

Performance monitoring and post-error adjustments in adults with attention-deficit/hyperactivity disorder: an EEG analysis

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Background: Recently, research into attention-deficit/hyperactivity disorder (ADHD) has focused increasingly on its neurobiological underpinnings, revealing (among other things) frontal lobe alterations. Specifically, action-monitoring deficits have been described, including impaired behavioural adjustments following errors. Our aim was to examine the neurophysiological background of post-error behavioural alterations in an adult ADHD sample for the first time, hypothesizing that people with ADHD would differ from controls in neurophysiological markers of cognitive preparation and stimulus processing, specifically following errors. **Methods:** In total, 34 people with ADHD and 34 controls participated in an electroencephalography measurement while performing a flanker task. The final number of electroencephalography samples included in the analyses ranged from 23 to 28. We recorded event-related potentials for the erroneous response itself (error-related negativity) and for events following errors (intertrial interval: contingent negative variation; next flanker stimulus: P300). **Results:** Over frontal electrode sites, error-related negativity amplitudes were significantly reduced in people with ADHD across response conditions. Both groups showed reduced P300 amplitudes on flanker stimuli following errors. Moreover, during the intertrial interval, patients exhibited significantly reduced contingent negative variation, specifically following errors. At the behavioural level, we observed no significant group differences in post-error data. **Limitations:** Only adults were examined (no longitudinal data). **Conclusion:** Based on previous reports of post-error behavioural alterations in childhood samples, we conclude that people with ADHD develop compensatory strategies across the lifespan that lead to inconspicuous post-error behaviour in adulthood. Neurophysiologically, however, subtle alterations remain, indicating a perseverance of at least some frontal lobe deficits in people with ADHD who are partially medicated, particularly with respect to action-monitoring and post-error adaptation.

Introduction

In recent years, research into the diagnosis and treatment of attention-deficit/hyperactivity disorder (ADHD) has gained momentum, with a particular focus on its neurobiological underpinnings and complementary treatment options. Although ADHD was originally understood to be a childhood disorder, it has become apparent that its symptomatology frequently persists into adulthood as a full or incomplete syndrome.¹ In addition to core symptoms of inattention, hyperactivity and impulsivity, ADHD is frequently associated with neuropsychological and neurophysiological alterations that have been proposed to serve as intermediate phenotypes (“endophenotypes”) in the etiopathogenesis of the disorder.² Different frontal lobe functions have been studied in both children and adults with ADHD, suggesting deficits in executive function, working memory and response control/inhibition in at least some participants.²⁻⁴ In line with neuro-

psychological findings at the behavioural level, neuroimaging studies confirm structural⁵ and functional alterations⁶⁻⁸ in the frontal areas of both children and adults with ADHD.⁹

One of the more recently studied intermediate phenotypes concerns the monitoring of one’s own behaviour, particularly in the context of response errors. In electroencephalography (EEG) data, erroneous responses elicit distinct event-related potentials (ERPs) that constitute neurophysiological correlates of an action-monitoring system: error-related negativity/error negativity (ERN/Ne), a negative deflection that occurs within the first 100 ms following a response error, and error positivity (Pe), a positive peak around 250 ms after an incorrect response.¹⁰⁻¹² While the ERN/Ne has been assumed to reflect early, automatic aspects of the action-monitoring process, the Pe might correspond to the conscious appraisal of the situation and/or strategic adjustments following errors.^{11,13} Despite the functional differences between these action-monitoring components, source localization indicates that both originate

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from medial prefrontal areas, including the anterior cingulate cortex.¹⁴ Finally, an ERN/Ne-like potential of smaller amplitude has also been found to occur after correct responses (correct-response negativity, CRN/Nc),^{15,16} possibly reflecting the monitoring of response conflict¹⁷ or the processing of uncertainty (e.g., regarding the correctness of one's behaviour).¹⁸

Both children and adults with ADHD have been shown to exhibit diminished ERN/Ne and Pe amplitudes,^{19–22} although the results have been mixed, with some negative findings, particularly for the ERN/Ne.^{23–26} Functional imaging studies have confirmed alterations in the action-monitoring network of unmedicated patients with ADHD, with reduced activation in medial and lateral prefrontal areas on error trials.^{27,28} Moreover, in a behavioural study, Yordanova and colleagues²⁹ found that adolescents with ADHD showed increased behavioural instability in trials following incorrect responses ("post-error trials"), with respect to increased error rates and reaction time (RT) variability, indicating difficulties with the behavioural adjustments usually initiated after response errors (see also Wiersema and colleagues,²⁵ Schachar and colleagues³⁰ and Mohamed and colleagues³¹). However, the neurobiological correlates underlying this altered post-error behaviour have rarely been investigated. The present study aimed at characterizing the neurobiological underpinnings of post-error behavioural alterations in adults with ADHD using ERPs.

In healthy participants, post-error behavioural adjustments have been observed as post-error slowing (increased RTs on trials following errors),^{32,33} reduction of interference³⁴ and improvements in accuracy³⁵ (see also Danielmeier and Ullsperger³⁶). The neurobiological basis of these behavioural adjustments might involve recruitment of lateral prefrontal areas to achieve post-error adaptation by modulating both response-related activity in the sensorimotor cortex³⁷ and target processing in task-relevant sensory areas.³⁸ Among the prefrontal areas that showed enhanced activation in post-error trials was the anterior prefrontal cortex, possibly due to modulations in attention allocation. In line with this interpretation, post-error RTs were found to correlate with the amplitude of the P300,³⁹ a stimulus-triggered ERP that is closely linked to attentional processes.⁴⁰

Based on these previous findings in people without ADHD, we focused our analyses on post-error ERPs related to both stimulus processing (P300 amplitude elicited by flanker stimuli following incorrect button-presses) and attentional/preparatory processes in the intertrial interval (ITI; contingent negative variation [CNV]⁴¹ following incorrect responses) in addition to the above-mentioned action-monitoring potentials (ERN/Ne and Pe on error trials). Using this design, we aimed at replicating previous findings of diminished action-monitoring in adults with ADHD as indicated by reduced ERN/Ne or Pe amplitudes or both; and investigating the neurophysiological basis of post-error behavioural alterations. For the latter, we hypothesized that the behavioural instability following errors that has been previously reported in children/adolescents with ADHD²⁹ might be related to reduced target stimulus processing on trials following errors (P300) or compromised preparatory/attentional processes in the ITI (CNV). Both find-

ings would be in line with results from an fMRI study indicating compensatory recruitment of the temporal cortex in children with ADHD, possibly to resolve stimulus conflict due to difficulties in the reallocation of attention on task-relevant information in post-error trials.⁴²

Methods

Participants

We enrolled 34 adult outpatients with ADHD and 34 healthy controls in the study, which took place between March 2014 and July 2015. For recruitment, we used notice-board postings at various places in Tübingen, Germany, and university-wide emails advertising the study. Inclusion criteria for the ADHD group were as follows: an ADHD diagnosis according to DSM-IV criteria; a score of ≥ 18 on the German ADHD self-report scale ADHS-SB;⁴³ and a score ≥ 30 on a short form of the Wender Utah Rating Scale,⁴³ retrospectively assessing ADHD childhood symptoms. Exclusion criteria were comorbid axis I disorders as assessed by the Structured Clinical Interview for DSM-IV (SCID),⁴⁴ with the exception of mild to moderate depression (Beck Depression Inventory II⁴⁵ score < 28) and specific phobias. Antisocial and borderline personality disorders were also excluded based on SCID-II and a short version of the Borderline Symptom List (cutoff score > 32).⁴⁶ Controls were assessed for psychiatric and neurologic illnesses using SCID-I and general screening questionnaires, as well as an oral interview. All participants gave written informed consent. The study was carried out in accordance with the Declaration of Helsinki in its latest revision (64th World Medical Association General Assembly, Fortaleza, Brazil) and approved by the local ethics committee (University Hospital of Tübingen).

Paradigm

We adopted a modified version of the Eriksen flanker task based on Yordanova and colleagues²⁹ and our own studies.⁴⁷ Our version consisted of a target stimulus (arrow or triangle) pointing to the left or right, which was flanked by 2 more stimuli of the same type on each side, providing incongruent information by pointing either completely (all 4 flankers) or partly (2 of 4 flankers) in the opposite direction (Fig. 1). The resulting 8 types of flanker stimuli were presented in randomized order (by shuffling the stimulus array using Presentation software [Neurobehavioural Systems Inc.]) for 125 ms. Participants were to indicate the direction of the target stimulus by pressing a button (left or right finger) as quickly and accurately as possible; the shape (arrow or triangle) further determined the response hand, resulting in 4 response options. Additionally, we combined the flanker task with a Go/No-go instruction by presenting all stimuli in either blue or red. We switched the assignment of colour to instruction (Go/No-go) and stimulus shape to response hand (left/right) for each participant between 2 blocks of the experiment, and counterbalanced the sequence of events across participants.

After each button press, visual feedback appeared to indicate whether responses were correct, incorrect, or correct but slow (Fig. 1), although participants generally said that they did not need the external feedback to recognize errors (or even slow responses, especially for the second half of the experiment). We determined the RT threshold individually based on a 40-trial pretest: we used the individual median of RTs for correct responses during this pretest as the threshold for differentiating responses that were on time versus too slow, discarding the first 10 practice trials.^{47,48} Generally, a total of 400 trials were presented in 2 blocks with an ITI of 4000 ms; however, in some cases (2 in the ADHD group, 3 in the control group), up to 600 trials were presented if fewer than 10 errors were made during the first 400 trials. One participant in the ADHD group and 1 in the control group aborted the experiment after 321 and 387 trials, respectively. Post hoc analyses of the number of trials presented revealed that the groups did not differ significantly with respect to the total number of trials, Go/No-go stimuli or fully/partially incongruent flanker stimuli (all $p > 0.2$).

EEG recording and analysis

We recorded EEG using a 32-channel DC-amplifier (Brain Products) and 23 Ag/AgCl ring electrodes placed according to the international 10/20 system, with 3 additional electro-oculography electrodes. We used a frontocentral electrode position (FCz) as the recording reference (all impedances below 5 k Ω) and Brain Vision Recorder (Brain Products) for data recording (sampling rate 1000 Hz; online filter 0.1–100 Hz). At the same time, we conducted near-infrared spectroscopy measurements using the ETG-4000 (Hitachi Medical Co.), the results of which will be reported elsewhere.

We analyzed ERPs using Vision Analyzer 2.0 (Brain Products). After visual inspection of the EEG raw data, we applied high-pass (0.1 Hz) and low-pass (50 Hz; 48 db/oct) filters before correction of eye-movement artifacts based on an algorithm implemented in the software. After that, data were re-referenced to an average reference and segmented according to the relevant conditions. For analysis of the ERN/Ne and Pe, we segmented the data in a response-locked manner, (i.e., relative to correct and incorrect button presses; segment

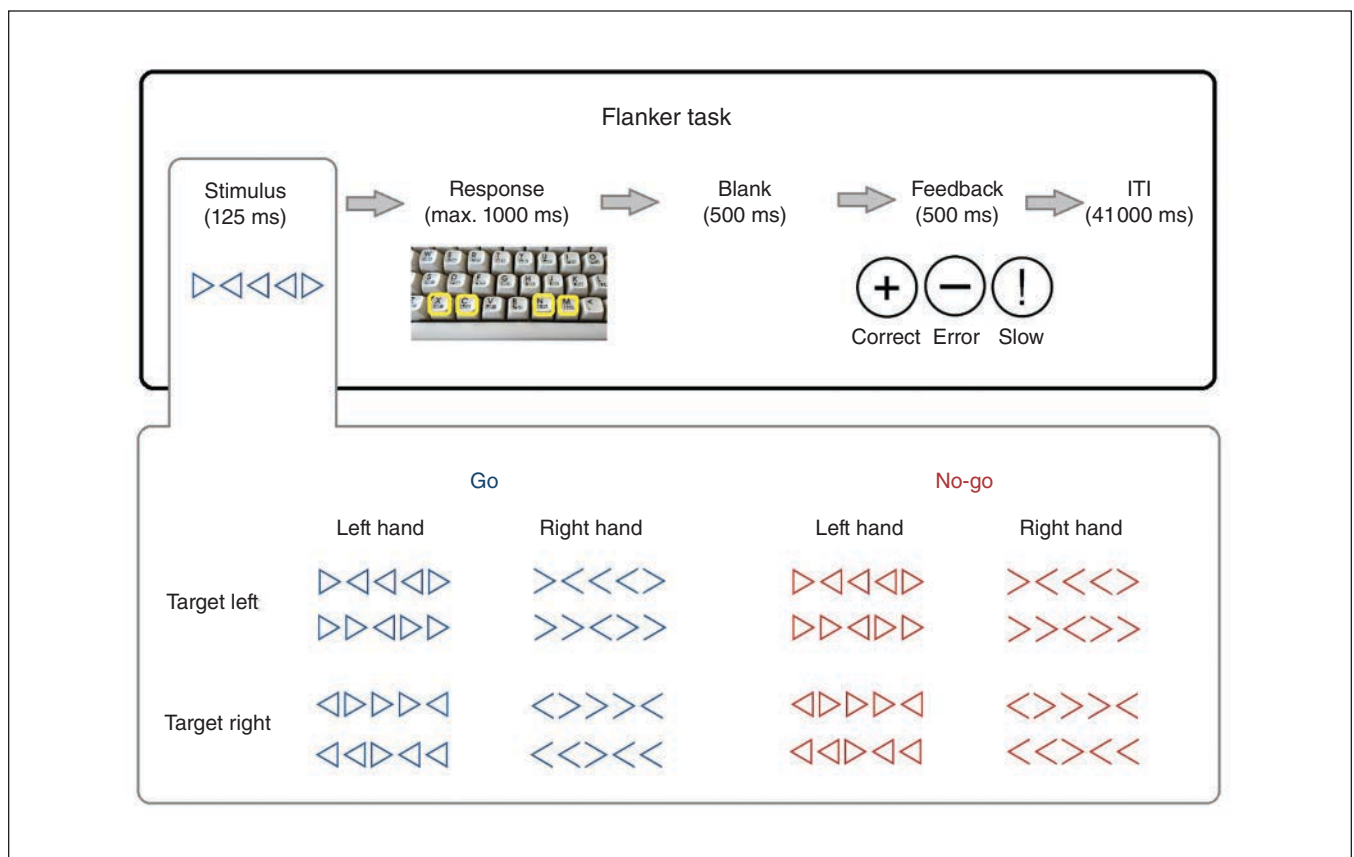


Fig. 1: Illustration of the flanker task and stimuli. Top: sequence of events within each flanker trial. The feedback always appeared exactly 1625 ms after the onset of the stimulus, with a fixed response window of 1000 ms. The 4 response keys are marked in yellow. Bottom: flanker stimuli containing information about the response hand (left or right, depending on the type of stimulus [triangle v. arrow]), the response finger (left or right, depending on the orientation of the central/target stimulus) and the action required (Go = button-press v. No-go = inhibition of motor response, depending on the colour). We switched assignment of stimulus type to response hand and colour to Go/No-go instruction between 2 blocks of the experiment for each participant (this example: Go = blue; left hand = triangle), and we counterbalanced the sequence of these assignments across participants.

length = -250 to 750 ms). Because the number of false alarms was very low (i.e., button presses to No-go stimuli), we considered only incorrect responses on Go trials. For analysis of the P300 and CNV, we performed a stimulus-locked segmentation relative to the flanker stimuli, following either correct or incorrect responses (segment length P300: -500 to 1000 ms; CNV: -3500 to 1000 ms, comprising the last 3.5 s of the ITI). Segments containing amplitudes that exceeded $\pm 70 \mu\text{V}$ or voltage steps of more than $70 \mu\text{V}$ per sampling point were excluded from the analysis. Correct but slow responses were not considered.

Peak amplitudes of the ERN/Ne were individually determined -30 to 120 ms around correct and incorrect button-presses at frontal (Fz) and frontocentral (FCz) electrode positions after baseline correction (250–150 ms prereshponse). While the ERN/Ne was sharply defined in both groups (at Fz and FCz) with a relatively typical topography, the Pe was not clearly discernible, and the positive deflection detected after the ERN/Ne (which usually corresponds to the Pe) had an atypical broad frontal distribution. Therefore, we discarded analysis of the Pe, because we could not confidently identify this component in the data set, possibly due to reduced error awareness,¹³ reduced accumulated error evidence⁴⁹ and/or a reduced subjective/emotional assessment of errors¹¹ in this unusually complex flanker task. We defined the P300 as the most positive peak 300 to 630 ms after stimulus presentation (at central [Cz] and parietal [Pz] electrode positions; baseline period: 500–250 ms prestimulus) following correct and incorrect previous trials. Finally, we analyzed the CNV in the ITI following either correct or incorrect responses to Go stimuli at position Cz. Because of the breadth of potential, we analyzed mean amplitudes between 2000 and 1000 ms, as well as between 1000 and 0 ms before the next flanker stimulus (employing a 2 Hz low-pass filter and baseline correction from 3500 to 3250 ms pre-stimulus). Based on results obtained for the reliability of the ERN/Ne,⁵⁰ we excluded data sets with fewer than 8 artifact-free error segments; for all other conditions/ERPs, we required a minimum of 15 artifact-free segments. Based on these criteria, as well as general considerations for signal-to-noise ratio and task performance, our final samples consisted of 28 (ERN/Ne), 27 (P300) and 24 (CNV) from the ADHD group and 25 (ERN/Ne), 25 (P300) and 23 (CNV) from the control group.

Statistical analysis

At the behavioural level, we analyzed RTs, standard deviation of reaction times (SD-RTs) and error rates (overall, as well as for post-correct and post-error trials; for errors, we considered only incorrect button presses to Go stimuli). We determined traditional post-error slowing (PES) by subtracting RTs on trials after correct responses from RTs following errors. Additionally, we calculated a putatively more robust PES measure by defining pairs of RTs around each error, consisting of the individual pre-error ($E - 1$) and post-error ($E + 1$) trials.⁵¹ Based on these matched pairs, we calculated PES(robust) as the difference between the mean RTs on post-error and pre-error trials. We compared behavioural

data between groups using t tests for independent samples or (in cases of non-normality) Mann–Whitney U tests. We conducted within-group comparisons using matched-samples t test or Wilcoxon test.

We analyzed EEG data using repeated-measures analyses of variance (ANOVAs), consisting of the between-subject factor of group (ADHD v. control) and within-subject factors of electrode position (ERN/Ne: Fz, FCz; P300: Cz, Pz) and condition (ERN/Ne: correct v. incorrect response; P300, CNV: post-error v. post-correct trials). If the sphericity assumption was violated, we used Huynh–Feldt correction. We further analyzed significant interactions using post hoc t tests for matched or independent samples. Effect sizes were given as η_p^2 (ANOVAs), Cohen's d (t tests/group comparisons) and Cramer's V (crosstabs), respectively.

For brain-behaviour correlations, ERN/Ne amplitudes were correlated with the overall number of errors, as well as RTs and SD-RTs separately for both groups, using Spearman's r . The CNV and P300 amplitudes following errors, slow responses and correct responses were correlated with RTs and SD-RTs of the same conditions. Exploratively, we also calculated correlations between ADHD symptom scores (assessed by Adult ADHD Self-Report Scale [ASRS]⁵² and Eysenck's Impulsiveness Questionnaire [I7]⁵³) and key flanker variables (post-error RTs, SD-RTs, P300 and CNV, as well as ERN/Ne amplitudes). Because of the multiple test situation, we adjusted the α level to $p < 0.01$ for the correlation analyses. We applied 2-sided testing throughout.

Results

Sample characteristics

The 2 groups did not differ significantly with respect to age, school education, distribution of sex, smoking status or handedness.⁵⁴ Based on the *Mehrfachwahl-Wortschatz Intelligenztest*,⁵⁵ they also did not differ in terms of mean IQ (Table 1). As expected, people with ADHD had significantly higher scores on the hyperactivity and inattention subscale of the ASRS, as well as on the impulsivity subscale of the I7. Within the ADHD group, the following use of medication was reported: methylphenidate ($n = 6$), dexamphetamine ($n = 1$), atomoxetine ($n = 1$) and bupropion ($n = 1$). In all but 4 cases, ADHD medication was discontinued 2 to 7 days before the trial. Both groups consisted largely of university students ($n = 22$ controls, $n = 24$ ADHD), but 11 from the control group and 2 from the ADHD group reported working as executive or regular employees; 2 from the ADHD group were unemployed; 3 from the ADHD group were self-employed; 1 from the ADHD group was a trainee; and 2 from the ADHD group were homemakers (information was missing for 1 member of the control group).

Flanker task performance

Groups did not differ significantly with respect to mean RTs; however, the ADHD group showed increased SD-RTs as a measure of response variability for all responses, as well as

for button-presses on trials following correct responses (Table 2). The 2 groups did not differ significantly with respect to the number of incorrect responses, although the ADHD group showed a statistical trend toward increased error rates overall, and for trials following correct responses. The 2 groups did not differ significantly with respect to the traditional index of PES; for PES(robust), the ADHD group tended to show increased values.

For within-group comparisons, both groups showed significant PES in terms of longer RTs on correct Go trials following errors versus correct responses (traditional PES measure, ADHD: $t_{33} = 4.16, p < 0.001$; control: $t_{33} = 2.15, p < 0.05$) and longer RTs on individually paired post-error versus pre-error trials (PES[robust], ADHD: $t_{33} = 5.98, p < 0.001$; control: $t_{33} = 5.65, p < 0.001$), respectively. Also in both groups, SD-RTs did not differ significantly on post-error versus post-correct trials (ADHD: $t_{33} = 0.82$, not significant; control: Wilcoxon $Z = 1.19$, not significant). Finally, both groups showed post-error adaptation in terms of fewer errors following incorrect re-

sponses versus correct responses (both $Z = 5.09, p < 0.001$). For ADHD symptom correlations, in controls, I7 impulsivity scores correlated significantly with SD-RTs following errors ($r = 0.447, p < 0.01$), indicating higher variability of the response behaviour following errors with increasing impulsivity.

EEG data

Action-monitoring potentials

For analysis of the ERN/Ne, a $2 \times 2 \times 2$ (condition \times position \times group) ANOVA revealed a significant main effect of group ($F_{1,51} = 4.89, p < 0.05, \eta_p^2 = 0.09$), with overall greater negativity in the control group ($-5.16 \pm 2.79 \mu\text{V}$) than in the ADHD group ($-3.55 \pm 2.50 \mu\text{V}$). We also found significant interactions of condition \times position ($F_{1,51} = 42.06, p < 0.001, \eta_p^2 = 0.45$) and position \times group ($F_{1,51} = 5.88, p < 0.05, \eta_p^2 = 0.10$). Post hoc t tests showed a significant difference between conditions only for electrode position FCz, with significantly more negative amplitudes for errors ($-5.24 \pm 3.19 \mu\text{V}$) than for correct

Table 1: Sample characteristics

Characteristic	Control ($n = 34$)	ADHD ($n = 34$)	Test statistic with effect size (group comparison)
Age, yr, mean \pm SD	27.62 \pm 7.43	30.29 \pm 9.47	$t_{66} = 1.30$, NS; $d = 0.31$
IQ, mean \pm SD*	118.6 \pm 15.7	115.7 \pm 14.6	$t_{65} = -0.79$, NS; $d = -0.19$
Years of education, mean \pm SD†	12.88 \pm 1.21	12.75 \pm 0.76	$t_{64} = -0.51$, NS; $d = -0.13$
ASRS hyperactivity, mean \pm SD‡	8.7 \pm 4.3	20.5 \pm 4.9	$t_{66} = 10.60, p < 0.001$; $d = 2.56$
ASRS inattention, mean \pm SD§	10.4 \pm 4.3	24.6 \pm 4.6	$t_{66} = 13.15, p < 0.001$; $d = 3.19$
I7 impulsivity, mean \pm SD¶	4.9 \pm 2.2	11.9 \pm 2.9	$t_{66} = 10.66, p < 0.001$; $d = 2.72$
Sex, female/male, no.	18/16	13/21	$\chi^2 = 1.48$, NS; $V = 0.15$
Handedness, right/left, no.	30/4	28/6	$\chi^2 = 0.47$, NS; $V = 0.08$
Smoker/nonsmoker, no.	10/24	11/22**	$\chi^2 = 0.12$, NS; $V = 0.04$

ADHD = attention-deficit/hyperactivity disorder; ASRS = Adult ADHD Self-Report Scale; I7 = Eysenck's Impulsiveness Questionnaire; IQ = intelligence quotient; NS = not significant; SD = standard deviation.

*IQ was assessed based on the *Mehrfachwahl-Wortschatz Intelligenztest* (information missing for 1 participant).

†Information missing for 3 participants.

‡Hyperactivity subscale of the Adult ADHD Self-Report Scale.

§Inattention subscale of the Adult ADHD Self-Report Scale.

¶Impulsivity subscale of Eysenck's Impulsiveness Questionnaire I7 (data missing for 4 people with ADHD and 2 controls).

**Information missing for 1 participant.

Table 2: Flanker task performance

Measure, mean \pm SD	Controls ($n = 34$)	ADHD ($n = 34$)	Test statistic with effect size (group comparison)
Mean RT, ms	504.74 \pm 90.13	532.32 \pm 87.62	$Z = 1.41$, NS; $d = 0.31$
RT post-correct, ms	509.59 \pm 97.17	532.85 \pm 91.92	$t_{66} = 1.01$, NS; $d = 0.25$
RT post-error, ms	531.18 \pm 90.61	576.79 \pm 111.06	$t_{66} = 1.86, p < 0.1$; $d = 0.45$
SD-RT, ms	110.97 \pm 26.26	127.44 \pm 29.50	$Z = 2.49, p < 0.05$; $d = 0.59$
SD-RT post-correct, ms	107.71 \pm 23.94	124.88 \pm 29.83	$Z = 2.58, p < 0.01$; $d = 0.63$
SD-RT post-error, ms	119.09 \pm 50.29	130.38 \pm 44.84	$Z = 0.80$, NS; $d = 0.24$
Total errors, no.	40.56 \pm 23.36	53.00 \pm 28.67	$Z = 1.77, p < 0.1$; $d = 0.48$
Errors post-correct, no.	24.06 \pm 15.45	31.12 \pm 16.58	$Z = 1.85, p < 0.1$; $d = 0.44$
Errors post-error, no.	5.29 \pm 5.20	8.97 \pm 9.01	$Z = 1.60$, NS; $d = 0.50$
PES(traditional), ms	21.59 \pm 58.53	43.94 \pm 61.64	$t_{66} = 1.53$, NS; $d = 0.37$
PES(robust), ms	28.97 \pm 29.91	44.74 \pm 43.64	$t_{66} = 1.74, p < 0.1$; $d = 0.42$

ADHD = attention-deficit/hyperactivity disorder; NS = not significant; PES(robust) = more robust measure of post-error slowing (mean RTs on individually paired post-error trials [E + 1] – pre-error trials [E – 1]; see methods section); PES(traditional) = traditional measure of post-error slowing (mean RTs after incorrect responses – mean RTs after correct responses); RT = reaction time to Go stimuli (only correct responses included); SD = standard deviation; SD-RT = standard deviation of reaction times to Go stimuli (only correct responses included).

responses ($-3.44 \pm 3.62 \mu\text{V}$; $t_{52} = -3.55$, $p = 0.001$), while ERN/Ne amplitudes showed a similarly pronounced negativation for both conditions at Fz ($t_{52} = 0.35$, not significant; Fig. 2). For the position \times group interaction, post hoc testing revealed a significant difference between groups only at Fz, with higher amplitudes in the control group ($-5.46 \pm 2.96 \mu\text{V}$) than in the ADHD group ($-3.22 \pm 2.57 \mu\text{V}$; $t_{51} = 2.95$, $p < 0.01$, $d = 0.81$). We observed no between-group differences at FCz ($t_{51} = 1.24$, not significant, $d = 0.34$; Fig. 2). The group difference at Fz occurred independent of the response condition for both errors (ERN/Ne) and correct responses (CRN/Nc).

Regarding brain-behaviour correlations, the amplitudes of the CRN/Nc at FCz showed a significant negative correlation with SD-RTs ($r = -0.482$, $p < 0.01$), but only in the ADHD group (this numerically negative correlation indicates a

greater response time variability with a more pronounced CRN/Nc—that is, stronger action-monitoring on correct trials). Moreover, also in the ADHD group only, CRN/Nc correlated significantly with ASRS hyperactivity scores (Fz; $r = 0.544$, $p < 0.01$), indicating a more pronounced CRN/Nc with lower symptomatology. At a 1% significance level, no other correlations reached the statistical threshold.

Processing of flanker stimuli

For analysis of the P300 elicited by flanker stimuli (Fig. 3), a $2 \times 2 \times 2$ (condition \times position \times group) ANOVA revealed a significant position \times group interaction ($F_{1,50} = 4.61$, $p < 0.05$, $\eta_p^2 = 0.08$) and a main effect of condition ($F_{1,50} = 10.98$, $p < 0.01$, $\eta_p^2 = 0.18$), but no significant 3-way interaction (condition \times position \times group; $F_{1,50} = 0.001$, $p > 0.9$, $\eta_p^2 < 0.001$).

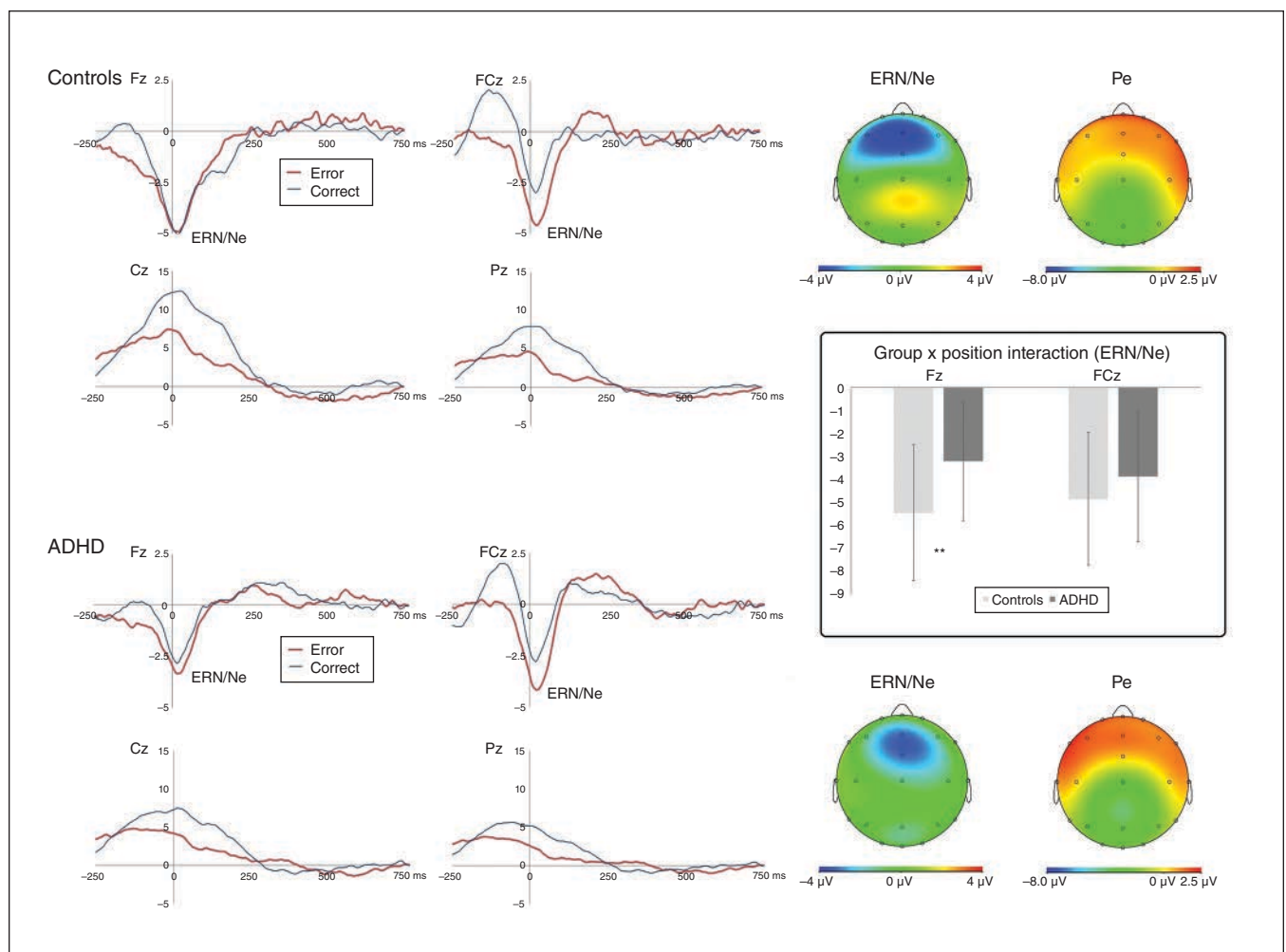


Fig. 2: Illustration of ERN/Ne (and CRN/Nc) findings. Left side: grand averages of response-locked potentials elicited by correct button-presses (thin blue lines) and errors (bold red lines) at electrode positions Fz, FCz, Cz and Pz for controls (upper 4 panels) and people with ADHD (bottom 4 panels); y-axis with unit μV . For illustration purposes, we applied a 30 Hz low-pass filter (x-axis 0 marks the correct/error button-press). Topographical maps show the field distribution of the ERN/Ne (blue field, left) and subsequent Pe (red field, right). The middle panel on the right illustrates a significant group \times position interaction indicated by the ANOVA on ERN/Ne amplitudes. **Indicates a significant group difference for mean correct/error potentials at electrode position Fz, $p < 0.01$. ANOVA = analysis of variance; CRN/Nc = correct response negativity; Cz = central electrode position; ERN/Ne = error-related negativity/error negativity; FCz = frontocentral electrode position; Fz = frontal electrode position; Pe = error positivity; Pz = parietal electrode position.

Overall, participants showed significantly higher P300 amplitudes on flanker stimuli following correct trials ($8.88 \pm 3.72 \mu\text{V}$) than following errors ($7.98 \pm 4.20 \mu\text{V}$). With respect to the group \times position interaction, the ADHD group did not differ significantly from controls at either Cz (8.88 ± 4.54 v. $10.52 \pm 4.42 \mu\text{V}$; $t_{50} = 1.32$, $p = 0.19$, $d = 0.37$) or Pz (6.83 ± 3.23 v. $7.58 \pm 3.16 \mu\text{V}$; $t_{50} = 0.86$, $p = 0.40$, $d = 0.23$). However, the increase in P300 amplitudes from Pz to Cz was significantly more pronounced in the control group than in the ADHD group ($t_{50} = 2.15$, $p < 0.05$, $d = 0.61$), indicating a somewhat more frontal topography of the P300 in the control group.

Regarding brain-behaviour correlations, P300 amplitudes showed significant negative correlations with RTs in the ADHD group following slow responses (Cz: $r = -0.570$, $p < 0.01$; Pz: $r = -0.570$, $p < 0.01$) and, as a statistical trend, incorrect responses (Pz: $r = -0.474$, $p = 0.013$). Similarly, SD-RTs tended to correlate negatively with P300 amplitudes in the ADHD group following both incorrect responses (Cz: $r =$

-0.506 , $p < 0.01$) and slow responses (Cz: $r = -0.469$, $p = 0.014$; Pz: $r = -0.455$, $p = 0.017$), indicating shorter RTs and smaller SD-RTs with higher amplitudes (but only after a suboptimal response outcome).

Inter-trial interval

For analysis of the CNV in the ITI, a $2 \times 2 \times 2$ ANOVA with the factors condition and group, as well as time bin (-2000 to -1000 ms vs. -1000 to 0 ms before the next stimulus) revealed a significant condition \times group interaction ($F_{1,45} = 5.26$, $p < 0.05$, $\eta_p^2 = 0.11$) in addition to the expected main effect of time bin ($F_{1,45} = 7.36$, $p < 0.01$, $\eta_p^2 = 0.14$), with more negative CNV amplitudes as the duration of the ITI increased. Post hoc tests for the interaction showed a significantly more negative CNV amplitude in the control group compared to the ADHD group, specifically following errors (-1.36 ± 2.09 v. $0.53 \pm 1.86 \mu\text{V}$; $t_{45} = 3.29$, $p < 0.01$, $d = 0.96$; Fig. 4), with comparable amplitudes following correct trials (-0.75 ± 2.85 v. $-0.25 \pm 2.51 \mu\text{V}$; $t_{45} = 0.65$, not significant, $d = 0.19$). At an uncorrected

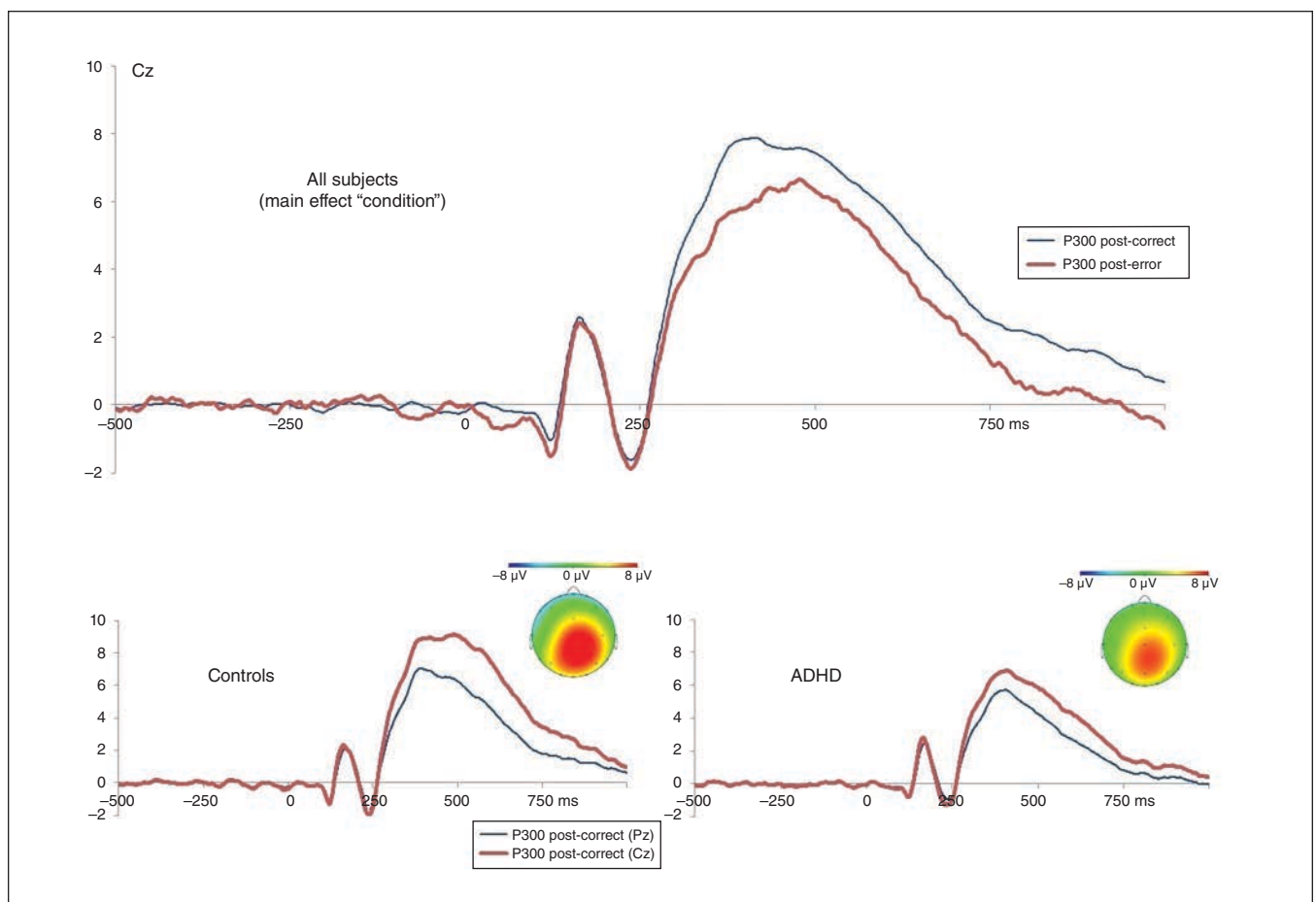


Fig. 3: Illustration of P300 findings. Top: grand averages of ERPs elicited by flanker stimuli following either correct responses (thin blue line) or errors (bold red line) on the previous trial (0 marks the stimulus presentation); note the pronounced P300 component that was significantly smaller after errors v. correct responses (main effect condition). Bottom: stimulus-elicited ERPs at electrode positions Cz (bold red lines) and Pz (thin blue lines) in controls (left) and people with ADHD (right), illustrating a group \times position interaction. (Depicted here: trials following correct responses; similar effects were also observed on post-error trials.) Maps show the field distribution of the P300 at the peak of the GFP. ADHD = attention-deficit hyperactivity disorder; Cz = central electrode position; ERP = event-related potential; GFP = global field power; Pz = parietal electrode position.

significance level, amplitudes of the later part of the CNV (second time bin) following slow responses correlated positively with RTs of the same condition in healthy controls ($r = 0.462$, $p = 0.026$), indicating slower RTs with more positive (i.e., smaller) CNV amplitudes.

Discussion

This study investigated neurophysiological correlates of action-monitoring and post-error adaptation in adult ADHD outpatients compared with age-matched controls

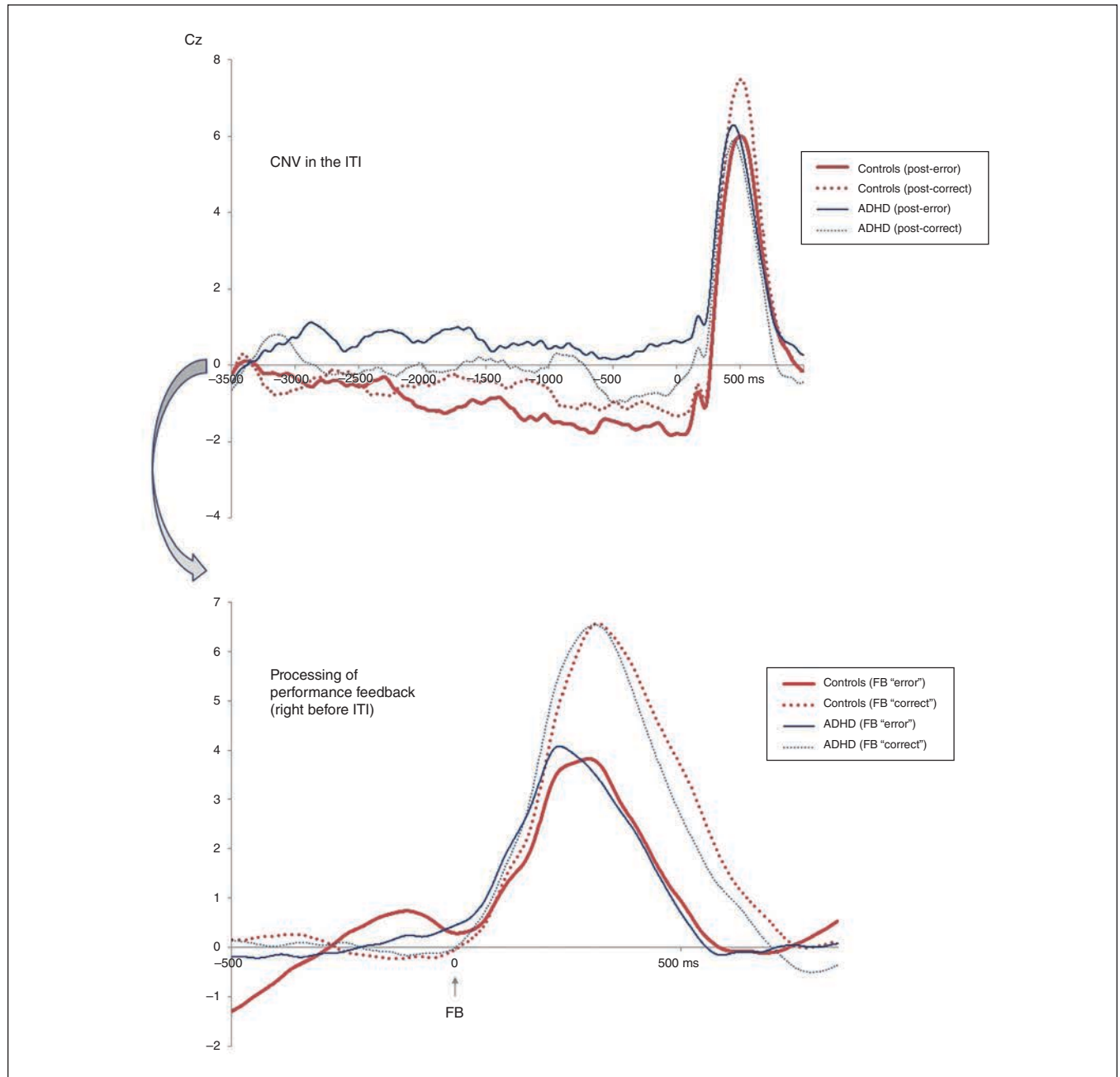


Fig. 4: Illustration of CNV effects in the ITI. Top: grand average of the ITI following either error (solid lines) or correct responses (dotted lines) in Go trials for people with ADHD (thin blue lines) and controls (bold red lines) at position Cz (0 marks the beginning of the next trial: Go or No-go flanker stimulus). Note the reduced CNV in people with ADHD (v. controls) that occurred specifically after errors (comparison of the 2 solid lines; $p < 0.01$). Bottom: grand averages locked to the feedback stimulus directly preceding the ITI, for feedback indicating correct responses (dotted lines) and errors (solid lines) in people with ADHD (thin blue lines) and controls (bold red lines). While the type of feedback affected the resulting ERP, groups showed no discernible differences; therefore, group differences in feedback processing were unlikely to explain subsequent group differences in CNV for ITIs after error trials. CNV = contingent negative variation; Cz = central electrode position; ERP = event-related potential; FB = feedback; ITI = intertrial interval.

using a modified flanker task. Specifically, we aimed at revealing the neurophysiological basis of post-error behavioural alterations previously observed in children/adolescents with ADHD.²⁹ Partly replicating previous findings,^{19–22} we observed reduced amplitudes of action-monitoring potentials (ERN/Ne, CRN/Nc) in people with ADHD, indicating a diminished processing of response conflict for both correct and incorrect responses in this difficult, high-conflict task (inconsistencies with previous reports of partly negative ERN/Ne findings may depend on the task conditions; specifically, ERN/Ne amplitudes elicited by No-go/commission errors were repeatedly not found to differ between patients with ADHD and controls).^{23,25,26} We observed an unusually large early negativity following correct responses (CRN/Nc), which might be indicative of increased uncertainty of the participants¹⁸ or, possibly more likely, increased response conflict¹⁷ in our combined flanker-Go/No-go task, with 4 response options on Go trials alone. Interestingly, this action-monitoring component was associated with lower symptomatology (ASRS hyperactivity scores), but also with higher SD-RTs in the ADHD group, indicating increased response variability with more pronounced action-monitoring on correct trials.

Regarding post-error processes (the main topic of this study), we observed specific alterations in the ITI that were in line with our hypotheses. Reduced CNV amplitudes specifically following incorrect responses indicated compromised preparatory processes for the next trial after an error in patients with ADHD. At the same time, a general relationship between the CNV and response readiness on the next trial was confirmed by a tendency for quicker responses with higher CNV amplitudes in controls, particularly following slow responses. Based on previous data showing a strong link between the CNV and attention,⁵⁶ we conclude that response errors seemed to have a negative effect on attentional processes related to the cognitive preparation for the next trial in the current sample of adults with ADHD.

However, despite this neurophysiological alteration, we found no behavioural indications of an altered post-error adaptation as previously described in younger ADHD populations.^{25,29,30} Specifically, both groups displayed PES (with PES[robust] showing a statistical trend for higher values in patients with ADHD) and made significantly fewer errors following incorrect responses compared with correct responses. Regarding more general behavioural measures, we replicated the well-known finding of enhanced SD-RTs in ADHD, suggesting an increased variability of RTs at the intraindividual level.⁵⁷ Interestingly, in controls, impulsivity scores correlated positively with SD-RTs, showing an effect also at a subclinical level; the fact that this finding was particularly observed for post-error trials is in line with this study's hypothesis of a specific effect of ADHD symptoms on post-error adaptation.

In the present sample of adult ADHD patients, we could not replicate increased error rates or SD-RTs specifically following errors as previously reported in a younger group (7 to 16 years of age).²⁹ We interpret these findings to indicate a normalization of post-error adaptive strategies across the

lifespan that leads to inconspicuous behavioural data in adults with ADHD (although the cross-sectional nature of the present study, focusing on a single age group, leaves this interpretation to be somewhat speculative). This conclusion is also in line with our previous findings suggesting a normalization of action-monitoring potentials and post-error adaptive behaviour in older adults with ADHD.²¹ It is also possible that people with ADHD develop compensatory strategies across the lifespan that allow them to optimize their response to unfavourable action outcomes, which may be particularly frequent as a result of their symptomatology. However, longitudinal data in optimally unmedicated patients are needed to confirm this interpretation. Alternatively, ITIs were long in the present study (4 s), possibly enabling patients to initiate behavioural adjustments that might not have occurred otherwise (because of the greater challenge shorter ITIs would have posed for adaptation/control capacity).⁵⁸ Also, given the strict exclusion criteria for psychiatric comorbidities, we might have investigated an ADHD subsample that showed particularly good compensatory behaviour, as reflected in their relatively high-functioning state. Finally, behavioural effects may be subtler than assumed overall, since Plessen and colleagues⁵⁹ reported reduced post-error adaptation in motor networks (fMRI data) without group differences at the behavioural level, also for children with ADHD.

With respect to attentional processes on stimuli following correct/incorrect responses, P300 amplitudes to flanker stimuli were significantly reduced for post-error versus post-correct trials, independent of diagnosis. Patients differed from controls only in a somewhat less frontal distribution of the P300 field (see Fallgatter and colleagues⁶⁰ for data showing a similarly reduced "frontalization" of the P300 in people with ADHD on other Go/No-go tasks, possibly indicating reduced frontal lobe involvement). Overall reduced amplitudes of the flanker P300 on post-error trials indicate that both groups had mild difficulties reallocating their attention on task-relevant information following incorrect responses, an observation previously made specifically in ADHD samples.⁴² The notion that the flanker P300 seemed to be a good indicator of attentional processes was confirmed by partly significant brain-behaviour correlations in terms of quicker responses and less variable RTs with higher P300 amplitudes.

Limitations

We investigated only adults in the present study, which prevents any definite conclusions about developmental processes that might underlie changes in the neurocognitive characteristics of ADHD across the lifespan. Regarding the potential effect of sex, we included both female and male participants, whereas previous studies have focused on male participants only.²⁹ For statistical analysis, we took a rather liberal approach in terms of corrections for multiple testing in this explorative study (especially given the relatively large number of tests performed for the correlation analyses). Future studies are needed to confirm whether the

findings can be replicated and to test the main hypotheses more stringently. Finally, because our sample was recruited from a relatively small university town using (among other options) university-wide emails, the sample might not be fully representative, and might also explain missing group differences in terms of general demographic and psychometric variables.

Conclusion

We conducted, to our knowledge, the first ERP study on post-error adaptation in adults with ADHD. Replicating previous findings of reduced action-monitoring potentials, a specific focus was on post-error processes in trials following incorrect responses. Here, post-error behavioural alterations previously reported in younger samples could not be replicated, possibly suggesting the development of compensatory strategies across the lifespan. Neurophysiologically, however, post-error alterations were still apparent, underlining the potentially endophenotypic nature of frontal lobe deficits in ADHD. Longitudinal data are needed to further investigate changes in error-monitoring and error-adaptation across the lifespan, as well as their contribution to ADHD symptoms and clinical outcomes.

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