ERROR MONITORING IN CHILDREN WITH ADHD OR READING DISORDER: AN

EVENT-RELATED POTENTIAL STUDY

Séverine Van De Voorde[1], Herbert Roeyers[1], Jan Roelf Wiersema[1]

[1]Department of Experimental-Clinical and Health Psychology, Ghent University, Belgium

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Address correspondence to: Séverine Van De Voorde

Ghent University

Department of Experimental-Clinical and Health Psychology

H. Dunantlaan 2, B-9000 Ghent

Belgium

Tel: +32-9-2649414

Fax: +32-9-2646489

E-mail: severine.vandevoorde@ugent.be

Abstract

This study compared children with ADHD, reading disorder (RD), ADHD+RD, and control children on behavioural (post-error slowing and post-error accuracy) and event-related potential (Ne and Pe) measures of error monitoring. Children with ADHD did not differ from children without ADHD in post-error slowing but showed less post-error accuracy enhancement, as evidenced by a higher proportion of double-errors. We found a smaller Ne but normal Pe amplitude in children with RD, and a smaller Pe but normal Ne amplitude in children with ADHD. Children from the comorbid group showed both a smaller Ne and a smaller Pe amplitude, which suggests that they showed the additive combination of the deficits found in both separate disorders. The results of the present study suggest that it might be important to control for the presence of comorbid RD when examining error monitoring in ADHD and that various measures of post-error adaptation should be included.

Keywords: ADHD; reading disorder; error monitoring; Ne; Pe; ERP.

Introduction

ADHD and reading disorder (RD) are two of the most common developmental disorders in childhood. They also co-occur much more often than can be expected by chance, with rates of overlap estimated between 15% and 40% (e.g., Del'Homme, Kim, Loo, Yang, & Smalley, 2007; Semrud-Clikeman et al., 1992; Shaywitz, Fletcher, & Shaywitz, 1995; Willcutt & Pennington, 2000). Children with ADHD are characterized by behavioural symptoms of inattention and/or hyperactivity and impulsivity. Children with RD are characterized by persistent reading problems despite adequate cognitive ability and educational opportunities. Although both disorders are diagnosed in different ways (ADHD by parent reports; RD by reading tests), they share some behavioural symptoms like inattentive behaviour and academic difficulties (Hinshaw, 1992). This makes differential diagnosis difficult and urges research into cognitive and neurobiological variables that might better distinguish between both disorders (Rashid, Morris, & Morris, 2001).

Although for a long time it was believed that the core cognitive deficit of ADHD is an inhibition deficit (Barkley, 1997) and that the core deficit of RD is of phonological nature (Snowling, 2000), it becomes more and more clear that this is not the whole story for either disorder. With respect to ADHD, it has been found that children with the disorder show a general inaccurate response style, not only in tasks measuring inhibition, but also in other neuropsychological tasks (Rommelse et al., 2007; Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003; van der Meere, 2005). In addition, it has been claimed that no evidence of a response inhibition deficit can be found when tasks with an experimental manipulation of inhibition load are used and performance is compared to a control condition (Rommelse et al.; Van De Voorde, Roeyers, Verté, & Wiersema, in press). The latter has also been found for other executive function (EF) deficits that are frequently attributed to ADHD, such as deficits in working memory (Karatekin, Bingham, & White, 2009; Klein, Wendling, Huettner, Ruder,

& Peper, 2006; Shallice et al., 2002; Van De Voorde et al., in press) and cognitive flexibility (Rommelse et al., 2007). With respect to RD, there have been reports of additional deficits that cannot easily be explained by a pure phonological deficit. These include deficits in temporal processing (see review by Farmer & Klein, 1995), visual processing (Stein & Walsh, 1997), working memory (e.g., Swanson, Ashbaker, & Lee, 1996; Swanson, Mink, & Bocian, 1999; Van De Voorde et al., in press), and response inhibition (e.g., Purvis & Tannock, 2000; van der Schoot, Licht, Horsley, & Sergeant, 2000; Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005). In the study by Van De Voorde et al. it was found that children with RD did not only make more errors in linguistic tasks (e.g., reading, phonology, rapid naming) but also in a Go/no-go task, independent of the modality of the stimulus that had to be processed (letters, digits, or meaningless symbols that could not be labeled). It appeared that the inaccurate response style found in children with ADHD was not unique to the disorder, as it was also observed in children with RD. It was also found that both in ADHD and in RD this high error rate could not be explained by a deficit in response inhibition alone. However, correct performance in such speeded reaction time (RT) tasks is not only dependent upon the efficacy of inhibition processes but also of the activation of a monitoring system that signals the need to adjust behaviour when confronted with conflict or errors (Hajcak & Simons, 2008; O'Connell et al., 2009; Zhang, Wang, Cai, & Yan, 2009). Therefore, it is possible that the deteriorated accuracy in these clinical populations is rather the result of deficiencies in the higher-order error monitoring system than of isolated problems with inhibition.

Error monitoring is an executive control process that enables online detection of errors and subsequent adjustment of performance so as to increase future accuracy (Schachar et al., 2004). These processes are highly relevant in daily life as detection and future avoidance of errors are important parts of self-regulatory and goal-directed behaviour, necessary to flexibly

adjust to internal and external needs (Ullsperger & Falkenstein, 2004) and to learn from previous behaviours (Garavan, Ross, Murphy, Roche, & Stein, 2002).

These processes have been studied with the behavioural measure of post-error slowing (Rabitt, 1966), that is, slowing down response speed on the trial following an error to prevent future errors. However, it is not clear which aspect of error monitoring (e.g., error detection or error correction) is disturbed when problems with post-error slowing are observed. The discovery of electrophysiological indices has made it possible to study error processes more accurately and has renewed interest in these processes. The two event-related brain potentials (ERPs) that are observed after an erroneous response have been labeled error negativity (Ne; Falkenstein, Hohnsbein, Hoormann, Blanke, 1990), also known as error-related negativity (ERN; Gehring, Coles, Meyer, & Donchin, 1990; Gehring, Goss, Coles, Meyer, & Donchin, 1993), and error positivity (Pe; Falkenstein, Hohnsbein, Hoormann, Blanke, 1991). The Ne is a sharp negative potential with fronto-central maximum peaking between 0 and 160 ms after an erroneous response, whereas the Pe is a more extended positive potential that follows the Ne with a parietal maximum between 200 and 500 ms after an incorrect response (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). The generator of both processes seems to be located in the anterior cingulate cortex (ACC), more specifically in the dorsal/caudal part (dACC) for the ERN and in the ventral/rostral part (vACC) for the Pe (e.g., Herrmann, Römmler, Ehlis, Heidrich, & Fallgatter, 2004; O'Connell et al., 2007; van Boxtel, van der Molen, & Jennings, 2005; van Veen & Carter, 2002). Although different hypotheses exist on the functional significance of the Ne and Pe, it seems that the Ne reflects an early, more automatic, error detection system, whereas the Pe reflects the conscious or emotional evaluation of the error (Falkenstein et al., 2000; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005; van Veen & Carter, 2002). It has been found by Nieuwenhuis et al. (2001) that the Pe is only elicited following aware errors

and not following unaware errors, whereas the Ne is not affected by error awareness. This has recently been confirmed by other studies (e.g., Endrass, Franke, & Kathmann, 2005; O'Connell et al., 2007) and suggests that the Ne and Pe are distinct parts of the error monitoring process with the Pe occurring only after the conscious recognition that an error was made.

People with ADHD show a general inaccurate response style in speeded RT tasks and they seem to have difficulties with learning from their mistakes in daily life. Therefore, it is not surprising that error monitoring has become an important research topic in ADHD. Since the first report by Sergeant and van der Meere (1988) and more recent reports by Schachar et al. (2004) and Wiersema, van der Meere, and Roeyers (2005), that have injected new life into this research line, several papers have been published yielding somewhat inconsistent results. The most consistent finding has been a reduced Pe amplitude in children with ADHD, first reported by Overtoom et al. (2002) and later confirmed with different paradigms (Groen et al., 2008; Jonkman et al., 2007; Wiersema et al., 2005; Zhang et al., 2009) and within adult ADHD populations (O'Connell et al., 2009; Wiersema et al., 2009). However, there are also studies that did not find Pe differences between persons with and without ADHD (Albrecht et al., 2008; Burgio-Murphy et al., 2007; McLoughlin et al., 2009; Wild-Wall et al., 2009). With respect to the Ne, results have been far less consistent: the Ne amplitude has been found to be normal (Jonkman, van Melis, Kemner, & Markus, 2007; O'Connell et al., 2009; Wiersema et al., 2005; Wiersema, van der Meere, & Roeyers, 2009; Wild-Wall, Oades, Schmidt-Wessels, Christiansen, & Falkenstein, 2009; Zhang et al., 2009), reduced (Albrecht et al., 2008; Groen et al., 2008; Liotti, Pliszka, Perez, Kothmann, & Woldorff, 2005; McLoughlin et al., 2009; van Meel, Heslenfeld, Oosterlaan, & Sergeant, 2007), or even enhanced (Burgio-Murphy et al., 2007) in patients with ADHD. Thus, the ERP results generally suggest that children and adults with ADHD have problems with error monitoring with the most consistent finding of a

reduced Pe amplitude, implying aberrant conscious evaluation of errors. Together with the finding of reduced or abnormal post-error slowing (e.g., Krusch et al., 1996; Schachar et al., 2004; Sergeant & van der Meere, 1988; Wiersema et al., 2005), these results suggest that a deficient error monitoring system may, at least partly, explain the deteriorated task performance in children with ADHD. Transferred to daily life, this could mean that they do not seem to learn from their mistakes because of deviant error monitoring processes that hamper them in adequately adjusting their behaviour (Groen et al., 2008).

However, since a comparable inaccurate response style in some neuropsychological tasks has also been reported in RD (e.g., Burgio-Murphy et al., 2007; Van De Voorde et al., in press), it is important to investigate the role of problems with error monitoring as a possible underlying factor. In everyday life, children with RD continue to make decoding/reading errors despite intensive remedial therapy on top of the normal reading instruction at school (Lyon, Shaywitz, & Shaywitz, 2003). As the pattern of errors they make seems to be rather inconsistent (Horowitz-Kraus & Breznitz, 2008), it could be that they are less efficient in detecting their own reading errors. Although little is known about the error monitoring system in RD, there has been a report of reduced Ne amplitude in adults with RD during a lexical decision task (Horowitz-Kraus & Breznitz, 2008) and of a marginally more negative correct negativity (Nc) in children with RD during a choice RT task (Burgio-Murphy et al., 2007). The Nc, a wave similar to the Ne but smaller in amplitude and evoked by correct responses (Falkenstein et al., 1990; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000), has been suggested to reflect some degree of uncertainty about one's response selection (Falkenstein et al., 1990; Pailing & Segalowitz, 2004), sometimes caused by misperceived or incomplete stimulus processing (Scheffers & Coles, 2000), or to reflect the response monitoring process on correct trials (Falkenstein et al., 2000; Vidal et al., 2000). With respect to ADHD, no differences in Nc have been reported (e.g., Burgio-Murphy et al., 2007; van Meel et al., 2007).

The aim of the present study was to make an attempt to unravel the underlying mechanisms of the inaccurate response style that has been found both in children with ADHD and in children with RD by examining different aspects of the error monitoring process. Comparing both disorders in the same investigation may provide insight into the deficits that are specific for one of the disorders and the deficits that are shared. Comorbidity of ADHD with other disorders such as RD has often been neglected in studies on error monitoring. However, if specific error monitoring deficits are also present in RD, then failing to control for the presence of RD in studies on ADHD could distort research results and could give rise to inconsistencies across studies. Therefore, it is important to clarify the influence that comorbid RD might have on the results that are found with respect to ADHD. We did not only investigate a group of children with ADHD-only and a group of children with RD-only, but we also included a comorbid group with both disorders. Children with ADHD+RD might exhibit the deficits of only one of the disorders, the additive combination of the deficits of both disorders, or they might represent a separate subtype with deficits that are different from both single disorder groups.

Behavioural and ERP indices were used to examine processes that are engaged following the commission of an inhibition error. In order to show that differences are specific for errors and that impaired performance is related to malfunctioning of the error monitoring system, it is necessary that the error-related components Ne and Pe are examined relative to the correct-related potentials in the same time window (Falkenstein et al., 2000; Wild-Wall et al., 2009). Therefore, the Ne was compared with the Nc, and the Pe was compared with the correct ERP wave in the same time window. The latter will be referred to as Pc, although it must be noted that this is not a distinguishable component. On the behavioural level, we did not only investigate post-error slowing but also post-error accuracy enhancement (i.e., improvement in performance on trials following an error). Although the latter reflects an

alternative compensatory behavioural adjustment caused by error-induced control processes (Burle, Possamai, Vidal, Bonnet, & Hasbroucq, 2002; Hajcak & Simons, 2008), it has been neglected in studies on error monitoring in ADHD.

Method

Participants

Four groups of children aged 8-12 years participated: 18 children with ADHD, 15 children with RD, 13 children with ADHD+RD, and 16 typically developing controls ('control group'). All children were recruited through newspaper advertisements, through referral by speech therapists or paediatric psychologists, and through letters to parents distributed in schools. Children were selected for the screening procedure if they had a diagnosis of ADHD and/or had a history of reading problems (diagnosis of RD or referral to a speech therapist). Parents completed the following questionnaires: the Disruptive Behaviour Disorder Rating Scale (DBDRS; Pelham, Gnagy, Greenslade, & Milich, 1992), the Child Behaviour Checklist (CBCL; Achenbach, 1991), and the Social Communication Questionnaire (SCQ; Berument, Rutter, Lord, Pickles, & Bailey, 1999). Children were included in the control group if they had no history of learning or psychiatric problems and scored in the normal range on these questionnaires. The first two questionnaires were used as selection instruments in the control group only; in the clinical groups they were used to obtain a description of possible comorbid problems. Exclusion criteria for all groups were: (1) neurological problems, uncorrected hearing or vision, or speech problems, (2) native language different from Dutch, (3) a clinical score on the SCQ (symptoms of autism), (4) presence of other diagnoses (e.g. anxiety disorder), or (5) an estimated Full Scale IQ (FSIQ) below 80, based on the Vocabulary, Similarities, Picture Arrangement and Block Design subtests of the WISC-III (Wechsler, 1991). This short version of the WISC-III is the one recommended by

Grégoire (2000) and has a high correlation ($r = .93$) with FSIQ (Kaufman, Kaufman, Balgopal, & McLean, 1996). Sociodemographic information was obtained from the parents; The Hollingshead Index (Hollingshead & Redlich, 1958) with 5 classes of social status was used as a measure of socioeconomic status (SES).

Children's ADHD diagnosis was validated with the parent-administered Diagnostic Interview Schedule for Children for DSM-IV (DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). Reading problems were evaluated with 2 standardized Dutch reading measures: the Dutch One-Minute-Test (Brus & Voeten, 1973) and the Klepel, a pseudoword reading task (van den Bos, lutje Spelberg, Scheepsma, & de Vries, 1994). The raw scores on these reading measures were converted into standard scores (SS) using grade related norms with a mean of 10 and a standard deviation (SD) of 3. Children in the control group had to obtain a SS of at least 8 on both reading measures. Assignment to one of the 3 clinical groups was based on the DISC-IV (diagnosis of ADHD) and the reading measures (SS $\leq$ 5 on at least 1 of the 2 reading tasks). Children with a clinical diagnosis but insufficient symptom levels to meet these criteria were excluded from the study to make groups as homogeneous as possible. The ODD (Oppositional Defiant Disorder) and CD (Conduct Disorder) modules of the DISC-IV were administered to evaluate the presence of comorbid behavioural disorders. Children with ADHD were included if they had an ODD diagnosis but excluded if they had a CD diagnosis.

Sample Characteristics

As can be seen in Table 1, there were no significant differences between the groups with respect to IQ, gender composition or SES. There were significant differences in age, such that the children with ADHD were younger than the children with RD.

The mean score of ADHD symptoms on the DBDRS was significantly higher for the ADHD groups than for the non-ADHD groups, and the RD groups had a significantly lower reading score than the non-RD groups. However, children with ADHD had a significantly lower reading score and children with RD displayed significantly more inattention symptoms compared to the control group, although they did not meet the cut-off for RD or ADHD, respectively. The comorbid group did not significantly differ from the ADHD-only group on ADHD symptoms or from the RD-only group on the reading score. Based on the DISC-IV ADHD diagnoses, we found no differences between both ADHD groups in the proportion of ADHD subtypes ($\chi^2(1) = .39$, $p = .54$). In the ADHD-only group, 6 children (33%) met criteria for the inattentive type, and 12 children (67%) for the combined type. In the ADHD+RD group, 3 children (23%) met criteria for the inattentive type, and 10 children (77%) for the combined type. Both ADHD groups had significantly more ODD symptoms on the DBDRS than both groups without ADHD. There were no differences between both ADHD groups in percentage of children meeting a DISC-IV ODD diagnosis ($\chi^2(1) = .01$, $p = .93$): 8 children (44%) in the ADHD-only and 6 children (46%) in the comorbid group. With regard to CD, all groups exhibited insufficient symptoms on the DBDRS to make a sound comparison, and none of the children had a DISC-IV CD diagnosis.

[Insert Table 1 about here]

Neuropsychological Measure: Go/no-go Task

The Go/no-go task used in the present study was based on the one adopted by Kim, Iwaki, Uno, and Fujita (2005) to study the Ne in Japanese school-age children. The reason for choosing this task was its potential to elicit a high percentage of commission errors ($\pm$ 30% in control children). Stimuli consisted of equilateral triangles (1.5 cm on each side) in four different orientations: pointed up, pointed down, tilted 6° to the right, tilted 6° to the left.

Participants were instructed to press a response button with their dominant hand when the triangle was pointed up (Go stimulus), but not to press to any other triangle (No-go stimuli). They were told to do this as quickly and accurately as possible, but with speed being stressed more heavily to increase the rate of commission errors in normal control children.

The task consisted of 600 trials, divided into 15 blocks containing 40 trials each. In one block, a Go stimulus appeared in 60% of the trials, and a No-go stimulus in 40% of the trials (with respect to the latter: 20% were triangles that pointed side down, 10% were tilted to the right, and 10% were tilted to the left). At the start of each trial, a fixation cross was presented for 500 ms at the centre of the computer screen, immediately followed by the target stimulus that was displayed for 200 ms, ending in a variable blank response interval (between 1100 and 1300 ms). Consequently, the total trial duration varied between 1800 and 2000 ms. The task lasted for about 20 minutes, breaks not included.

Procedure

The study was approved by the Ethical Committee of Ghent University. After written consent was obtained from the parents, appointments for the two testing days were made. During the first session all diagnostic measures were administered. The neuropsychological task combined with the EEG measurement took place at the second testing day. Besides the Go/no-go task children also performed a flanker task (results reported elsewhere). Task order was counterbalanced across participants. After explaining the procedures, the electrode cap was attached, the Go/no-go task was explained and a practice block was performed. The main task was not started until the child thoroughly understood the task instructions. Frequent breaks were provided to minimize the effects of fatigue and problems with sustaining attention. During the task, the experimenter was present but sat out of sight of the child, and no interaction with the experimenter was allowed. Children on psychostimulant medication

discontinued it at least 24 hours before testing. No medication other than methylphenidate was used.

Overt Performance

Accuracy was scored as percentage of commission errors (i.e., pressing the button after a No-go stimulus) and percentage of omission errors (i.e., no response after a Go stimulus). Post-error accuracy was defined as the proportion of errors following a commission error (double-errors). Mean reaction time (MRT) was calculated as the mean RT of all correct Go trials, and within-subject variability in RT was operationalized as within-subject standard deviation of RTs (SD-RT). RTE was calculated as the mean RT of all commission errors. RTE+1 was based on all first correct Go trials after a commission error. RTC was based on all correct Go trials except for correct trials immediately following a commission error. Post-error slowing was examined by comparing the RTs of correct responses that were preceded by an error with correct responses that were not. Trials with premature responses (i.e., with a latency under 150 ms) were excluded from all RT measures except for RTE calculation.

Electrophysiological Measures

The Electro-encephalogram (EEG) and Electro-oculogram (EOG) were recorded with 63 scalp electrodes, mounted in a customized cap (EasyCap Active; EasyCap GmbH), and one additional electrode placed below the right eye (all active electrodes). Because the ERP components of interest were most pronounced at these sites, only data from FCz and CPz were analyzed for the purpose of this study.

Data were digitized with a sampling rate of 500 Hz and amplified with an open pass-band from DC to 100 Hz, using a Brain Vision Quickamp amplifier (Brain Products, Gilching, Germany), which uses a common average reference.

The EEG was offline filtered with a low pass filter of 30 Hz, a time constant of 1s (phase shift-free Butterworth filters; 24 dB/octave slope) and a 50-Hz notch filter. Ocular artefacts were corrected using the Gratton and Coles algorithm (Gratton, Coles, & Donchin, 1983), as implemented in Brain Vision Analyzer (Brain Products, Gilching, Germany). Signals were segmented into epochs of 500 ms before to 600 ms after response onset (response-locked epochs) and baseline-corrected relative to the -400 to -200 pre-response interval. Epochs with physiological artefacts in any EEG channel were rejected before averaging. These were identified by the following criteria: a voltage step of more than 50 µV between sample points, and a voltage difference of more than 200 µV within an epoch. Signals were averaged according to accuracy to obtain correct and incorrect response-locked ERPs; only trials with correct responses to Go stimuli (with a RT between 150 and 1500 ms) or incorrect responses to No-go stimuli were included in the analyses.

To measure Ne/Nc and Pe/Pc amplitudes, time interval boundaries were chosen based on inspection of the grand average waveforms. The amplitudes of the Ne and Nc were defined as the mean amplitude in the -25 to 75-ms interval at FCz. As the slow-going Pe appeared to consist of two parts, an "early part of the Pe" was defined as the mean amplitude in the 175 to 345-ms interval at CPz and a "late part of the Pe" was quantified as the mean amplitude in the 345 to 475-ms interval at CPz (see also Wild-Wall et al., 2009).

Statistical Analyses

The following performance measures were analyzed with 2 (ADHD vs. no ADHD) x 2 (RD vs. no RD) factorial ANOVAs: MRT, SD-RT, commission errors, omission errors,

double-errors. A mixed design ANOVA, with ADHD and RD as between-subjects factors and Trial type as repeated measure, was adopted to test whether groups differed in response time on error trials (MRT versus RTE) and to examine differences in post-error slowing (RTC versus RTE+1). The electrophysiological measures (Ne, Pe) were analyzed by mixed design ANOVAs with ADHD and RD as between-subjects factors and Trial type (correct vs. incorrect response) as within-subjects factor. Post hoc group comparisons were made using a Bonferroni correction. Two subjects with RD and two with ADHD were omitted from the ERP analyses (but not from the behavioural analyses) due to technical difficulties or bad EEG signals. Because there were significant group differences in age, we explored the relationship between this factor and the dependent measures of interest by calculating Pearson correlation coefficients. A significant correlation was found between age on the one hand and MRT ($r =$ -0.42, $p = .001$) and SD-RT ($r = -0.44$, $p < .001$) on the other hand. Therefore, the analyses of both dependent measures were conducted with age as a covariate and age-adjusted scores are reported. Since there was no significant correlation between age and the difference score of MRT and RTE, the ADHD x RD x Trial type (MRT vs. RTE) ANOVA was performed without age as a covariate.

<center>Results</center>

Behavioural Data

   Overall performance.   The means of the four groups on each variable are presented in Table 2. With respect to the percentage of commission errors, there was a significant main effect of ADHD ($F(1,58) = 8.60$, $p < .01$, $\eta^2_p = .13$) together with a significant interaction between ADHD and RD ($F(1,58) = 10.74$, $p < .01$, $\eta^2_p = .16$). Post-hoc analyses comparing the four groups revealed that only children from the ADHD-only group made more commission errors than children without ADHD. Children with ADHD+RD did not differ from children without ADHD and made significantly less errors than the ADHD-only group ($p < .01$).

Children with ADHD also made more omission errors than children without ADHD ($F(1,58) = 14.78$, $p < .001$, $\eta^2_p = .20$). The comorbid group did not differ from the ADHD-only group. For MRT, an ADHD x RD interaction emerged ($F(1,57) = 6.23$, $p < .05$, $\eta^2_p = .10$) and post-hoc analyses comparing the four groups indicated that children with ADHD+RD were marginally slower than children with ADHD-only ($p = .05$).

All children with ADHD showed greater within-subject variability in response speed than children without ADHD ($F(1,57) = 8.59$, $p < .01$, $\eta^2_p = .13$). The comorbid group did not differ significantly from the ADHD-only group.

The repeated measures analysis that was conducted to examine RT on error trials versus RT on correct trials, revealed a significant main effect of Trial type ($F(1,58) = 323.91$, $p < .001$, $\eta^2_p = .85$), indicating that all children responded more quickly when they made an incorrect than when they made a correct response.

   Post-error adjustment effects.   Contrary to expectations, we did not find a significant amount of post-error slowing in any of the groups as indicated by a non-significant main effect of the within-subjects factor Trial type (RTC vs. RTE+1). There were no

interactions between this within-subjects factor and the between-subjects factors ADHD and RD, which suggested that none of the groups showed a different pattern of post-error RT adjustment.

With respect to the proportion of double-errors, used as an index of post-error accuracy enhancement, we found a significant main effect of ADHD ($F(1,58) = 11.92$, $p = .001$, $\eta^2_p = .17$), which indicated that children with ADHD made more double-errors than children without ADHD. Post-hoc tests comparing the four groups showed that, although the ADHD and ADHD+RD group did not significantly differ from each other, it was merely the ADHD-only group that differed significantly from the control group ($p < .01$).

[Insert Table 2 about here]

Electrophysiological Data

Grand average waveforms following correct and incorrect responses for each group at FCz and CPz are depicted in Figure 1.

Ne/Nc. To analyze group differences in Ne/Nc amplitude, an ADHD (absent vs. present) x RD (absent vs. present) x Trial type (correct vs. error) ANOVA was conducted. There was a significant main effect of Trial type ($F(1,54) = 4.16$, $p < .05$, $\eta^2_p = .07$) due to more negative amplitudes following errors than after correct responses. With respect to the diagnostic factors, there were no significant main effects ($ps > .49$). There was, however, a significant interaction between Trial type and RD, $F(1,54) = 4.16$, $p < .05$, $\eta^2_p = .07$. It appeared that the amplitude difference between Ne and Nc was less pronounced in children with RD compared to children without RD. Follow-up analyses revealed that children with RD differed from children without RD only in Ne and not in Nc amplitude ($p > .54$). Post-hoc analyses comparing the four groups revealed no significant differences between the comorbid

group and the RD-only group ($p = 1.00$), although descriptively the RD group had smaller Ne amplitude than the comorbid group.

Pe/Pc. To analyze group differences in Pe/Pc amplitude, an ADHD (absent vs. present) x RD (absent vs. present) x Trial type (correct vs. error) ANOVA was conducted for the early (150 – 320 ms) and late part (320 – 450 ms) of the Pe separately.

With respect to the early part of the Pe, there was a significant main effect of Trial type ($F(1,54) = 90.32$, $p < .001$, $\eta^2_p = .63$) due to more positive amplitudes following errors than after correct responses. With respect to the diagnostic factors, there were no significant main effects ($ps > .13$). There was, however, a significant interaction between Trial type and ADHD, $F(1,54) = 3.97$, $p = .05$, $\eta^2_p = .07$. It appeared that the amplitude difference between Pe and Pc was less pronounced in children with ADHD compared to children without ADHD. Follow-up analyses revealed that children with ADHD differed from children without ADHD only in Pe and not in Pc amplitude ($p > .67$). Post-hoc analyses comparing the four groups revealed no significant differences between the comorbid group and the ADHD-only group ($p = 1.00$), although descriptively the comorbid group had smaller Pe amplitude than the ADHD-only group.

With respect to the late part of the Pe, there was a significant main effect of Trial type ($F(1,54) = 117.38$, $p < .001$, $\eta^2_p = .69$) due to more positive amplitudes following errors than after correct responses. With respect to the diagnostic factors, there were no significant main effects ($ps > .17$).

[Insert Figure 1 about here]

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Discussion

A typical response style that is often reported in children with ADHD and to a lesser extent in children with RD when performing neuropsychological tasks is the commission of a high error rate. The aim of the present study was to make an attempt to elucidate the cognitive processes that might underlie this response style by examining different stages of the error monitoring process. Comparing both disorders in the same investigation provides insight into unique and shared characteristics of both disorders. Comorbidity of ADHD with other disorders such as RD has often been neglected in studies on error monitoring. Because comorbidity can distort research results, a second aim was to explore the influence that comorbid RD might have on the results that are found with respect to ADHD. To this end, we included a comorbid group (i.e., children with both ADHD and RD), and compared the profile of deficits of this group to that of the single disorder groups (ADHD-only and RD-only).

Behavioural Data

With respect to overall performance, we found no significant differences on any of the measures between children with RD and children without RD. They did not make significantly more errors, attained the same reaction speed and were not more variable in their response speed compared to children without RD. This is at odds with some studies that found an inaccurate and variable response style in children with RD when they had to perform a speeded RT task in the laboratory (e.g., Burgio-Murphy et al., 2007; Van De Voorde et al., in press). In contrast, we found that children with ADHD made significantly more errors and were more variable in their response speed than children without ADHD. This is a finding that has frequently been reported by others in a diversity of neuropsychological tasks (Rommelse et al., 2007; Sergeant et al., 2003; van der Meere, 2005). The absence of

significant group differences in correct versus error RT suggests that the higher error rate in children with ADHD was not due to more impulsive behaviour.

Groups were also compared on post-error strategy adaptation. It has been shown that controls make some behavioural adjustments when they have committed an error: they slow down their response speed on the next trial (post-error slowing; Rabbitt, 1966) and they improve their accuracy (Laming, 1979). With the current task we found no evidence of post-error slowing in any of the groups (see also Jonkman et al., 2007). The presence and amount of post-error slowing might depend on the study paradigm or on specific task parameters such as the length of the interval between response and subsequent stimulus (Jentzsch & Dudschig, 2009). In our study, the absence of post-error slowing might be a consequence of the fact that the task instructions stressed response speed (to elicit sufficient errors in control subjects). For example, it was found by van Meel et al. (2007) that the amount of post-error slowing decreased from 25 ms under low time pressure to a mere 5 ms under high time pressure conditions, which was about the amount of slowing we found in our control group. It will be important to conduct more research into the factors that determine the occurrence of post-error slowing and other forms of behavioural adaptation (e.g., error correction) in typically developing children before we can make clear statements about the efficiency of such processes in clinical disorders. With respect to ADHD, it has been found by some researchers that children with the disorder show less RT slowing after an error (e.g., Krusch et al., 1996; Schachar et al., 2004; Sergeant & van der Meere, 1988; Wiersema et al., 2005), but there are also studies that report no differences between groups of children (e.g., Jonkman et al., 2007; van Meel et al., 2007; Wild-Wall et al., 2009) or adults (O'Connell et al., 2009; Wiersema et al., 2009). In line with the latter ones, we found no significant differences between groups on the post-error slowing measure, however, we did find that children with ADHD had a significantly higher proportion of double-errors. This indicates that they were less able to

correct their erroneous response pattern than children without ADHD (see also van Meel et al., 2007 who reported shorter sequences of correct responses in children with ADHD). Although post-error accuracy enhancement is also an important behavioural compensatory mechanism (Burle et al., 2002; Hajcak & Simons, 2008), it has not been included in previous studies on error monitoring in ADHD. The current study shows that differences in post-error processing are not always visible in RT measures but may be found in accuracy measures. The absence of differences in post-error slowing together with differences in post-error accuracy has also been found in other study populations such as patients with prefrontal lobe damage (e.g., Gehring & Knight, 2000). This suggests that it is important not to focus exclusively on post-error slowing and that future studies should include various measures of post-error adaptation to gain further insight into the efficiency of the error monitoring system in ADHD.

When comparing children from the comorbid group to children from the single disorder groups on the behavioural level, it appeared that they showed a rather distinct response style. Children with ADHD+RD showed a very inattentive reaction style (slow and variable RT with frequent omissions), which was slightly worse than that of the ADHD-only group. However, they were not as impulsive as children with ADHD-only, as evidenced by a smaller amount of commission errors and slower RTs when committing an error. They did not differ from the ADHD-only group in the proportion of double-errors, which means that they also had problems with improving their accuracy after the commission of an error. It is not clear what is responsible for the differences between both ADHD groups. One explanation could lie in possible group differences in ADHD symptomatology. Although both groups did not differ from each other in *number* of inattention or hyperactivity symptoms (as measured by the DBDRS), it could be that they differed in the *kind* of inattention problems they suffer from. For example, it was found by Lahey and colleagues (1988) that attention problems can

be subdivided in an inattention-disorganization and a sluggish cognitive tempo (sluggish, slow, hypoactive) factor. As children from our comorbid group were slower and more variable than children with only ADHD, it could be that the comorbid group contained more children with a sluggish cognitive tempo than the ADHD-only group. Future research should control for this possibility. Another possible explanation for the differences could be that RD is a protective factor in ADHD when it comes to the commission of inhibition errors. However, in a previous study conducted by our research group (Van De Voorde et al., in press) it was found that the comorbid group was most severely impaired in performance accuracy on a Go/no-go task. It must be noted that in the latter study the RD-only group also committed more commission errors than children without RD, which was not the case in the present study. Both subject and task characteristics could be responsible for the differences between studies.

Ne Data: An Index of Early Error Detection

There was a significant difference between the amplitude of the negativity following correct responses (Nc) and the negativity following error responses (Ne), with the latter being largest. Whereas the Ne is usually observed within the 0 to 160-ms post-response interval (Falkenstein et al., 2000), the Ne in the current study fell much earlier, that is, around response onset. This was also found by Groen et al. (2008) who explained this in terms of the Ne being closely time-locked to the electromyographic activity onset in the finger (Gehring et al., 1990, 1993) which precedes the actual button press by about 100 ms (Ridderinkhof & van der Molen, 1995).

The Ne/Nc amplitude difference was *smaller* in children with RD compared to children without RD. There were no significant differences between the comorbid group and the RD-only group. When further analyses were conducted to disentangle the nature of the

smaller Ne/Nc amplitude difference in RD, it appeared that they differed from children without RD only in Ne and not in Nc amplitude, indicating that they may be impaired in the early automatic/preconscious detection of errors (Falkenstein et al., 1991; Gehring et al., 1993). This could explain why they continue to make word decoding errors despite intensive remedial therapy on top of the normal reading instruction at school. However, we found a reduced Ne on the background of intact behavioural performance. This could be related to the intactness of the Pe, which has been claimed to be more closely related to performance than the Ne (Dywan, Mathewson, & Segalowitz, 2004; Falkenstein et al., 2000; Wild-Wall et al., 2009). It must be noted that children with RD were significantly older than children without RD, which may have influenced the Ne results. However, since Ne amplitudes increase with age, our results may rather underestimate the true Ne difference between children with and without RD.

In contrast to the children with RD, the Ne/Nc amplitude difference and the Ne amplitude itself were of equal size in children with ADHD compared to children without ADHD. This finding is in line with other studies of children (Jonkman et al., 2007; Wiersema et al., 2005; Wild-Wall et al., 2009; Zhang et al., 2009) and adults with ADHD (O'Connell et al., 2009; Wiersema et al., 2009), but inconsistent with other studies reporting a smaller (Albrecht et al., 2008; Groen et al., 2008; Liotti et al., 2005; McLoughlin et al., 2009; van Meel et al., 2007) or larger Ne (Burgio-Murphy et al., 2007) compared to persons without ADHD. However, there are some factors that complicate comparison of results between studies (Wild-Wall et al., 2009). These include differences in the task used and consequently the type of error studied (e.g., inhibition errors in Go/no-go tasks vs. hand errors in choice-reaction tasks such as the flanker task) and differences in task instructions (e.g., stressing speed versus accuracy). As the Ne is sensitive to such task-related factors (e.g., Falkenstein et al., 2000; Ullsperger & Szymanowski, 2004), it is possible that this effect is more pronounced

in some groups but not in others. There are also differences in the method of analysis, in that not all studies examined the Ne on the background of the Nc. This is, however, necessary in order to show that differences are specific for errors and that impaired performance is related to malfunctioning of the error monitoring system (Falkenstein et al., 2000; Wild-Wall et al., 2009). A last complicating factor is the fact that comorbidities have not always been taken into account. As shown in the present study, it is possible that some deficiencies that are found in ADHD are actually more specific for another disorder like RD. When this comorbidity is not controlled for, deficits can be mistakenly attributed to ADHD (Pennington & Ozonoff, 1996), and inconsistencies across study results can, therefore, partly be due to differences between studies in the presence of (subclinical) RD.

Pe Data: An Index of Error Awareness and Conscious Error Evaluation

There was a significant difference between the amplitude of the positivity following correct responses (Pc) and the positivity following error responses (Pe), with the latter being largest. Our results also indicated that the Pe/Pc amplitude difference was *smaller* in children with ADHD compared to children without ADHD, but that it was of equal size in children with RD compared to children without RD. There were no significant differences between the comorbid group and the ADHD-only group. When further analyses were conducted to disentangle the nature of the smaller Pe/Pc amplitude difference in ADHD, it appeared that they differed from children without ADHD only in Pe and not in Pc amplitude. This suggests that they may be impaired in the awareness and conscious evaluation of an error (Falkenstein et al., 2000; Nieuwenhuis et al., 2001; O'Connell et al., 2007; Overbeek et al., 2005).

The results of the present study are consistent with other studies that found reduced Pe amplitude in children (Groen et al., 2008; Jonkman et al., 2007; Overtoom et al., 2002; Wiersema et al., 2005; Zhang et al., 2009) and adults with ADHD (O'Connell et al., 2009;

Wiersema et al., 2009) but are inconsistent with studies that did not find any group differences (Albrecht et al., 2008; Burgio-Murphy et al., 2007; McLoughlin et al., 2009; Wild-Wall et al., 2009).

The same complicating factors as mentioned above in comparing Ne results between studies apply to comparing Pe results between studies. In addition, it has been claimed by Wild-Wall and colleagues (2009) that there may be a relationship between behavioural performance and Pe amplitude. Studies in which no differences in performance errors were found also reported no Pe amplitude differences (e.g., Albrecht et al., 2008; Burgio-Murphy et al., 2007; McLoughlin et al., 2009; Wild-Wall et al., 2009), whereas studies in which children with ADHD made significantly more errors are the ones in which Pe reductions are reported (e.g., Groen et al., 2008; Jonkman et al., 2007; Wiersema et al., 2005). This has been replicated in the current study as Pe reductions were found in children with ADHD in combination with impaired behavioural performance. However, children from the comorbid group showed reduced Pe amplitude in the absence of deteriorated accuracy. This suggests that differences in accuracy can not be the only explanation for the group differences in Pe amplitude.

A remark should be made with respect to the bipartite nature of the Pe in our data. Based on visual inspection of the grand average waveforms, we made a distinction between an early (150 to 320 ms) and a late part (320 to 450 ms) of the Pe. Separate early and late Pe components were also observed in other studies (e.g., Burgio-Murphy et al., 2007; O'Connell et al., 2007, 2009; van Veen & Carter, 2002; Wild-Wall et al., 2009). However, in the studies by O'Connell and van Veen and Carter, the early part of the Pe had a far more frontal topography than the one in our study and had a distinct topography from the late parietal Pe in their studies, which suggests that these represent dissociable components, reflecting distinct elements of the error monitoring process. Indeed, this early frontal Pe has been interpreted as part of a pre-conscious performance monitoring system, closely related to, but yet dissociable

from, the Ne (O'Connell et al., 2007, 2009). In contrast, the Pe with a more parietal focus, has been argued to reflect error awareness. Importantly however, in our study the early part of the Pe did not have a frontal focus and it did not differ from the late part of the Pe in topography, as both had a parietal focus. This suggests that these most likely relate to two parts of the late parietal Pe. A bipartite parietal Pe was also observed in the studies by Burgio-Murphy et al. (2007) and by Wild-Wall et al. (2009). Further research should aim to explain this bipartite nature of the Pe.

When comparing children from the comorbid group to the single disorder groups on the electrophysiological level, it was found that they did not differ from the RD group in Ne amplitude or from the ADHD group in Pe amplitude. This supports the view that ADHD and RD are, at least partly, independent disorders caused by distinct underlying factors and that ADHD+RD is not a separate (subtype of the) disorder.

The current study had some limitations that need to be mentioned. In view of the relative small sample sizes in all groups, the reported results will need to be replicated by future studies with larger samples. Due to these small sample sizes, it was not possible to distinguish between ADHD subtypes. Future research should investigate whether the reported effects apply for each of the ADHD subtypes as it has been suggested that they may differ in the cognitive profile they exhibit (Nigg et al., 2002). Lastly, the current results only apply to inhibition errors in the Go/no-go task. It has been claimed that results can differ depending on the paradigm that is used. Therefore, further research should investigate whether the same results can be found when another paradigm and/or a different type of error (e.g., hand error) is used.

In sum, the present study shows that difficulties in error monitoring may contribute to the deteriorated performance of children with ADHD and children with RD in speeded RT tasks and/or reading tasks. However, the results suggest that there is a double dissociation between ADHD and RD on the neural level. Children with RD appeared to have problems with the early automatic detection of errors, as reflected by a smaller Ne. In contrast, children with ADHD showed a normal Ne but were less efficient in the conscious evaluation/awareness of errors, as indexed by a smaller Pe. It will be important to further examine the relevance of both aspects of self-regulation as potential endophenotypes in ADHD and RD. The identification of biological or cognitive endophenotypes is not only important to guide molecular genetic studies (Doyle et al., 2005; Gottesman & Gould, 2003), but might also be informative for early identification of disorders and for setting up diagnostic procedures and treatment programs. It is warranted that future studies investigating error monitoring in ADHD should control for the presence of RD to exclude deficits that are not specifically related to ADHD. In addition, RD theories might have to be adjusted in order to be able to account for these new findings. A last implication of our results is that, since children from the comorbid group showed the cognitive deficits of both single disorders (reduced Ne and reduced Pe), treatment should be directed to both types of problems (Purvis & Tannock, 2000). It is therefore very important that during the diagnostic process the presence of symptoms of the other disorder is also evaluated in order to implement the correct treatment program.

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Table 1

Means of the Four Groups on Descriptive and Diagnostic Measures

Measure	Control (*N*=16) M (SD)	ADHD (*N*=18) M (SD)	RD (*N*=15) M (SD)	ADHD+RD (*N*=13) M (SD)	*F*
Age	119.8_{ab} (16.9)	118.1_a (15.5)	132.2_b (12.7)	130.5_{ab} (12.3)	3.85*
SES	4.0 (0.6)	3.9 (0.8)	4.2 (0.6)	3.7 (0.6)	1.10
Sex (M:F)	10:6	14:4	7:8	5:8	[a]
FSIQ	110.4 (10.1)	102.9 (12.7)	103.5 (7.6)	102.6 (6.3)	2.27
Inattention symptoms[b]	1.3_a (1.6)	16.7_c (5.4)	6.8_b (5.5)	19.9_c (4.7)	52.94***
Hyperactivity symptoms[b]	1.5_a (1.3)	13.4_b (6.7)	4.3_a (3.3)	15.9_b (5.8)	31.38***
ODD symptoms[b]	0.6_a (1.1)	7.7_b (4.8)	2.7_a (2.1)	8.6_b (6.8)	12.98***
CD symptoms[b]	0.1 (0.3)	1.3 (2.1)	0.2 (0.4)	1.0 (1.4)	
Reading score[c]	10.3_a (1.7)	8.4_b (1.3)	3.7_c (1.7)	3.7_c (1.5)	71.57***

Note. ADHD = attention-deficit/hyperactivity disorder. RD = reading disorder. SES = socioeconomic status. FSIQ = full-scale intelligence quotient. ODD = oppositional defiant disorder. CD = conduct disorder. Means with different subscripts are significantly different by Bonferroni post hoc tests.

[a] $\chi^2(3) = 5.85$.

[b] Disruptive Behavior Disorder Rating Scale (DBDRS) raw score.

[c] Mean of Klepel and One-Minute-Test standard score.

* *p* <.05, *** *p* <.001

Table 2

Means of the Four Groups on the Go/no-go Variables

Measure	Control (N=16) M (SD)	ADHD (N=18) M (SD)	RD (N=15) M (SD)	ADHD+RD (N=13) M (SD)
Commission errors (%)	23.4 (9.1)	41.7 (9.1)	28.4 (14.5)	27.4 (13.5)
Omission errors (%)	2.9 (2.3)	7.5 (4.3)	2.8 (2.0)	6.3 (6.7)
Double-errors (%)	12.7 (5.6)	23.6 (8.5)	13.4 (7.5)	17.3 (11.8)
MRT (ms)[a]	512.2 (72.2)	487.0 (69.1)	494.8 (76.8)	560.4 (80.5)
SD-RT (ms)[a]	156.8 (33.7)	174.0 (31.7)	154.7 (34.9)	186.5 (36.9)
RTE (ms)	421.2 (81.1)	412.2 (84.4)	378.4 (57.9)	444.7 (67.6)
RTC (ms)	522.5 (82.1)	500.0 (88.5)	476.0 (77.6)	546.4 (63.4)
RTE+1 (ms)	528.7 (114.8)	499.5 (94.2)	467.5 (94.3)	532.2 (77.3)

Note. ADHD = attention-deficit/hyperactivity disorder. RD = reading disorder. RT = reaction time. MRT = mean RT. SD-RT = standard deviation of RTs.

RTE = RT of commission errors; RTC = RT of correct trials after a correct trial; RTE+1 = RT of correct trials after an error trial.
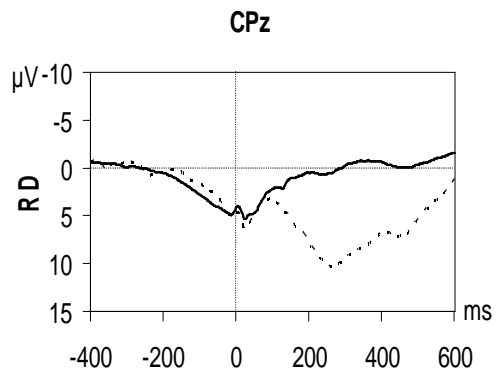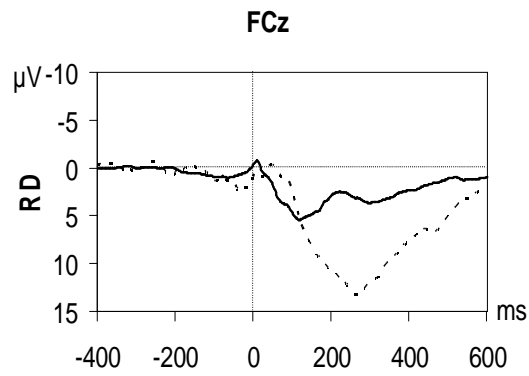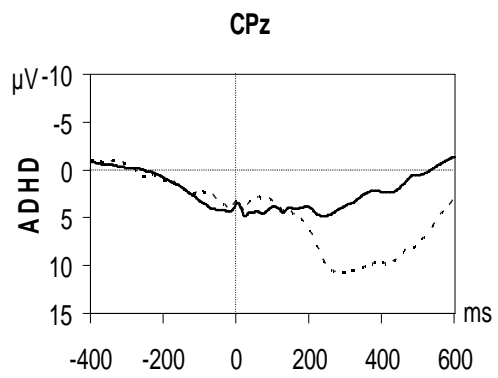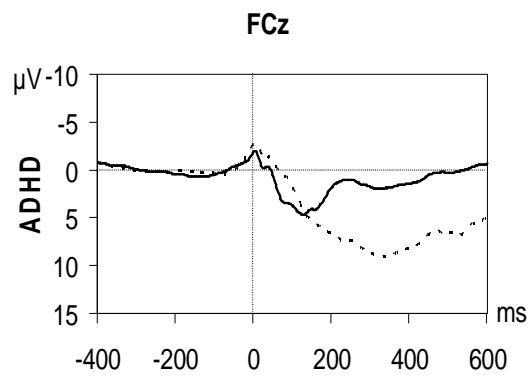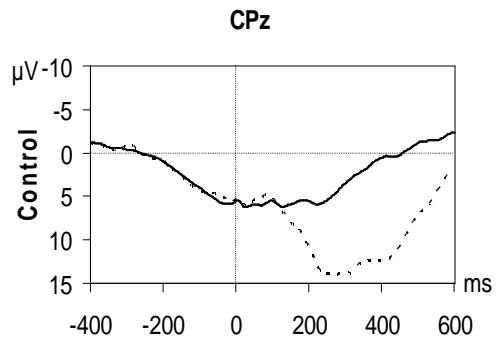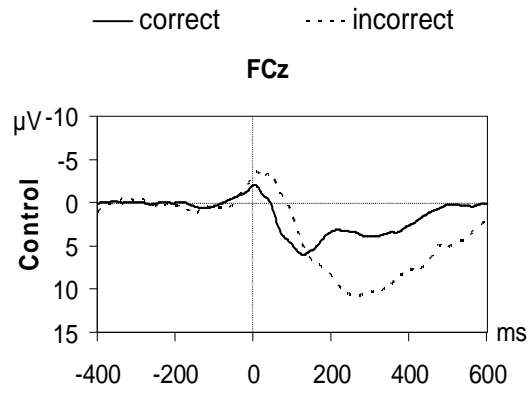
[a] age-adjusted scores
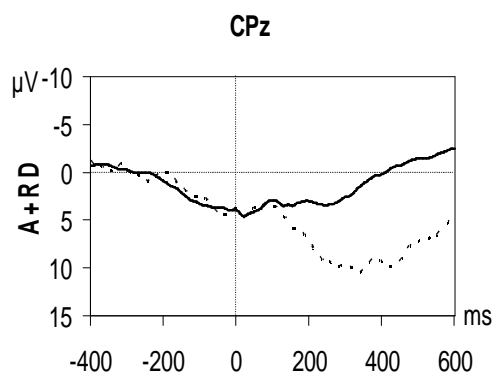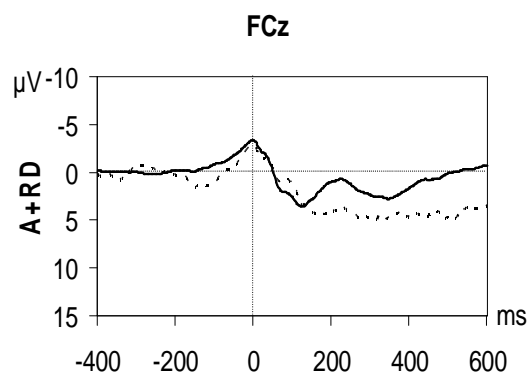
Figure 1. Grand average response-locked event-related potentials for the four groups at FCz and CPz. Solid lines represent correct response trials, dotted lines represent incorrect response trials. The moment of response is indicated by the vertical dotted line.