J (1992). Cleverness and wisdom in 12-year-olds: Electrophysiological evidence for late maturation of the frontal lobe. Developmental Neuropsychology 8: 279-298.
Spear LP (2000a). The adolescent brain and age-related behavioral manifestations. Neuroscience and Biobehavioral Reviews 24: 417-463.
Spear LP (2000b). Neurobehavioral changes in adolescence. Current Directions in Psychological Science 9: 111-114.
Stuss DT (1992). Biological and psychological development of executive functions. Brain and Cognition 20: 8-23.
Van Bogaert P, Wikler JD, Damhaut P, Szliwowski HB, Goldmant S (1998). Regional Changes in Glucose Metabolism during Brain Development from the Age of 6 Years. Neuroimage 9: 62-68.
Van Hoesen G, Morecraft RJ, Vogt BA (1993) Connections of the monkey cingulate cortex. In: Neurobiology of cingulate cortex and limbic thalamus: A comprehensive handbook (Vogt BA and Bagriel M, ed),pp249-284. Boston: Birkhauser.
Van Veen, V., & Carter, C. S. (2002). The timing of action-monitoring processes in the anterior cingulate cortex. J Cogn Neurosci 14: 593-602.
Veit R, Flor H, Erb M, Hermann C, Lotze M, Grodd W, et al. (2001). Brain circuits involved in emotional learning and antisocial behavior and social phobia in humans. Neuroscience Letters 328: 233-236.
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 Res 281: 41-52.
Vogt BA (1993) Structural organization of cingulate cortex: Areas, neurons, and somatodendritic transmitter receptors. In: Neruobiology of cingulate cortex and limbic thalamus: A comprehensive handbook (Vogt BA and Gabriel M, ed), pp19-70. Boston: Birkhauser.

Error-related ERP Components and Source Monitoring in Older and Younger Adults

Jane Dywan, Karen J. Mathewson, & Sidney J. Segalowitz
Department of Psychology, Brock University, St. Catharines, Ontario, Canada

The effects of aging on two ERP components – the error negativity (Ne) and error positivity (Pe) – were investigated using the standard Eriksen flanker task and also an age-sensitive source-memory exclusion task. Older adults made more errors in both tasks and generated Ne and Pe components that were greatly attenuated relative to those of younger adults. Ne amplitude was similar across tasks and did not correlate with the number of errors in either paradigm, whereas the Pe was markedly more sensitive to task, age and errors. For both groups, Pe amplitude was reduced in the source memory task relative to the flanker task. However, for the younger group it shifted from a central parietal maximum in the flanker task to a more frontal scalp maximum during the source memory task. In addition, even the very attenuated Pe observed in older adults was predictive of errors in the source memory task for that group. These data support the view that the Ne and Pe represent functionally independent aspects of error monitoring. The amplitude of both components may be affected by the reduction in dopamine levels associated with aging but only the Pe appears to reflect the evaluative aspects of the error response.

Introduction

The ability to examine the neurophysiological basis of response monitoring has been enhanced by the relatively recent discovery of an electrophysiological component that is particularly evident with the commission of an error during speeded response tasks. The error negativity (Ne, Falkenstein et al., 1990) or, alternatively, the error-related negativity (ERN, Coles et al., 1991; Gehring et al., 1993) consists of a distinct negative deflection in averaged EEG recordings occurring approximately 80 ms after an incorrect response. A second, independent error-related