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Error-related event-related potentials in children with Attention-Deficit Hyperactivity Disorder, Oppositional Defiant Disorder, Reading Disorder, and Math Disorder

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Abstract

We studied Error-Related Negativity (ERN) and Error Positivity (Pe) during a discrimination task in 319 unmedicated children divided into subtypes of ADHD (Not-ADHD/ Inattentive/ Combined), Learning Disorder (Not-LD/Reading/Math/Reading+Math), and Oppositional Defiant Disorder. Response-locked ERPs contained a frontocentral ERN and posterior Pe. Error-related Negativity and Positivity exhibited larger amplitude and later latency than corresponding waves for correct responses matched on reaction time. ADHD did not affect performance on the task. The ADHD/Combined sample exceeded controls in ERN amplitude, perhaps reflecting patients' adaptive monitoring efforts. Compared with controls, subjects with Reading Disorder and Reading +Math Disorder performed worse on the task and had marginally more negative Correct-Related Negativities. In contrast, Pe/Pc was smaller in children with Reading+Math Disorder than among subjects with Reading Disorder and Not-LD participants; this nonspecific finding is not attributable to error processing. The results reflect anomalies in error processing in these disorders but further research is needed to address inconsistencies in the literature.

Keywords

Attention-deficit Hyperactivity Disorder; Learning Disorders; Error monitoring; Error-related negativity; Event-related potentials

1. Introduction

1.1 Scope of the paper

The present research examined two-error related components of event-related potentials (ERPs), Error Related Negativity (ERN; also known as Ne) and Error Positivity (Pe), in children with major subtypes of Attention-Deficit Hyperactivity Disorder (ADHD) and

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unaffected peers. ERN has been related to error monitoring, a crucial aspect of executive functions (EF) that allows an individual to readjust or correct performance so as to increase future accuracy. Although the functional significance of Pe is less completely understood (Overbeek, Nieuwenhuis, and Ridderinkhof, 2005), this ERP component has also been related to error processing.

There are several models of executive functions, but they all generally encompass those supervisory operations that control and manage other cognitive processes (e.g., planning, cognitive flexibility, abstract thinking, rule acquisition, inhibition, self-regulation). Current emphasis on disordered executive function in cognitive and emotional aspects of ADHD (Barkley, 1997; Pennington and Ozonoff, 1996) prompted interest in deficits in error monitoring among children with ADHD. We also researched whether deficits in error monitoring in ADHD vary as function of three comorbid conditions: Oppositional Defiant Disorder (ODD), Reading Disorder (RD), and Math Disorder (MD).

1.2 ADHD and related conditions

Research has identified differences among ADHD subtypes in clinical and executive functions. Children with Predominantly Hyperactive-Impulsive (ADHD/H) and Combined Types of ADHD (ADHD/C) are more aggressive, impulsive, and unpopular than their peers with Predominantly Inattentive Type of ADHD (ADHD/I; Carlson and Mann, 2000). In addition, children with ADHD/C are more impaired than those with ADHD/I on tower tests of planning (Klorman et al., 1999; Kopecky, Chang, Klorman, Thatcher, and Borgstedt, 2005; Nigg, Blaskey, Huang-Pollock, and Rappley, 2002); motor inhibition (Nigg et al., 2002); and, less consistently, set shifting (Barkley, Grodzinsky, and DuPaul, 1992; Houghton et al., 1999; Klorman et al., 1999; Nigg et al., 2002). Following a review of this type of evidence, Milich, Balentine, and Lynam (2001) proposed that these two ADHD subtypes represent distinct disorders. In contrast to this position, though, Chhabildas, Pennington, and Willcutt (2001) found that symptoms of inattention, rather than those of hyperactivity/impulsivity, were closely associated with deficits of inhibition, vigilance, and processing speed.

The two ADHD subtypes also co-occur with other disorders that may affect cognitive processing. ODD, a condition commonly comorbid with ADHD, does not magnify cognitive deficits of ADHD children (Barkley et al., 1989; Dykman and Ackerman, 1992; Klorman et al., 1999). However, ADHD comorbid with physical aggression or Conduct Disorder may involve qualitatively and quantitatively different EF weaknesses (Giancola et al., 1998; Halperin, O'Brien, Newcorn, and Healey, 1990).

Many studies have reported that learning disabilities overlap with ADHD. Studies of children with ADHD, RD, and both RD and ADHD have found that children with only ADHD are cognitively impaired on EF measures relative to unaffected peers or subjects with RD alone (Tant & Douglas, 1982; Tarnowski et al., 1986). In turn, children with RD demonstrate impairments on language measures (Pennington et al., 1993; Purvis and Tannock, 2000; Shaywitz et al., 1995). Nevertheless, some investigations identified EF deficits in RD (Dainer et al., 1981; Kelly et al., 1989). Children with both ADHD and RD exhibit linguistic and EF deficits, sometimes exceeding those of their single-diagnosis counterparts (Ackerman et al., 1986; August and Garfinkel, 1990; Felton and Wood, 1987; Kupietz, 1990; McGee et al., 1989; Shaywitz et al., 1995).

Although MD is a common learning disorder (Geary, 1993), there is far less research on the overlap of ADHD with MD than is the case for RD. Fletcher (2005) reported that the presence of ADHD did not affect cognitive profiles associated with MD. However, children with RD and RD+MD demonstrated divergent profiles on measures associated with math

difficulties, but not those involving reading difficulties. The math literature frequently identifies MD and MD+RD as separate disorders, reflecting differences in the nature of the math difficulties and their cognitive correlates. Children with only MD are often identified as experiencing difficulties with procedural knowledge and correlated difficulties on measures of set switching, problem solving, and concept formation (Lyon, Fletcher, and Barnes, 2003). Interestingly, Fuchs et al. (2005) found that behavioral ratings of inattention, but not those of hyperactivity-impulsivity, accounted for significant and robust unique variance in a variety of mathematics outcomes in a large sample of children with MD and MD+RD. Thus, whereas the presence of ADHD does not appear to modify the expression of MD (except that children with MD+ADHD appear more severely impaired), specific MD is associated with EF difficulties. Altogether, an adequate account of EF and ADHD requires that different comorbid conditions be accounted for in the analysis.

1.3 Error monitoring in ADHD

Three reports provide behavioral evidence for failure of error-monitoring in children with ADHD, who exhibited reduced or abnormal post-error slowing (Schachar et al., 2004; Sergeant and Meere, 1988; Wiersema et al., 2005). Anomalous post-error slowing supports a deficient error-monitoring mechanism and provides a possible explanation for at least part of the deficient task performance by children with ADHD. Consistent with these results, our group found that methylphenidate, a drug that remedies clinical disturbances in ADHD, increased post-error slowing by these children in a Sternberg task while also increasing speed and accuracy (Krusch et al., 1996). In contrast to these reports, Meel, Heslenfeld, Oosterlaan, and Sergeant (unpublished) detected comparable post-error slowing in their ADHD and control samples. Thus, the majority, but not all, investigations suggest disturbances in error monitoring in ADHD.

1.4 Error-related ERPs

We focused on ERN and Pe, two components of the ERP synchronized with an error response and considered the neural correlates of a self-monitoring system. When subjects make errors, ERPs display a frontocentral negative wave (ERN) with latency of approximately 100 ms and a subsequent parietally maximal positive wave (Pe; Falkenstein, Hohnsbein, Hoormans, and Blanke, 1990; Falkenstein, Hohnsbein, and Hoorman, 1995; Gehring, Goss, Coles, Meyer, and Donchin, 1993). ERN and Pe appear to depend on the detection of errors and not on the execution of an erroneous motor response insofar as ERN is linked, not only to overt errors in discrimination tasks (e.g., Falkenstein et al., 1995), but also to feedback on the commission of errors (Luu, Tucker, Derryberry, Reed, and Poulsen, 2003; Miltner, Braun and Coles, 1997). Dipole analyses of ERN point to a likely source in the anterior cingulate cortex (ACC; Dehaene, Posner, and Tucker, 1994; Luu et al., 2003; Miltner et al., 1997; Veen and Carter, 2002). These findings are consistent with imaging evidence that the ACC is active during an error and, to a lesser extent, during the execution of a correct response (Carter et al., 1998). Holroyd and Coles (2002) have theorized that ERN is generated when a negative reinforcement signal is conveyed to the ACC via the mesencephalic dopamine system; this signal is used by the ACC to modify performance on the task at hand.

Some investigators have argued that ERN reflects the detection or processing of errors and that Pe is elicited by the evaluation of the incorrect response (Falkenstein et al., 1990; Falkenstein, Hoorman, Christ, and Hohnsbein, 2000; Leuthold and Sommer, 1999; Overbeek et al., 2005). Gehring et al. (1993) found that ERN may be modulated by the perceived importance of an incorrect response. In addition, memory, visual load, mapping conditions, time on task, and subject performance all influence ERN amplitude (Scheffers, Humphrey, Stanney, Kramer, and Coles, 1999). ERN amplitude has been associated with

greater slowing on the trial following an error (Gehring et al., 1993; Scheffers, Coles, Bernstein, Gehring, and Donchin, 1996) and with greater discrepancy between the desired correct and the actual response (Bernstein, Scheffers, and Coles, 1995). ERN amplitude is increased by motivational manipulations like reward value and evaluation of performance (Hajcak, Moser, Yeung, and Simons, 2005). In addition, ERN amplitude is enhanced in several clinical conditions involving affective distress, including Obsessive-Compulsive Disorder (Gehring, Himle, and Nisenson, 2000; Ruchow, Grön, Reuter, Spitzer, Hermle, and Kiefer, 2005). In contrast, ERN amplitude is reduced in schizophrenia (Mathalon et al., 2002).

Correct responses evoke CRN, a wave similar to ERN albeit of smaller amplitude (Falkenstein et al., 1990; Vidal, Hasbrouq, Grapperon, and Bonnet, 2000). Falkenstein et al. (1990) suggested that CRN may reflect some degree of uncertainty about the subject's response selection. In turn, Vidal et al. (2000) argued that because ERN/CRN is not uniquely tied to error responses, ERN may not, in fact, reflect an error detection process; in contrast, they proposed, Pe may more sensitively reflect the error detection process because Pe was observed only after error responses. Notably, ERN was present following both conscious and unconscious errors in antisaccade (Nieuwenhuis, Ridderinkhof, Blom, Band, and Kok, 2001) and saccade countermanding tasks (Endrass, Franke, and Kathman, 2005). In contrast, these studies found that Pe was larger for conscious than unconscious errors.

1.4 Present study

We investigated ERN and Pe in children varying with respect to DSM-IV subtypes of ADHD and comorbid conditions: ODD, RD, and MD. The present study is a reanalysis of data collected for different purposes (Klorman et al., 2002). Based on evidence that ADHD children have deficits in response selection, motor adjustment, and inhibition of prepotent responses (Douglas, 1999; Oosterlaan et al., 1998; Sergeant et al., 1999), we investigated the impact on ADHD and comorbid disorders of two factors affecting response selection: stimulus probability and sequence. Demands on response selection are greater when targets are rarer than nontargets than when they are equiprobable. Specifically, the bias to make the more frequent nontarget response (Sternberg, 1969) is hypothesized to lower speed and accuracy for low-probability events relative to equiprobable targets. Sequence effects concern faster reactions to identical than discrepant consecutive stimuli, particularly in long stimulus runs (Remington, 1969) and involve response selection (Bertelson, 1963) as well as earlier processing stages (Pashler and Baylis, 1991). Notably, stimulus alternation increases and repetition reduces amplitude and latency of N2 and P3b, ERP components linked to the earlier stage of stimulus identification (Squires et al., 1977; Giese-Davis et al., 1993). The modulation of ERPs by sequence regularity is likely mediated by concomitant changes in subjective probability. Briefly, we (Klorman, 2002) found no differences among our ADHD and LD samples in the impact of stimulus probability. In contrast, the extent of alternation sequences elicited less systematic increases in errors, P3b latency, and P3b amplitude among children with ADHD than controls. Finally, for children with MD and RD+MD, alternation sequences had a greater impact on performance and smaller effect on concurrent ERPs.

The data obtained in our study provided the opportunity to examine error-related ERPs in this sample. When the present work was initiated in 1999, no previous research was available on ERN and Pe in children, but our working hypothesis was that children would exhibit ERN and Pe in their error-synchronized ERPs. Generalizing from the literature documenting deficient EF in ADHD (Barkley, 1997; Pennington and Ozonoff, 1996), we expected evidence of deficient error monitoring in ADHD. Another theoretical connection between ERN and ADHD comes from Holroyd and Coles' (2002) hypothesis that ERN involves the connection between the mesencephalic dopamine system and the ACC. Insofar as ADHD involves a dysfunction of the dopaminergic system and has been related to

impaired reinforcement learning (Castellanos, 1999), this work suggests that the processes involved in ERN may be diminished in ADHD. Therefore, we tentatively hypothesized that children with ADHD, particularly those with ADHD/C, would have smaller ERN and Pe amplitude during error trials than controls.

Extrapolating from our previous findings on deficient EF among children with MD (Klorman et al., 1999), we tentatively hypothesized that children with MD (especially those with RD+MD), but not those with RD, would exhibit smaller amplitude of ERN and Pe than nondisabled children. No abnormalities of ERN or Pe were expected for children with ODD. We had no hypotheses concerning the corresponding waves synchronized with correct responses (CRN and Pc, respectively).

2. Method

2.1 Participants

The sample consisted of 319 subjects recruited for a study of learning and attention at the Yale University Center for Learning and Attention, including (a) 290 children referred by schools, parent groups, and professionals or enlisted by means of media announcements; and (b) 29 subjects without learning or disruptive disorders who were recruited by newspaper advertisements, letters to schools, and fliers posted in toy stores and libraries. As already mentioned, we reported this sample's ERP results for correct trials in a previous publication (Klorman et al., 2002). The present paper is focused on error-related ERPs in the same cohort.

All subjects met the following criteria: (a) 7 to 13.5 years old; (b) Wechsler Intelligence Scale-Revised (1974) Verbal or Performance IQ > 75; (c) English as a primary language; (d) Absence of Pervasive Developmental Disorder, Schizophrenia, Bipolar Disorder, or neurological disorder as assessed by history and parental report; (e) Normal or corrected vision and hearing as evaluated specifically in this study; (f) Abstinence from all medications for at least 7 days (except over-the-counter analgesics or antibiotics) before all testing; and (g) No siblings in the sample.

DSM-IV diagnoses of ADHD subtypes and ODD were derived from a widely used and reliable structured parent interview (Diagnostic Interview Schedule for Children [DISC-IV], Shaffer, Fisher, Lucas, Dulcan, and Schwab-Stone, 2000). For patients undergoing stimulant medication, interviews were conducted at the end of the period in which the child was withdrawn from treatment.

The psychometric evidence supporting the DISC-IV can be summarized as follows. For eight disorders covered in the parent interview, Shaffer et al. (2000) reported one-week test-retest reliability (kappas) ranging from .43 (Conduct Disorder) to .96 (Specific Phobia). For an earlier version of the DISC, internal reliability (intraclass correlations) of symptom counts for 10 disorders ranged from .43 (Panic Disorder) to .85 (Oppositional Defiant Disorder). Sensitivity of the instrument relative to diagnoses drawn from independent clinical interviews ranged from .73 to 1.0. Notably, for each of these psychometric criteria, ADHD always placed at the higher end of the range.

Reading Disorder (RD) was diagnosed if the average of the age-based standard score on reading achievement subtests (mean of Letter-Word Identification and Word Attack) of the Woodcock-Johnson Psychoeducational Battery (Woodcock, 1978) was (a) at least 1.5 standard errors below the reading score predicted from the subject's WISC-R Full Scale IQ by a regression equation derived from a normal population (Shaywitz et al., 1995); or (b) in the lowest quartile of reading scores for this standardization population. Previous research

indicated that children classified by these two criteria of reading disability exhibit comparable differences from nondisabled subjects (Fletcher et al., 1994, 1998). Math disorder (MD) was defined analogously to reading disorder based on the Woodcock-Johnson Calculation subtest (Woodcock, 1978).

Behavior in school was evaluated via parents reports on the DISC interview. In addition, teacher questionnaires documented significant behavioral disturbances by the present ADHD sample (Klorman et al., 1999).

2.2 Comparison of samples on demographic, intelligence, and achievement variables

Results for children with diagnoses of ADHD/C ($n = 155$) and ADHD/H ($n = 27$) were pooled. Subjects were grouped along three dimensions: ADHD (Not-ADHD, ADHD/I, or ADHD/C), learning disorder (Not-LD, RD, MD, and RD+MD), and Oppositional Defiant Disorder (Not ODD or ODD).

Table 1 presents demographic, IQ, and achievement results for each sample. Several differences were found among samples. The ADHD samples included a greater proportion of boys than the Not-ADHD group, $\chi^2(2, N = 319) = 8.41, p < .02$. In addition, there was a marginal excess of minority subjects in the ADHD vs. Not-ADHD samples, particularly the ADHD/C cohort, $\chi^2(2, N = 319) = 5.59, p < .10$. Similarly, there was a higher number of minority members in the LD categories, especially the MD sample, $\chi^2(3, N = 319) = 11.79, p < .01$.

ADHD samples differed with respect to Wechsler Full Scale IQ, $F(2,307) = 5.27, p < .01$, a result reflecting higher IQs for Not-ADHD children than either ADHD/I or ADHD/C subjects ($p < .02$). LD samples differed on IQ, $F(3,307) = 27.48, p < .0001$, reading achievement, $F(3,307) = 145.06, p < .0001$, and Calculation scores, $F(3,307) = 179.67, p < .0001$. In each case, statistical comparisons ($p < .03$ or less) indicated that Not-LD children obtained higher scores than all other samples whereas subjects with RD+MD scored lower than all other LD samples.

Demographic and psychoeducational differences among diagnostic samples were taken into account as follows: (a) Age was treated as a covariate in data analyses; (b) Separate analyses evaluated the effects of ODD; findings for ODD did not qualify the results and will not be mentioned further; (c) All analyses were repeated without minority subjects and the findings were unchanged; and (d) IQ was shown to be nonsignificantly correlated with measures of ERN, Pe, and performance, so that there was no justification for statistical adjustments of sample differences in IQ. We note that lower IQ and academic achievement in children with ADHD, RD, or MD are not due to sampling variation but are characteristic of these populations.

2.3 Procedure

The study was reviewed and approved by the Institutional Review Boards at Yale University and the University of Rochester, and parents provided informed consent. Subjects participated in a three-hour session held after a 7-day washout period for subjects treated with stimulants. EEG and the electrooculogram (EOG) were recorded by means of Grass Model 12 Neurodata amplifiers set for nominal upper and lower cutoff frequencies of .03 and 100 Hz, respectively. MedVivo sintered silver electrodes were taped to five scalp sites on an Electro-Cap: Fz, Cz, Pz, C3, and C4 and referenced to linked earlobes. EOG was detected from infra- and supraorbital electrodes. Subjects were grounded by a midforehead electrode. Impedance was < 3 Kohms at all sites.

The child was tested alone in two tasks involving randomly ordered presentations on a TV monitor of the letters O or X (125 ms, $1.43 \times 0.95^\circ$ visual angle) at intervals of 1,500 ms. Each task included one practice block of 100 trials followed by four test trial blocks of 200 trials. In the Rare task, the letters O and X were presented with probabilities of .17 and .83 respectively; in the Equiprobable task the probability of each letter was .50. The subject responded to presentations of both O and X by pressing predesignated microswitches positioned in a slanted surface under his/her right and left hand. Subjects were randomly assigned to press to O with their right hand (and to X with their left hand) or to the opposite arrangement. Task order was counterbalanced over subjects. Instructions stressed speed while maintaining accuracy.

2.4 Scoring: Performance

The following measures of overall performance were scored for each task after excluding the first trial of each block: (1) Proportion of active errors: (a) misses, that is, presses with the wrong button when O was presented, and (b) false alarms, that is, presses with the wrong button when X was presented; (2) Proportion of nonresponses, that is, failures to respond within an unannounced deadline of 1,125 ms; (3) Proportion of premature responses, that is, presses to either X or O with latency under 200 ms; (4) Mean reaction time for correct responses to O and X; and (5) Within-subject standard deviation of correct reaction time responses to O and X. Complete results on these measures of performance were available for 312 of 319 subjects.

We also evaluated slowing on trials following an error but will not report these results because they varied complexly as a function of the combination of task and stimulus. The findings can be found in Burgio-Murphy (2001).

2.5 Scoring: ERPs

For each subject, we identified all active error trials (false alarms or misses) with reactions that were not considered premature (> 200 ms). Each of these error trials was individually matched within ± 25 ms of reaction time with a correct trial from the same task (Rare or Equiprobable) and stimulus (X and O). The mean (SD) reaction times for matched correct and error trials, respectively, were 479.9 (103.2) and 473.7 (97.8) ms for the Rare condition and 526.2 (99.8) and 507.8 (93.3) ms for the Equiprobable condition. Although initially an equal number of matched correct and error trials were selected, slight discrepancies arose after eliminating trials involving voltages exceeding the range of the A/D converter. The mean (SD) number of trials for ERPs based on correct and error ERPs, respectively, was 62.89 (36.95) and 74.28 (72.97) in the Rare condition and 64.02 (45.26) and 75.66 (76.95) in the Equiprobable condition.

Performance and physiological data were collected on-line by a Northgate 386 PC outfitted with a Labmaster DMA A/D board. The computer digitized EEG and EOG data at a rate of 200 Hz and epoched these data from 150 ms before through 1,100 ms post-stimulus. For the present work, EEG data for each trial, electrode, task, and condition were (1) aligned at a point 100 ms before the button press, that is, the estimated onset of the electromyographic (EMG) response (Coles, Gratton, Bashore, Eriksen, and Donchin, 1995; Illiczky, Klorman, and Thatcher, unpublished data; Ridderinkhof and van der Molen, 1995); (2) segmented into epochs spanning 150 ms before and 500 ms after the estimated onset of the EMG response; (3) adjusted separately for vertical and horizontal activity corresponding to eyeblinks and other eye movements (Gratton, Coles, and Donchin, 1983); (4) submitted to a 51-point low-pass Hamming filter with a half-amplitude cutoff frequency of 6 Hz; and (5) deviated from the mean amplitude in the 150-ms estimated pre-EMG baseline. Because of equipment and

other difficulties (e.g., insufficient number of trials in one or more categories), ERP data were available for 304 of 319 children.

We note that after the completion of the present research, Yordanova, Falkenstein, Hohnsbein, and Kolev (2004) reported that ERN involves a performance monitoring system operating in the delta range (1.5-3.5 Hz) and a movement monitoring system operating in the theta range (4-8 Hz). Unfortunately, our low-pass filter attenuated, but did not entirely eliminate, activity in the theta range (50% gain at 6 Hz and 23% gain at 8 Hz).

Figures 1 and 2 display grand averages for ADHD and LD samples, respectively, of response-synchronized ERPs for each task, error/correct trials, and electrode. The topography of the present ERPs conforms with that reported for adults: A frontally maximal, ERN/CRN with mean maximum latency of 120 ms ($SD = 34$) was followed by a centrally maximal Pe/Pc with mean peak latency of 250 ms ($SD = 30$). As shown in the Figures 1 and 2, ERN/CRN and Pe/Pc were present in ERPs for both error and correct responses.

ERN/CRN latency and amplitude were scored as follows. For each subject, we computed a grand average based on four ERPs (2 tasks $\times$ error/correct) at Fz. A computer program identified the most negative voltage between 100 and 160 ms post estimated EMG onset for this grand average and, subsequently, scored the latency of ERN for the ERP from each subject's 20 ERPs (5 electrodes $\times$ 4 conditions) as the most negative value in a window ± 50 ms around the latency for ERN/CRN in the subject's grand average at Fz. For each ERP, the program also scored the amplitude of the wave at the identified latency minus the mean amplitude in the 150-ms interval preceding the estimated onset of EMG response. We scored the late positivity (Veen and Carter, 2002) for error (Pe) and correct trials (Pc) as the mean voltage, relative to the pre-EMG baseline, for the interval 305 - 500 ms post estimated EMG onset. We also analyzed early Pe/Pc waves but the results were similar to those from the late Pe/Pc and we have omitted them to save space.

3. Results

3.1 Analytic approach

Dependent variables were submitted to the BMD P4V implementation of weighted multivariate and univariate analyses of variance with two between-subject diagnostic factors: ADHD (Not-ADHD, ADHD/C, and ADHD/I) and Learning Disorder (Not-LD, RD, MD, and RD+MD). This design permitted all potential comorbidities and their interactions to be examined in the analysis.

The following repeated measures were included, as applicable: task (Equiprobable vs. Rare), stimulus (X vs. O), and electrode sites (Fz, Cz, Pz, C3, and C4). The Greenhouse-Geisser adjustment for nonsphericity was applied to probability values for repeated measures. Age was treated as a covariate. Effect sizes are reported as partial omega squared (η^2_p). Omnibus tests were evaluated with two-tailed .05 alpha.

Planned pairwise comparisons were performed among all three ADHD subtypes and four LD categories. The following planned comparisons between selected electrodes assessed differences along the midline and between the two lateral sites: Fz vs. Cz, Cz vs. Pz, and C3 vs. C4. Interactions of ADHD $\times$ LD as well as higher order interactions involving ADHD $\times$ LD did not approach significance for any dependent variable and, therefore, will not be mentioned further.

In a complex design like that of the present study, there is an opportunity to test many hypotheses of interest for numerous dependent variables and the need to consider possible confounding factors. We employed the following strategy in order to minimize Type I error.

For families of three pairwise tests, we used unadjusted alpha levels of .05, an approach that successfully maintains alpha for this number of comparisons (Myers and Well, p. 252). For families comprising more than three tests, we adjusted alpha levels by the Bonferroni correction. We report nominal p levels and identify results as significant, marginal (familywise alpha $< p < \text{per comparison alpha}$), or not significant.

3.2 Measures of overall performance

The results for performance will be described only briefly because they were reported previously (Klorman et al., 2002). Means for the five measures of performance appear in Table 2. A multivariate analysis of covariance adjusting for age indicated that, as expected, the impact of the two stimuli differed across tasks, Task $\times$ Stimulus $F_{mult}(5,296) = 431.58$, $p < .0001$. Specifically, the letter O (infrequent) evoked relatively worse performance than X (frequent) in the Rare condition than was the case in the Equiprobable task. In fact, significant univariate Task $\times$ Stimulus interactions ($p < .0001$) emerged for each performance measure except percentage of premature responses. The impact of tasks was similar across ADHD cohorts, Task $\times$ Stimulus $\times$ ADHD $F_{mult}(10,592) = 1.24$, $n.s.$, $\eta^2_p = .02$, and LD samples, Task $\times$ Stimulus $\times$ LD $F_{mult}(15,818) = 1.46$, $n.s.$, $\eta^2_p = .03$.

As shown in Table 2, averaging across tasks, there were no differences in performance among children as a function of ADHD categories, $F_{mult}(10,590) < 1$, $n.s.$, $\eta^2_p = .01$. In contrast, there were large differences among LD groups, $F_{mult}(15,815) = 4.38$, $p < .0001$, $\eta^2_p = .08$, and univariate comparisons of LD samples were significant ($p < .0001$) for each dependent variable except for active errors (false alarms and misses). Planned comparisons indicated that the Not-LD sample made fewer nonresponses and premature responses as well as faster and less variable reactions than children in all samples. These differences were marginal ($p < .05$) relative to the MD and RD groups and significant ($p < .003$) in comparison to the RD+MD cohort. Both the RD ($p < .02$) and the MD groups ($p < .0001$) made fewer nonresponses than the RD+MD cohort, and the MD sample had significantly less variable reaction times than did the RD+MD group ($p < .0005$).

3.3 ER/CRN latency

As depicted in Figures 1 and 2, ERN peaked later than CRN ($M \pm SE = 123.4 \pm 1.3$ vs. 118.2 ± 1.4 ms, respectively), $F(1,292) = 19.74$, $p < .0001$, $\eta^2_p = .06$. This difference was more pronounced in the Rare task for both waves, ERN/CRN $\times$ Task, $F(1,292) = 12.14$, $p < .001$, $\eta^2_p = .04$.

3.4 ERN/CRN amplitude: Overall effects

The effects of task were not significant for ERN, $F(1,292) < 1$, and for CRN, $F(1,292) = 1.22$. Therefore, no further mention of task will be made except for occasional interactions involving this factor.

As expected and illustrated in Figures 1 and 2, ERN had larger amplitude than CRN, $F(1,292) = 132.01$, $p < .001$, $\eta^2_p = .31$. As also shown in Figures 1 and 2, ERN/CRN amplitude varied over the scalp, Electrodes $F(4,1168) = 261.88$, $p < .0001$, $\eta^2_p = .47$. Planned comparisons revealed that ERN/CRN negativity decreased from frontal to posterior midline sites, Fz vs. Cz, $F(1,292) = 120.72$, $p < .0001$, $\eta^2_p = .29$; Cz vs. Pz, $F(1,292) = 414.66$, $p < .0001$, $\eta^2_p = .59$. In addition, ERN/CRN amplitude was larger at C3 than C4, $F(1,292) = 12.39$, $p < .001$, $\eta^2_p = .04$.

There were two interactions involving electrodes: Electrodes $\times$ ERN/CRN, $F(4,1168) = 55.70$, $p < .0001$, $\eta^2_p = .16$, and Electrodes $\times$ Task, $F(4,1168) = 12.15$, $p < .0001$, $\eta^2_p = .04$. Following McCarthy and Wood's (1985) caution that interactions of experimental

conditions by electrodes may merely reflect overall amplitude differences among electrodes rather than different effects of conditions on electrodes, we scaled ERN/CRN amplitudes by a vector solution.

The analysis of ERN vector scores yielded a significant interaction of ERN/CRN $\times$ Electrode, $F(4,1168) = 200.32, p < .0001, \eta^2_p = .41$. These results indicate that the extent of greater negativity for ERN than CRN varied over electrodes, independent of overall amplitude differences among electrodes. Simple effects analyses disclosed that scaled ERN was more negative than scaled CRN at Fz, $F(1,292) = 223.56, p < .0001, \eta^2_p = .43$; Cz, $F(1,292) = 120.91, p < .0001, \eta^2_p = .29$; C3, $F(1,292) = 24.61, p < .0001, \eta^2_p = .08$; and C4, $F(1,292) = 11.99, p < .001, \eta^2_p = .04$. In contrast, ERN was *less* negative than CRN at Pz, $F(1,292) = 227.93, p < .0001, \eta^2_p = .44$.

3.5 ERN/CRN amplitude: Diagnostic findings

This section focuses on differences between diagnostic groups in separate analyses of voltage measures of ERN and CRN. ERN amplitude differed significantly among ADHD groups, $F(2,291) = 3.43, p < .04, \eta^2_p = .02$. As shown in Figures 1 and 3, Not-ADHD children had significantly smaller ERN amplitude than did ADHD/C subjects, $F(1,291) = 6.47, p < .02, \eta^2_p = .02$. ERN amplitudes for ADHD/I children did not differ significantly from those of controls, $F(1,291) = 1.43, n.s., \eta^2_p < .01$, or ADHD/C patients, $F(1,291) = 1.62, n.s., \eta^2_p < .01$. Averaging over electrodes, age-adjusted $M_s \pm SE$ for ERN were $0.93 \pm 0.22 \mu V$ for controls, $0.11 \pm 0.19 \mu V$ for ADHD/I children, and $-0.47 \pm 0.12 \mu V$ for ADHD/C subjects. The differences in ERN amplitude among ADHD groups were maximal at Cz and Pz and smaller at other scalp sites, ADHD $\times$ Electrodes $F(8,1168) = 2.66, p < .01, \eta^2_p = .02$. This interaction prompted an analysis of ERN amplitudes in vector scores, which confirmed the overall differences among ADHD samples, $F(2,291) = 4.55, p < .02, \eta^2_p = .03$, and the larger ERN amplitudes for children with ADHD/C than controls, $F(1,291) = 8.65, p < .005, \eta^2_p = .03$.

Notably, ADHD samples did not differ in CRN amplitude, $F(2,291) < 1, n.s., \eta^2_p < .01$.

Whereas LD samples did not vary in ERN amplitude, LD $F(3,291) < 1, n.s., \eta^2_p < .01$, they did differ in CRN amplitude, LD $F(3,291) = 2.92, p < .05, \eta^2_p = .03$. As shown in Figures 2 and 3, Not-LD participants had marginally less negative CRNs than MD children, $F(1,291) = 5.66, p < .02, \eta^2_p = .02$, and RD+MD youngsters, $F(1,291) = 5.46, p < .03, \eta^2_p = .02$.

3.6 Pe/Pc: Overall effects

As shown in Figures 1 and 2, Pe was more positive than Pc, $F(1,292) = 231.50, p < .0001, \eta^2_p = .244$. As also expected, Pe/Pc amplitude varied significantly across scalp sites, Electrodes $F(4,1168) = 153.48, p < .001, \eta^2_p = .34$. Pe/Pc exhibited a posterior maximum, Fz vs. Cz $F(1,292) = 280.78, p < .001, \eta^2_p = .49$; Cz vs. Pz $F(1,292) = 160.94, p < .001, \eta^2_p = .36$. There were several interactions involving electrodes, including Electrodes $\times$ Task, $F(4,1168) = 19.58, p < .0001, \eta^2_p = .03$, Electrodes $\times$ Pe/Pc $F(4,1168) = 233.27, p < .001, \eta^2_p = .44$, and Electrodes $\times$ Task $\times$ Pe/Pc, $F(4,1168) = 5.64, p < .001, \eta^2_p = .02$. Because all these interactions were confirmed in analyses of vector scores ($p < .001, \eta^2_p > .02$), we undertook separate analyses of Pe and Pc vector scores. For Pe vector scores, there was an interaction of Electrodes $\times$ Task $F(4,1168) = 10.09, p < .001, \eta^2_p = .03$. This interaction reflected small differences between tasks in the gradient of scaled amplitude over the scalp. Regardless of task, Pe vector scores increased from anterior to posterior scalp, but differences in amplitude between Cz and Fz were greater in the Equiprobable task, $F(1,292) = 8.74, p < .003, \eta^2_p = .03$, whereas increases in amplitude from Cz to Pz were greater in the

Rare Task, $F(1,292) = 19.16, p < .001, \eta^2_p = .06$. In contrast, for Pc, these differences were absent, Electrodes $\times$ Task $F(4,1168) < 1, n.s.$

3.7 Pe/Pc: Diagnostic effects

Analyses of Pe/Pc are reported for voltage values because interactions of Electrodes with diagnostic factors concerned relative differences in the amplitude of Pe/Pc across tasks. Pe/Pc amplitude did not differ among ADHD groups, $F(2,291) < 1, n.s., \eta^2_p < .01$, but did vary among LD samples, $F(3,291) = 3.36, p < .02, \eta^2_p = .03$. Specifically, as illustrated in Figures 2 and 3, Pe/Pc was smaller among RD+MD subjects ($M = 1.24 \mu V, SE = 0.74$) than among RD children ($M = 3.44 \mu V, SE = 0.99$), $F(1,291) = 7.17, p < .008$, and Not-LD youngsters ($M = 3.89 \mu V, SE = 0.47, F(1,291) = 10.38, p < .002$).

4. Discussion

4.1 Overall performance

The results support the intended effect of the present task. Specifically, in comparison with the Equiprobable task, the Rare task increased the rate of errors, slowed reaction times, and increased the adverse impact of the letter O (infrequent) on accuracy and speed. Thus, as intended, performance was degraded by the requirement to inhibit the frequent response in the Rare task.

In contrast to the literature on cognitive deficits in ADHD (e.g. Douglas, 1999; Sergeant and Meere, 1999), children with both subtypes of ADHD performed comparably to their Not-ADHD peers on the test. These findings probably follow from our use of a relatively simple task in an effort to minimize performance decline over the session. As a result, the task permitted the unusual, but scientifically fortunate, circumstance of comparing ERPs for ADHD and control children in the absence of performance differences.

Contrary to the comparable performance among ADHD samples in the present task, many differences were found among LD groups. Compared to nondisabled participants, children with RD and RD+MD made more active errors. In addition, children in all LD samples differed from Not-LD subjects in having more nonresponses as well slower and more variable reactions. Overall, these results point to poorer performance by subjects with both RD and MD on the current task. Importantly, the findings for ADHD and LD samples apply regardless of comorbidity between these disorders.

4.2 ERN: Overall effects

The present ERN was maximal at Fz and decreased from anterior to posterior sites. This result is consistent with some studies with adult samples that found that ERN amplitude is maximal at frontal leads and diminishes across the midline (Falkenstein et al., 1990, 1995; Gehring et al., 1993); however, other reports have indicated that ERN peaked at Cz (Vidal et al., 2000). Recent studies of children spanning a wide age range (Davies, Segalowitz, and Gavin, 2004; Kim, Iwaki, Uno, and Fujita, 2005; Santesso, Segalowitz, and Schmidt, 2005) also found a frontal focus for ERN. The similarity between the scalp topography of the present pediatric ERN with the centro-frontal topography reported for adult subjects supports the assumption that the same component was measured in both children and adults.

Studies on adults have found a maximal ERN amplitude of approximately 10 microvolts (Gehring et al., 1993), although there is variability across studies in this respect. In the current study, ERN at Fz had a far smaller mean amplitude of approximately 2.5 microvolts. However, consistent with our findings, Davies et al. (2004) reported that ERN in a flanker task was smaller in children than in adults. In fact, in that study, it was not until adolescence

that ERNs exhibited negative amplitude relative to the pre-response baseline. Notably, other groups have also reported that ERN amplitude is smaller in children and increases through adolescence (Hogan, Vargha-Khadem, Kirkham, and Baldeweg, 2005; Kim et al., 2005; Ladouceur, Dahl, and Carter, 2004).

Another possible explanation for the smaller amplitude of the present ERN is that the children in the present study made a relatively high percentage of errors (12%) compared with adults (e.g. 2.4%, Vidal, et al., 2000). Following this reasoning, the smaller amplitude of ERN in children would reflect the association between ERN amplitude and frequency of errors (Scheffers et al., 1999). Contradicting this view, however, ERN amplitude did not differ between tasks even though the Rare condition evoked a higher error rate than the Equiprobable task.

Another apparent difference from past results on adults is that in the current study, ERN peaked later (approximately 120 ms vs. 80 – 100 ms; Falkenstein et al., 1991, 1995). Part of the discrepancy results from the fact that the present ERN latencies are based on the estimate of EMG onset at 100 ms before the button press whereas Falkenstein et al.'s (1990, 1995) ERNs were synchronized with their subjects' reaction times. Thus, their reported peak latencies of 70-80 ms are shorter than the present ones after adjustment for synchronization conventions. On the other hand, Gehring et al. (1993), who synchronized ERN measurement with the onset of the EMG response, reported mean ERN latencies of 110 ms, a value also close to that obtained in the present work. In addition, our ERN latencies are consistent with those reported by Kim et al. (2005) for children's EMG-synchronized ERNs. On the whole, the present finding of later ERN latencies for children than previously reported for adults is consistent with the widely established developmental finding that ERP latencies decrease with development (Dustman and Beck, 1969; Courchesne, 1978).

Another controversy concerns the emergence of CRN on correct trials. Though early reports (e.g., Gehring et al., 1993) did not detect CRNs, recent investigations have detected a response-locked negative wave on correct trials, that is, CRN. Davies et al. (2004) found that CRN was larger in children than in adults, though their CRN --like ours-- had positive polarity with respect to the pre-response baseline. Also consistent with other studies of children (Davies et al., 2004; Hogan et al., 2005; Kim et al., 2005; LaDouceur et al., 2004;) as well as adult studies reporting finding CRN (Falkenstein et al., 1990; Vidal et al., 2000), our CRN was smaller than ERN. Importantly, investigations that did not find CRN had matched correct and error trials on reaction time (Gehring et al., 1993; Dehaene et al., 1994) whereas this matching was not accomplished in many investigations reporting a CRN. Thus, it was arguable that the emergence of ERNs on correct trials was related to the failure to match error and correct trials on reaction time. The present finding of CRN following correct responses whose reactions times were matched with those of error trials argues against an explanation of this phenomenon based on scoring method.

4.3 Pe: Overall effects

The present Pe was maximal at Pz. This topography is consistent with the adult (Falkenstein et al., 1990, 2000) and emerging pediatric literature (Santesso et al., 2005), which found Pe to be maximal at centro-parietal sites. Notably, the late Pe was differentiated from the early Pe in part by a parietal versus a central maximum (Ruchow, Grön, Reuter, Spitzer, Hermle, and Kiefer, 2005; Veen and Carter). However, the early aspects of the present Pe were also maximal at the parietal site.

Our findings are also compatible with results from studies based on adults with respect to the emergence of Pe on both error and correct trials (Falkenstein et al., 1995). Again, the presence of a Pe-like wave on correct trials (i.e., Pc) has varied across studies, and when Pc

was reported, it was much smaller than Pe (Nieuwenhuis et al., 2001; Falkenstein et al., 1990), as was the case in the present study. If Pe reflects error monitoring, the presence of Pc on correct trials is consistent with Falkenstein et al.'s (1990) claim that error detection is relative rather than absolute. The assumption here is that subjects engage in some degree of error monitoring even when they are correct. The present results suggest that similar processes may occur in children.

4.4 ERN/CRN and Pe/Pc: Findings for ADHD

Children with ADHD/C, contrary to our hypothesis, had *larger* ERN amplitude on error trials than did Not-ADHD subjects. This finding suggests that children with ADHD/C, may be more sensitive to the detection of errors than controls. Importantly, the present subjects with ADHD were comparable to their Not-ADHD peers on accuracy, reaction time, and ERN amplitude on correct trials. Insofar as ERN is part of an error detection system, the results suggest that children with ADHD/C may have been more attentive to their mistakes and more intent on monitoring them. Following Nieuwenhuis et al.'s (2001) formulation, the difference in ERN amplitude on errors may reflect preconscious awareness of the commission of an active error. Greater vigilance in error monitoring may have aided children with ADHD/C to adjust their performance to the level of their Not-ADHD peers.

An alternative explanation might be based on Vidal et al.'s (2000) proposition that ERN represents an emotional response to perceived errors. In a related context, Santesso et al. (2005) found that, among psychiatrically normal 10-year old children, low ERN amplitude was related to low self-reported social skills. These authors proposed that low socialized children may underreact to or discount the emotional significance of errors and that ERN may reflect hypoactivity in the ACC with which this affective style may be associated. Although Santesso et al.'s work relates ERN amplitude to personality *traits* (socialization, in particular) rather than to *state* emotionality, their findings suggests a link between emotionality and ERN. Their work supports Vidal et al.'s proposition and is consistent with the possibility that children with ADHD may be more emotionally reactive to making errors.

It is notable that children with ADHD/I did not differ in ERN amplitude from controls or participants with ADHD/C. In fact, their ERN amplitudes were intermediate between those of these two groups. Apparently, children with ADHD/I display a smaller tendency to monitor errors than their counterparts with ADHD/C. This finding is consistent with evidence reviewed earlier that children with ADHD/I, compared with patients with ADHD/C, are less deviant from controls on some tests of EF (e.g., Klorman et al., 1999; Kopecky et al., 2005). However, the present result that the ADHD/C displayed greater than normal error monitoring points to a compensatory, rather than a dysfunctional, mechanism.

Since the completion of the present study, there have been several reports examining ERN in ADHD. Unfortunately, the findings have not been entirely consistent. In accord with our original hypothesis, Liotti, Pliszka, Perez, Kothman and Woldorff (2005) found smaller ERN amplitude in children with ADHD in a Stop Task. Similarly, Meel et al. (unpublished) found smaller ERN among children with ADHD than controls in a flanker task; apparently, the ADHD sample failed to exhibit greater negativity for ERN than CRN. In contrast, Wiersema, Meere, and Roeyers (2005) did not find differences in ERN amplitude between children with ADHD and controls in a Go/No Go task or in a choice reaction time paradigm. Finally, the present report yielded larger ERN amplitude by the ADHD cohort.

Notably, in all these studies except the present one, the ADHD cohort was more inaccurate (but not slower) than controls. This finding may explain part of the discrepancies in findings. Task complexity (Hogan et al., 2005) affects ERN amplitude and, obviously, accuracy. Perhaps the greater simplicity of the present task relative to those used in the

studies summarized above enabled our ADHD subjects to perform with comparable accuracy and speed to their unaffected peers. This factor may facilitate the maintenance of error monitoring in children with ADHD such that they were able to achieve normal performance; alternatively, the relative ease of the task may have promoted greater attention to errors.

In contrast to the results for ERN, children with ADHD did not differ from Not-ADHD subjects in Pe amplitude. Nieuwenhuis et al.'s (2001) formulation of ERN vs. Pe implies that, whereas children with ADHD/C are as consciously aware of their errors as children without this disorder, the earlier, preconscious error monitoring reflected in ERN was more active for the ADHD/C cohort. Interestingly, Santesso et al. (2005) found a correlation between low social skills (and, by inference, low emotional reactions to error) with low ERN amplitude but not with Pe amplitude.

As was the case for ERN, our findings are not consistent with those of other investigations of Pe in ADHD samples. Overtoom et al. (2002) found that children with ADHD performed worse on a Stop task and exhibited smaller amplitude of a posterior maximal failure-related wave interpreted by these authors as Pe. Similarly, Wiersma et al. (2005) found smaller amplitude of Pe in ADHD children. Thus, the findings for Pe, like those for ERN, are difficult to reconcile without further research. We suggest again consideration of task complexity and concomitant extent of performance deficits by the ADHD sample.

4.5 ERN/CRN and Pe/Pc: Findings for LD

Subjects with LD, particularly those with reading problems (RD and RD+MD) made more errors overall and were slower than Not-LD subjects. These findings are generally consistent with our previous report of greater EF impairment in the samples with RD+MD than those with MD alone (Klorman et al., 1999). On the other hand, neither participants with RD or MD differed from Not-LD children with respect to ERN amplitude. Thus, there was no evidence that diminished error monitoring accounted for the poorer performance by children with LD. Rather, subjects with math difficulties (MD and RD+MD) displayed marginally more negative CRN than Not-LD subjects. Although there was a similar trend among children with only RD, the smaller size of this sample may have afforded insufficient power for reaching statistical significance. The interpretation of these results is not clearcut but points to a possible anomaly in the functioning of the monitoring system. On the other hand, any interpretation of deviant CRN in children with LD needs to be tempered by the fact that the present CRN responses did not have negative polarity.

The main result involving LD was that children with RD+MD obtained smaller Pe/Pc waves than children with RD and Not-LD children. This finding cannot be related specifically to error processing insofar as it involves both correct and incorrect responses. In combination with the trends for larger CRN amplitude by subjects with MD, the present results suggest the possibility of deviance in error detection for RD+MD subjects but additional research is needed to ascertain the interpretation and replicability of these results. On the whole, these findings are consistent with our previous report that children with MD, particularly those with RD+MD, exhibit greater cognitive and EF deficits than those with RD (Klorman et al., 1999).

5. Conclusions

The present findings suggest that ADHD and LD differ substantially from nondisabled peers on error processing as measured by ERN and Pe. Notably, the impact of these diagnoses were additive for ERN and Pe as well as for performance measures. Thus, independent of

comorbidity, children with both ADHD and LD classifications engage in increased self-monitoring to maintain optimal task performance.

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Fig. 1. ERPs synchronized with error and matched correct responses for each ADHD sample, separately by task and electrode. The 0 time point indicates the estimated start of the EMG response, that is, 100 ms before the button press. Positivity is displayed upward.

Fig. 2. ERPs synchronized with error and matched correct responses for each LD sample, separately by task and electrode. The 0 time point indicates the estimated start of the EMG response, that is, 100 ms before the button press. Positivity is displayed upward.

Fig. 3. ERPs at Fz, synchronized with error and matched correct responses for each ADHD sample and each LD sample. The 0 time point indicates the estimated start of the EMG response, that is, 100 ms before the button press. Positivity is displayed upward.

Table 1

Frequency and mean + SD of demographic and psychoeducational measures

Diagnosis	n	White ^a	Male ^a	Age ^b	Full Scale		Woodcock-Johnson	
					WISC-R IQ ^b	Reading ^b	Calculation ^b	
Not Disabled								
Not-ADHD	29	28	13	10.34 ± 1.79	117.90 ± 11.84	110.55 ± 9.74	108.90 ± 12.98	
ADHD/I	41	39	29	9.95 ± 1.57	105.73 ± 12.13	104.33 ± 8.34	103.39 ± 8.38	
ADHD/C	96	88	73	9.59 ± 1.67	112.21 ± 12.36	105.89 ± 9.67	106.17 ± 10.54	
RD								
Not-ADHD	7	6	4	8.51 ± 1.02	103.29 ± 14.48	82.93 ± 9.42	99.71 ± 8.62	
ADHD/I	7	7	6	11.15 ± 1.51	105.71 ± 10.58	87.43 ± 4.26	105.15 ± 10.78	
ADHD/C	15	15	12	10.71 ± 1.86	103.53 ± 14.05	84.83 ± 6.54	99.53 ± 8.35	
MD								
Not-ADHD	9	9	5	8.74 ± 1.28	109.00 ± 13.81	101.33 ± 9.24	84.00 ± 6.06	
ADHD/I	18	15	12	10.02 ± 1.77	101.11 ± 13.77	102.80 ± 8.50	82.28 ± 7.67	
ADHD/C	44	36	36	10.02 ± 1.73	100.50 ± 12.41	100.57 ± 8.46	77.80 ± 10.17	
RD + MD								
Not-ADHD	15	15	13	9.97 ± 1.72	99.00 ± 14.74	80.27 ± 8.73	79.07 ± 3.06	
ADHD/I	11	11	7	10.99 ± 1.78	95.55 ± 10.06	79.82 ± 8.17	73.18 ± 11.46	
ADHD/C	27	23	20	10.53 ± 2.02	92.92 ± 11.24	77.89 ± 11.19	73.00 ± 12.50	

^a n

^b M ± SD

Table 2Age-adjusted mean $\pm$ SD of overall performance measures for ADHD and LD samples

Sample	Percent Active Errors	Percent Non-responses	Percent Premature Responses	Mean Reaction Time (ms)	SD Reaction Time (ms)
LD samples					
Not-LD	10.81 $\pm$ 10.67	7.40 $\pm$ 7.72	2.97 $\pm$ 3.58	598.79 $\pm$ 91.58	161.30 $\pm$ 35.92
RD	13.82 $\pm$ 15.83	11.15 $\pm$ 8.51	3.44 $\pm$ 3.68	632.98 $\pm$ 90.76	173.96 $\pm$ 36.21
MD	12.43 $\pm$ 11.25	10.42 $\pm$ 8.95	3.94 $\pm$ 4.50	628.54 $\pm$ 95.98	170.29 $\pm$ 33.64
RD + MD	14.30 $\pm$ 11.23	14.27 $\pm$ 9.50	4.81 $\pm$ 4.14	640.15 $\pm$ 83.90	181.39 $\pm$ 30.71
ADHD samples					
Not-ADHD	11.11 $\pm$ 11.10	8.55 $\pm$ 8.34	2.82 $\pm$ 3.27	613.32 $\pm$ 89.06	165.48 $\pm$ 40.81
ADHD/I	12.71 $\pm$ 11.18	9.13 $\pm$ 8.94	3.70 $\pm$ 4.39	604.22 $\pm$ 94.58	162.58 $\pm$ 36.05
ADHD/C	11.92 $\pm$ 11.72	9.83 $\pm$ 8.72	3.64 $\pm$ 3.94	619.25 $\pm$ 93.34	170.02 $\pm$ 33.05