Received: 27 June 2022 | Revised: 8 November 2022 | Accepted: 15 January 2023

DOI: 10.1111/ejn.15921

RESEARCH REPORT

EJN European Journal of Neuroscience FENS WILEY

Temporal dynamics of cognitive flexibility in adolescents with anorexia nervosa: A high-density EEG study

Cristina Berchio[1] | **Lucie Clémentine Annen**[1] | **Ynès Bouamoud**[1] | **Nadia Micali**[1,2,3,4]

[1]Department of Psychiatry, Faculty of Medicine, University of Geneva, Geneva, Switzerland

[2]Department of Pediatrics, Gynecology and Obstetrics, Faculty of Medicine, University of Geneva, Geneva, Switzerland

[3]Great Ormond Street Institute of Child Health, University College London, London, UK

[4]Mental Health Services in the Capital Region of Denmark, Eating Disorders Research Unit, Psychiatric Centre Ballerup, Ballerup, Denmark

Correspondence
Cristina Berchio, Department of Psychiatry, Faculty of Medicine, University of Geneva, Geneva, Switzerland.
Email: cristina.berchio@unige.ch

Funding information
Foundation Gertrude von Meissner; Fondation Ernst et Lucie Schmidheiny

Edited by: Susan Rossell

Abstract

Impairment in cognitive flexibility is a core symptom of anorexia nervosa (AN) and is associated with treatment resistance. Nevertheless, studies on the neural basis of cognitive flexibility in adolescent AN are rare. This study aimed to investigate brain networks underlying cognitive flexibility in adolescents with AN. To address this aim, participants performed a Dimensional Change Card Sorting task during high-density electroencephalography (EEG) recording. Anxiety was measured with the State–Trait Anxiety Inventory. Data were collected on 22 girls with AN and 23 controls. Evoked responses were investigated using global-spatial analysis. Adolescents with AN showed greater overall accuracy, fewer switch trial errors and reduced inverse efficiency switch cost relative to controls, although these effects disappeared after adjusting for trait and state anxiety. EEG results indicated augmented early visual orienting processing (P100) and subsequent impaired attentional mechanisms to task switching (P300b) in subjects with AN. During task switching, diminished activations in subjects with AN were identified in the posterior cingulate, calcarine sulcus and cerebellum, and task repetitions induced diminished activations in a network involving the medial prefrontal cortex, and several posterior regions, compared with controls. No significant associations were found between measures of cognitive flexibility and anxiety in the AN group. Findings of this study suggest atypical neural mechanisms underlying cognitive flexibility in adolescents with AN. More importantly, our findings suggest that different behavioural profiles in AN could relate to differences in anxiety levels. Future research should investigate the efficacy of cognitive training to rebalance brain networks of cognitive flexibility in AN.

Abbreviations: AN, anorexia nervosa; BMI, body mass index; CF, cognitive flexibility; DCCS, Dimensional Change Card Sorting task; ED, eating disorder; EEG, electroencephalography; ERP, event-related potential; GFP, global field power; HC, healthy controls; ICA, independent component analysis; IES, inverse efficiency score; ROI, regions of interest; RT, reaction times; SSRI, selective serotonin reuptake inhibitors; WCST, Wisconsin Card Sorting Test.

KEYWORDS
adolescence, anorexia nervosa, cognitive flexibility, ERP, source imaging

1 | INTRODUCTION

Anorexia nervosa (AN) is a mental disorder characterized by body image disturbance and behaviours that interfere with weight gain (A.P.A., 2013). Behavioural rigidity and psychological inflexibility are associated with AN (Buzzichelli et al., 2018) and impact on symptoms and treatment resistance (Tchanturia et al., 2013; Treasure & Schmidt, 2013).

Behavioural and mental rigidity in AN have been investigated by examining cognitive flexibility (CF) (Miles et al., 2020). CF is the ability to adapt one's mental strategies to unexpected situations and environmental changes, and it crucially depends on attentional control (Canas et al., 2006). The Wisconsin Card Sorting Test (WCST) is a valid neuropsychological measure of CF that has been extensively used to study AN (Miles et al., 2020; Roberts et al., 2007; Westwood et al., 2016). A large body of literature that has examined behavioural responses on the WCST has documented higher rates of perseverative errors and perseverative responses in adults with AN compared with healthy controls (HC) (Abbate-Daga et al., 2011, 2014; Aloi et al., 2015; Fagundo et al., 2012; Galimberti et al., 2013; Nakazato et al., 2010; Roberts et al., 2010; Spitoni et al., 2018; Steward et al., 2019; Tchanturia et al., 2012; Tenconi et al., 2010). Using different tasks, other studies have found higher switch costs in accuracy (i.e., reduced ability of task switching) (Shott et al., 2012) and worse performance (i.e., higher perseverative errors/total number of errors) in adults with AN when compared with HC (Danner et al., 2012).

Deficits of CF in adolescents with AN require further investigation. The majority of studies on adolescents with AN have consistently shown similar behavioural performance to that of HC (Andrés-Perpiña et al., 2011; Bohon et al., 2020; Dmitrzak-Weglarz et al., 2011; Fitzpatrick et al., 2012; Van Autreve et al., 2016). Using the WCST, several studies indicated a similar number of perseverative errors and of perseverative responses between adolescents with AN and HC (Andrés-Perpiña et al., 2011; Bohon et al., 2020; Dmitrzak-Weglarz et al., 2011; Fitzpatrick et al., 2012); two studies have documented more perseverative errors in children and adolescents with AN than HC (Lang et al., 2015; McAnarney et al., 2011). Another study, which has applied a different switching paradigm, indicated that there were no differences in switch costs of reaction times (RT) between AN and HC (Van Autreve et al., 2016).

Neuroimaging studies of CF in AN are scarce, especially in adolescence. Findings in adult populations provide evidence that dysfunctional fronto-parietal control networks might underlie CF difficulties in AN (Lao-Kaim et al., 2015; Sato et al., 2013; Zastrow et al., 2009). A recent study using a visuo-spatial shifting task, with shapes and colours as stimuli, documented reduced activations in adolescents with AN, compared with controls, in the inferior and middle occipital lobe, the lingual regions, the fusiform cortex and the cerebellum (Castro-Fornieles et al., 2019). The few available studies on adolescents following treatment/weight gain suggest that brain alterations may be reversible (Bohon et al., 2020; Castro-Fornieles et al., 2019). However, it remains unclear whether behavioural and neural correlates of CF might be explained by other clinical traits typical of active AN.

Individuals with high trait anxiety tend to show deficits in coping with interference responses (Wilson et al., 2018). High trait anxiety may lead healthy individuals to adopt alternative cognitive strategies (Eysenck et al., 2007), such as an increase in task effort (Berchio et al., 2019). Neuroimaging evidence indicates that trait anxiety negatively impacts the prefrontal cortex's role in inhibiting distractors (Bishop, 2009), and electrophysiological data have shown that trait anxiety is associated with reduced neural responses to task switching (Dennis & Chen, 2009).

There is good evidence that trait anxiety is associated with AN (Raney et al., 2008; Schulze et al., 2009). Whether trait anxiety contributes to CF (at the behavioural and neural level) in adolescents with AN requires further investigation.

High-density electroencephalography (EEG) and event-related potentials (ERPs) allow an investigation of brain large-scale networks with millisecond precision (Michel & Murray, 2012). High-density ERPs are therefore capable of capturing the brain dynamics of CF. Furthermore, this method is non-invasive, making it particularly suitable for the study of childhood and adolescent psychiatric disorders (Berchio & Micali, 2022; Berchio et al., 2022).

Early visual ERP components are believed to reflect sensory and perceptual processing (Woodman, 2010). P100, the first visual component, is usually localized in the middle occipital cortex (Creel, 2019).

ERP components that are usually linked to CF are the N200 and the P300 (Downes et al., 2017; Kopp et al., 2020). The N200 is a negative wave recorded over anterior frontal electrodes that peaks approximately 200 ms post-stimulus onset (Folstein & Van Petten, 2008) and is associated with conflict monitoring detection and activity of the anterior cingulate (Espinet et al., 2012; Folstein & Van Petten, 2008). The P300 occurs roughly 300–700 ms post-stimulus onset, with a broad central-parietal scalp distribution (Woodman, 2010). The P300a and the P300b are sub-components of the P300, which are believed to reflect attentional and memory processes, respectively (Polich, 2007). Brain sources of the P300a are estimated mainly in the prefrontal cortex and those of the P300b in parietal and inferior temporal regions (Bledowski et al., 2004).

To date, high-density EEG has not been used to study neural correlates of CF in adolescents with AN.

The main objective of the present study was to evaluate neural activity assessed by high-density EEG during a CF task in adolescents with AN compared with a group of HC. Furthermore, we aimed to assess whether trait and state anxiety influenced CF at the neural and behavioural levels.

We hypothesized that we would observe impaired brain temporal dynamics in AN adolescents compared with HC during task switching a reliable measure of CF. Because the N200 and P300 are markers of CF, we expected that adolescents with AN would have deficits in these components, mainly in prefrontal-visual networks. We expected higher scores of trait anxiety in AN compared with HC, and we hypothesized that brain network characteristics of CF in AN would be predicted by trait anxiety. We expected comparable performance of CF between AN and HC, and higher levels of trait anxiety would be associated with behavioural impairment.

2 | MATERIALS AND METHODS

2.1 | Participants

Twenty-two girls with AN and 23 HC participants were included in the study. All participants were females, and their age ranged from 12 to 20 years (we choose to refer to our population as adolescents according to the mean ages of the samples and based on indications from literature (Sawyer et al., 2018).

Patients with AN were recruited from the adolescent and adult eating disorder (ED) services at Geneva University Hospital (HUG). Individuals received diagnoses of restricting-type AN ($n = 19$) and binge/purge-type AN ($n = 4$) by the respective multidisciplinary teams during clinical evaluations.

Controls were recruited through advertisements posted in local community centres, universities and schools. General exclusion criteria were a history of any neurological disorder or brain damage, presence of any psychopathology and ED symptoms.

All controls were screened: for psychopathology using the Strengths and Difficulties Questionnaire (SDQ) [completed by parents and youths for participants aged 12–17 years old and youths only for participants aged >17 years old] (Goodman, 1997); for ED symptomatology using the Eating Disorder Examination Questionnaire (EDE-q) (Fairburn & Beglin, 1994) [a 28-item self-report questionnaire assessing ED features; four sub-scales (Restraint, Eating Concern, Shape Concern and Weight Concern) and a global score are obtained], and using the ED section of The Development and Well-Being Assessment (DAWBA) (Goodman et al., 2000) completed by parents for those aged <15 years old ($n = 6$).

Participants also completed the State–Trait Anxiety Inventory (STAI) according to their age: adult version (Spielberger, 1983) (ages 16 through 20) or child versions (Turgeon & Chartrand, 2003) (ages 12 through 16). For subsequent analysis, scores were z-transformed using normative scores for adults and children. Body mass index (BMI), socio-demographic information about participants' ethnicity and family education were also collected.

During the first visit, cognitive functioning was assessed for all participants using four sub-scales of the Wechsler Intelligence Scale for Children Fifth Edition [WISC-V, ages 12 through 16 (Wechsler, 2014)] or the Wechsler Adult Intelligence Scale Fourth Edition [WAIS-IV, ages 16 through 20 (Wechsler, 2008)]: similarities, vocabulary, matrix reasoning and block design. These sub-scales were administered to ensure no confounding due to cognitive function.

Participants who were aged 16 and older, and parents of those below the age of 18, provided written informed consent. The research was conducted according to the principles of the Declaration of Helsinki and approved by the University of Geneva's research ethics committee. Participants received vouchers from a general store as monetary compensation for their participation in the study.

2.2 | Experimental paradigm

In order to assess CF, we used the Dimensional Change Card Sorting task 'DCCS' (Zelazo, 2006). Participants are required to sort bivalent test cards using rules (see Figure 1). In this study, we applied a version of the

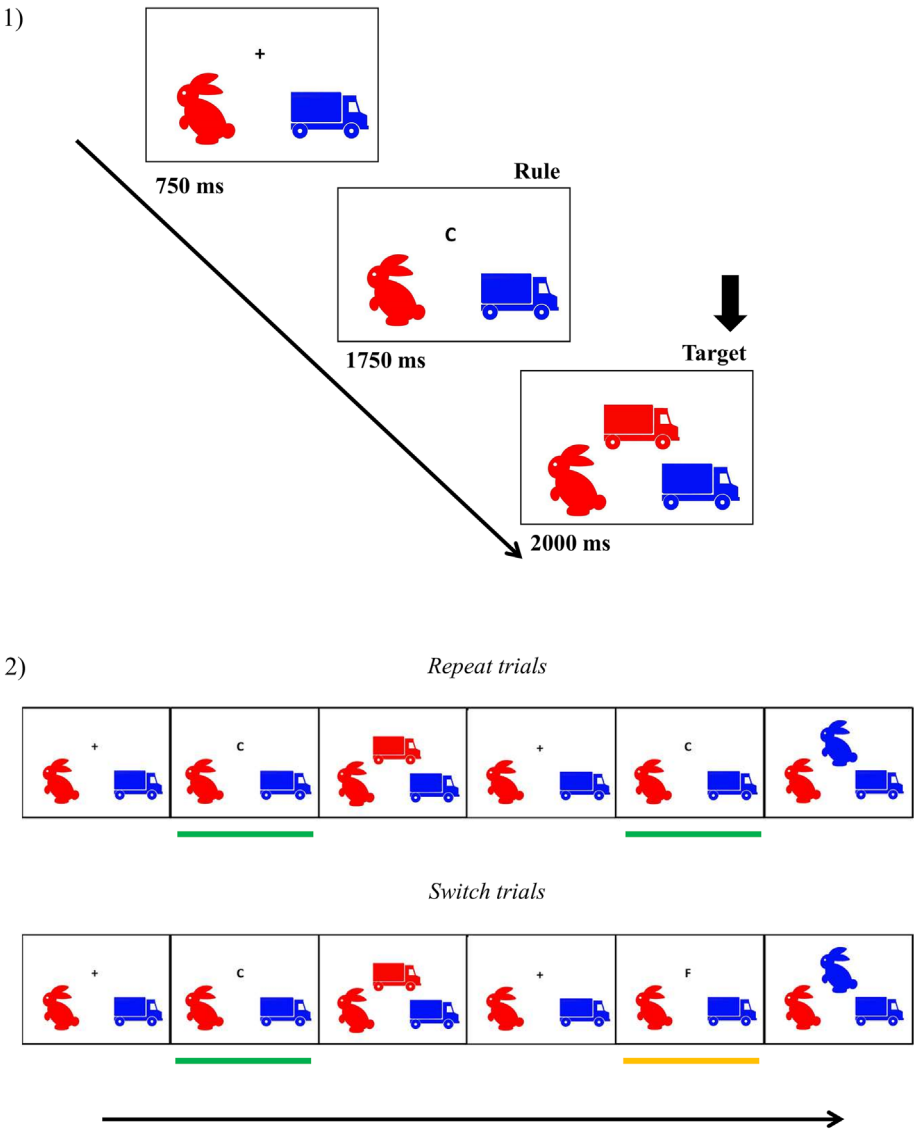

FIGURE 1 Schematic representation of the DCCS task. Each 'rule' (colour or shape) is presented for 1750 ms, followed by a stimulus target ('red truck' or 'blue rabbit') for 2000 ms. Participants were asked to answer according to the rule of the trial.

DCCS that has been proven suitable for older children and neuroimaging data collection (see Morton et al., 2009). Two bivalent images were always presented at the bottom of the screen: a red rabbit and a blue truck. On each trial, a fixation cross (750 ms) was followed by an instruction period (1750 ms) and a response period (2000 ms). After the fixation period, participants were instructed on the trial rule ('s' for shape or 'c' for colour), which was followed by the target stimulus: a blue rabbit or a red truck. The target stimulus matched each image presented at the bottom on a single dimension (either colour or shape). Participants responded by pressing one of two arrows on a keyboard using the right hand's index and middle finger. By pressing the right arrow, participants sorted the stimulus to the location of the right target (i.e., the blue truck); by pressing the left arrow, they sorted the stimulus to the location of the left target (i.e., the red rabbit). A 750-ms inter-trial interval separated trials.

Trials were administered in two blocks of 160 trials. Switch trials ($n = 80$) consisted of switch sorting rule trials (e.g., sort by shape 's' then by colour 'c') and repeat trials ($n = 80$) of sorting rule repeated (e.g., sort by colour 'c' then again by colour 'c'). Trials were administered in a semi-random order in each block.

The experiment was carried out in the afternoon (after 1 PM). The DCCS was preceded by a training block (10 trials) to ensure participants understood the task. The total task duration was approximately 20 min.

2.3 | EEG data acquisition and pre-processing

EEG data were acquired at 1000 Hz using a 256-channel system (EGI, Philips Electrical Geodesics, Inc.). Electrode impedance was kept below 30 kΩ, and data were acquired with the vertex (Cz) as a reference electrode.

EEG data time-locked to target stimuli onset (response period) were averaged separately for repeated and switching trials. EEG data were averaged only for correct trials. Data were band-pass filtered between .4 and 40 Hz, and a 50-Hz notch filter was applied. The original montage was reduced from 256 to 204 channels to exclude bad channels located at the periphery (see Berchio, Piguet, Gentsch, et al., 2017; Berchio, Piguet, Michel, et al., 2017). Infomax-based independent component analysis (ICA) was applied to remove eye blink, eye movements and electrocardiogram artifacts based on map topographies and the time course of the ICA component. Epochs for ERP analysis began 100 ms before the stimulus and ended 600 ms after its onset. A baseline correction was applied before the Target stimuli onset (−100 to 0 ms). Epochs contaminated by artifacts (muscle, eye blink/movements) were excluded by visual inspection. Epochs were re-referenced to the average and down-sampled to 250 Hz. Data pre-processing was performed using the CARTOOL Software (Brunet et al., 2011).

There were no group differences in the number of trials accepted (AN: repeated trials, $M = 74.36$, $SD = 5.1$; switching trials, $M = 73.90$, $SD = 4.19$; HC: repeated trials, $M = 71.34$, $SD = 7.82$; switching trials, $M = 72.65$, $SD = 6.04$; $p > .14$, two-tailed t test).

2.4 | Behavioural analyses

Responses were classified as correct (accuracy) or incorrect (errors) for repeated and switch trials. Missed responses (no response) were not considered in the analysis. Mean RT were measured for correct responses. We excluded outliers (trials with RT > 3 standard deviations from individual means). To reduce the heterogeneity of variance, errors were arcsine transformed (Ahrens et al., 1990). Switching costs between tasks were calculated by subtracting scores (i.e., RT and errors) on repeat trials from the corresponding values on switch trials (Rogers & Monsell, 1995).

To quantify in a single measure both aspects of performance (i.e., speed and errors), we calculated an inverse efficiency score 'IES' (Townsend & Ashby, 1983). The IES was created by dividing the RT of correct responses by the accuracy rates (Bruyer & Brysbaert, 2011). High values of IES indicate a lower efficiency (i.e., slow performance and reduced accuracy). IES switch costs were calculated as the difference between inverse efficiency sub-scores (i.e., switch trials IES − repeat trials IES) (see Hughes et al., 2014).

Behavioural data were analysed using a repeated measures ANOVA with Trials as within-subject factor ('Repeat' vs. 'Switch') and Group as a between-subject factor ('AN' vs. 'HC'). If the data were not normally distributed, non-parametric equivalent tests were applied (i.e., the Friedman test).

To investigate Group differences in switch costs (i.e., RT, error rates and IES), we performed one-way ANOVAs, or equivalent non-parametric tests (i.e., Mann–Whitney U-test) whenever normality assumptions were not satisfied.

To clarify the main effects and interactions, post hoc analysis used paired t-tests with $p < .05$ as the significance threshold after a Bonferroni correction for multiple comparisons.

Furthermore, in order to assess the effect of trait or state anxiety we included these as covariates in analyses where a significant effect of Group (as a single predictor or in interaction with another variable) was identified. For this purpose, we used repeated measures ANCOVAs, ANCOVAs and non-parametric equivalent tests (Quade's ANCOVA).

2.5 | ERP analysis

2.5.1 | Surface ERP analysis

ERP differences between conditions and groups were evaluated using two global tests across all electrodes (see Murray et al., 2008). To assess differences in the global strength of the electric field, we calculated the global field power (GFP), an index of the total amount of neuronal synchronization, which is equivalent to a spatial standard deviation (Skrandies, 1990). To assess differences in map topographies, we ran a 'topographic ANOVA' or TANOVA (see Brunet et al., 2011; Koenig et al., 2011). This analysis allows us to assume that EEG maps that are different between conditions/groups are generated by different underlying brain sources (Murray et al., 2008). GFP and TANOVA analyses were performed using the MATLAB-based open source toolbox Randomization Graphical User interface (Ragu, Koenig et al., 2011).

In the GFP and TANOVA models, we entered the factor Trial Type with two levels ('Repeat', 'Switch') and Group ('HC', 'AN') as the between-subjects factor. GFP and TANOVA were performed for each time point: from −100-ms pre-target to 600-ms post-target stimulus. To reduce the risk of false positive effects due to multiple tests, we opted for randomization statistics (Maris & Oostenveld, 2007), and we applied a time constraint of ≥20 ms of successive significant effects. The number of randomization runs was set to 5000, with a threshold of $p < .05$.

For each significant effect, post hoc tests were computed on the average signal across the statistically significant time interval to verify whether the effects were consistent across time points (Koenig et al., 2011).

Additional analyses were performed on the GFP to investigate the effect of trait and state anxiety on our findings. The GFP was selected as an exemplative global ERP measure. Group comparisons were performed using non-parametric (Quade's) ANCOVAs with trait and state anxiety as a separate covariate.

2.5.2 | Brain imaging analyses

Network differences between groups were tested using a linear distributed inverse solution model, which considers each voxel as a possible source of activity (LORETA, Pascual-Marqui et al., 2002). The inverse solution was estimated on average ERP using a Locally Spherical Model with Anatomical Constraints (Brunet et al., 2011) and on 5018 voxels located on the grey matter of a brain template of the Montreal Neurological Institute (http://www.bic.mni.mcgill.ca/brainweb). Following the computation of the inverse solution, data were transformed by a modified *z*-score normalization implemented in Cartool (3.80) (for technical details, see Michel & Brunet, 2019).

To minimize the problem of multiple comparisons, brain network analyses were performed using a randomization test implemented in Cartool on 116 regions of interest (ROI, Automated Anatomical Atlas; Tzourio-Mazoyer et al., 2002), with a *p*-value of <.05, and an average time window of interest. Student unpaired two-tailed *t*-tests were used for post hoc analyses. Time windows for brain source analyses were determined based on statistical evidence of ERP surface analysis.

2.5.3 | Associations between behaviour, clinical scores and ERP

In order to investigate relationships between performance on the DCCS task and anxiety, Spearman correlations were conducted between behavioural data and trait and state anxiety measures. Correlations were performed within each group. Additionally, in the AN group, Spearman correlations were performed to assess the relationship between behavioural measures and ED severity (i.e., EDE-q sub-scale scores and BMI).

For all correlations, bootstrapped confidence intervals (95%) were calculated to assess the significance of the effects estimated. The resulting *p*-values were corrected using a false discovery rate of $p < .05$ (Benjamini & Yekutieli, 2001).

To explore the potential effects of trait and state anxiety on ERP signatures in AN and HC, non-parametric series regression analyses were conducted.

3 | RESULTS

3.1 | Demographic and clinical variables

No differences in age [$F(1, 43) = .290$, $p = .59$], gender [$\chi^2(1) = .000, = 1$], handedness [$\chi^2(1) = 2.001$, $p = .157$] or family education [$\chi^2_s(1) < .06, = 1$] were observed between groups (see Table 1).

Two adolescents with AN had a clinical comorbid diagnosis of anxiety disorder, one of depression: One of these was taking psychotropic medication (selective serotonin reuptake inhibitors [SSRI]). Eleven adolescents with AN (50%) had amenorrhea at the time of study inclusion. For the AN sample, the mean global score on the EDE-q was 2.92 (SD:1.68) [Means of each sub-scale were Restraint = 3.06 (SD:1.95), Eating concerns = 2.31 (SD:1.44), Shape concerns = 3.36 (SD:1.79) and Weight concerns = 2.98 (SD:1.90)]. The median duration of illness in AN patients was 6.5 months (min = 3, max = 32).

AN participants showed significantly lower BMI compared with HC, as expected [$F(1, 43) = 32$, $p < .001$]. Differences between groups for STAI-state [$F(1, 43) = 29.33$, $p < .001$] and STAI-trait [$F(1, 43) = 15.11$, $p < .001$] were also observed, showing higher levels of state and trait anxiety in the AN group. No differences were observed between groups on cognitive function [all $F_s(1, 45) < 1.90$, all $p_s > 1.75$].

Demographic and diagnostic information are summarized in Table 1.

3.2 | Behaviour

Behavioural measures for both groups and experimental conditions are summarized in Table 2.

Accuracy and RT on switch and repeat trials (see Table 2) were compared using repeated measures ANOVAs. Repeated measures ANOVAs revealed a significant main effect of Trial Type on accuracy [$F(1, 43) = 30.9$, $p < .001$, $\eta_p^2 = .42$] and RT [$F(1, 43) = 21.67$, $p < .001$, $\eta_p^2 = .33$] with responses on switch trials significantly less accurate and slower than responses on repeat trials (post hoc tests, all $p_s < .001$). Importantly, there was a main effect of Group on accuracy, indicating greater accuracy independently of Trial Type in

TABLE 1 Demographic and clinical/cognitive measures.

	HC		AN		
	Mean	**SD**	**Mean**	**SD**	***p***
Number of participants	23		22		
Mean age (SD)	15.43	2.42	15.81	2.41	n.s.
Body mass index	20.74	2.81	16.86	1.53	<.001
Ethnicity (Caucasian)	81.1%		91.3%		
Parental education (one caregiver)					
Secondary education	35%		30%		n.s.
University studies	67%		70%		n.s.
STAI*					
State	−0.691	0.53	0.571	0.98	<.001
Trait	−0.477	0.56	0.488	1.04	<.001
Cognitive function (WISC/WAIS)					
Block design	14.65	11.53	11.41	2.75	n.s.
Similarities	15.78	8.06	14.68	2.62	n.s.
Matrix reasoning	12.17	5.04	11.31	2.58	n.s.
Vocabulary	15.65	9.27	13.13	2.66	n.s.
*z-scores					

TABLE 2 Summary of the DCCS behavioural performance for conditions and groups.

	HC		AN		Statistically significant group effects		
	Mean	**SD**	**Mean**	**SD**	***p*[b]**	***p*[c]**	***p*[d]**
Accuracy rates (%)							
Switch trials	*90.56*	6.37	*94.36*	4.01	$p = .04$ (Main Group effect)	n.s.	n.s.
Repeat trials	*95.74*	2.89	*97.14*[b]	1.75			
Switch cost (repeat-switch)	5.17	5.94	2.77	3.19	n.s.		
Mean RT (ms)							
Switch trials	763.06	166.71	757.71	177.92	n.s.		
Repeat trials	684.45	137.97	703.51	130.81	n.s.		
Switch cost (switch-repeat)	78.6	84.91	54.2	105.76	n.s.		
Errors[a]							
Switch trials	*0.84*	0.28	*0.68*	0.36	$p = .009$	n.s.	n.s.
Repeat trials	0.58	0.46	0.69	0.42	n.s.		
Switch cost (switch-repeat)	0.26	0.42	−0.01	0.59	n.s.		
Inverse efficiency (RT/accuracy)							
Switch trials	*853.02*	217.21	*783.4*[b]	161.99	$p = .01$ (Interaction Trial * Group)	n.s.	n.s.
Repeat trials	*717.47*	152.77	*723.87*	134.13			
Switch cost (switch-repeat)	*135.55*	122.6	*59.53*	64.2	$p = .01$	n.s.	n.s.

Abbreviations: DCCS, Dimensional Change Card Sort; RT, reaction times.
[a]Arcsine transformed proportion.
[b]Primary analyses.
[c]*P*-values adjusted for state anxiety.
[d]*P*-values adjusted for trait anxiety.

adolescents with AN compared with HC [$F(1, 43) = 4.43$, $p = .041$, $\eta_p^2 = .09$].

A one-way ANOVA indicated that the switch cost of RT was not significantly different between groups ($p > .3$).

Error data (without missed responses) did not meet parametric assumptions and were analysed using Friedman tests. No differences were found between errors of switch trials and errors of repeat trials ($p > .13$). We found that HC was more error prone in responses on switch trials than adolescents with AN [Mann–Whitney $U = 138$, $Z = -2.62$, $p = .002$, $\eta^2 = .153$], whereas no significant differences could be detected between the two groups on errors of repeat trials ($p > .14$).

A repeated measures ANOVA was conducted on the IES. The main effect of Group was not significant, in that the AN group was equally efficient as the HC group ($p > .5$). As expected, there was a main effect of Trial Type in that both AN and HC participants were less efficient on switch trials than they were on repeat trials [$F(1, 43) = 44.01$, $p > .001$, $\eta_p^2 = .50$]. Notably, there was a significant interaction between Group and Trial Type [$F(1, 43) = 6.69$, $p = .013$, $\eta_p^2 = .134$]. Although there were no statistically significant differences between groups in any of the IES scores, this interaction reflected the fact that the difference between the IES in the repeat trials and the IES in the switch trials was greater in the HC group (switch trials vs. repeat trials, $p < .001$) than it was in the AN group (switch trials vs. repeat trials, $p = .04$).

A one-way ANOVA indicated that switch cost of IES was significantly higher in HC than in AN [$F(1, 43) = 6.69$, $p = .013$, $\eta_p^2 = .134$].

3.3 | Effect of anxiety

A repeated measures ANOVA with trait anxiety as a covariate confirmed a significant main effect of Trial Type on accuracy [$F(1, 42) = 34.49$, $p < .001$, $\eta_p^2 = .451$], but the effect of Group on accuracy became non-significant [$F(1, 42) = 1.45$, $p > .2$, $\eta_p^2 = .33$]. No other significant effects or interactions were observed (all $p_s > .05$). A repeated measure ANOVA with state anxiety as a covariate confirmed the significant main effect of Trial Type [$F(1, 42) = 33.05$, $p < .001$, $\eta_p^2 = .44$], but the effect of Group became non-statistically significant [$F(1, 42) = 1.70$, $p > .19$, $\eta_p^2 = .39$]. No other significant main effects or interactions were observed (all $p_s > .05$).

Errors of switch trials were analysed using Quade's ANCOVA. These analyses indicated that the effect of Group was no longer significant after adjusting for trait anxiety [Quade's ANCOVA, F_{quade} (1, 44) = 1.85, $p = .18$, $\eta_p^2 = .41$], but also after adjusting for state anxiety [Quade's ANCOVA, F_{quade} (1, 44) = 1.82, $p = .18$, $\eta_p^2 = .41$].

For the IES, repeated measures ANOVAs were conducted with trait and state anxiety as separate covariates. After adjusting for trait anxiety, the main effect of Trial Type was confirmed [$F(1, 42) = 44.18$, $p > .001$, $\eta_p^2 = .51$], and the interaction effect between Group and Trial Type became marginal [$F(1, 42) = 2.94$, $p = .093$, $\eta_p^2 = .066$]. No other significant main effects or interactions were observed (all $p_s > .32$). After adjusting for trait anxiety, the main effect of Trial Type was confirmed [$F(1, 42) = 42.69$, $p > .001$, $\eta_p^2 = .50$], and the interaction effect between Group and Trial Type only trended toward significance [$F(1, 42) = 3.55$, $p = .066$, $\eta_p^2 = .078$]. No other significant main effects or interactions were observed (all $p_s > .05$).

ANCOVA analyses were performed on the switch cost of IES to adjust for trait and state anxiety separately. The effect of Group on switch cost of IES becomes non-significant after controlling for trait anxiety [$F(1, 44) = 2.94$, $p = .093$, $\eta_p^2 = .066$], and it only trended toward significance after adjusting for state anxiety [$F(1, 44) = 3.55$, $p = .066$, $\eta_p^2 = .078$].

3.4 | ERP

Visual inspection of grand averages revealed the following ERP components to target stimuli (see Figure 2): the P100, N200, P300a and P300b.

The GFP analysis showed a significant main effect of Trial Type on the N200 (192–204 ms; $p < .05$), of Group on the P100 (64–84 ms; $p < .05$) and a significant interaction for Trial Type * Group on the P300b (468–500 ms; $p < .05$) (see Figure 3a). Post hoc tests indicated that the N200 showed an augmented response for switch trials when compared with repeat trials ($p = .035$) and that the P100 showed an augmented response for the AN group when compared with the HC group ($p = .006$). Post hoc analysis of the interaction Trial Type * Group indicated an augmented response of the P300b for switch trials compared with repeat trials in the AN group ($p = .006$); no other significant effects were identified ($p_s > .05$).

The TANOVA indicated the main effects of Trial Type on the P100 (60–80 ms; $p < .05$) and of Group on the P100 (64–88 ms; $p < .05$) (see Figure 3b). Post hoc tests confirmed differences between conditions (60–80 ms; $p = .004$) and groups (64–88 ms; $p = .004$).

Effects of anxiety on the GFP were investigated on the Group effect at the P100 (64–84 ms) and on the

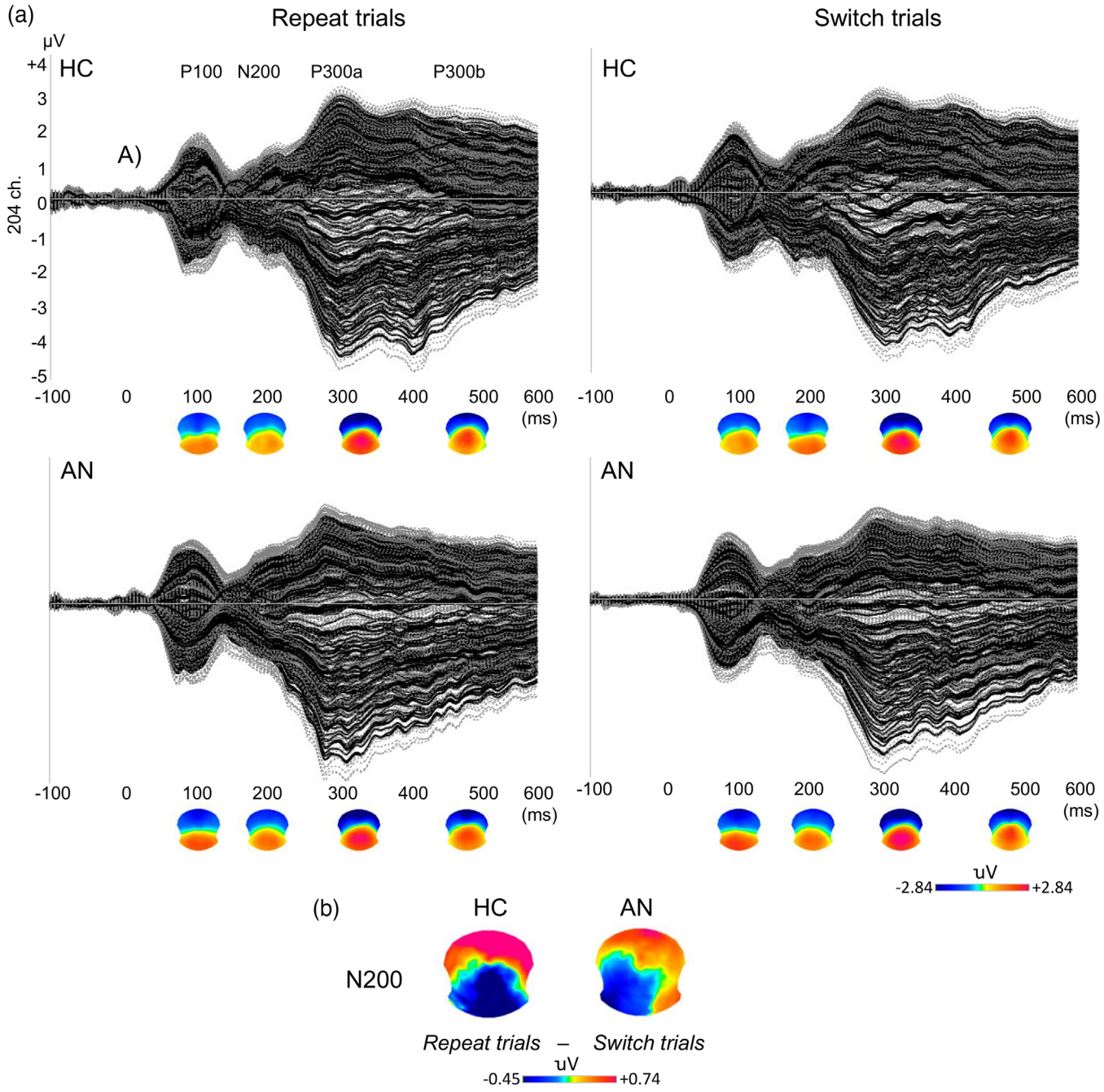

FIGURE 2 (a) Grand averages (butterfly plot) of trials repeated and trials switching: healthy controls (HC) and adolescents with anorexia nervosa (AN). Evoked responses are time-locked to the target stimuli. Dashed grey lines represent the standard deviation of single-channel EEG waveforms. Map topographies are plotted on global field power peaks of each component (P100, N200, P300a and P300b). (b) Difference maps of the N200 are obtained by subtracting repeat trials to switch trials; on the left for HC, on the right for AN adolescent.

significant interaction Trial Type * Group at the P300b (468–500 ms).

Quade's ANCOVAs indicated that the Group effect on the P100 remained significant after adjusting for trait anxiety [F_{quade} (1, 43) = 36.44, $p < .001$, $\eta_p^2 = .459$] and after controlling for state anxiety [F_{quade} (1, 43) = 36.47, $p < .001$, $\eta_p^2 = .459$].

The significant interaction Trial Type * Group showed an augmented response of the P300b for switch trials compared with repeat trials only within the AN group. Therefore, a switch cost of the P300b (switch trials GFP − repeat trials GFP) was calculated within each group. Group comparisons on the switch cost of the P300b were carried out with Quade's ANCOVA, with

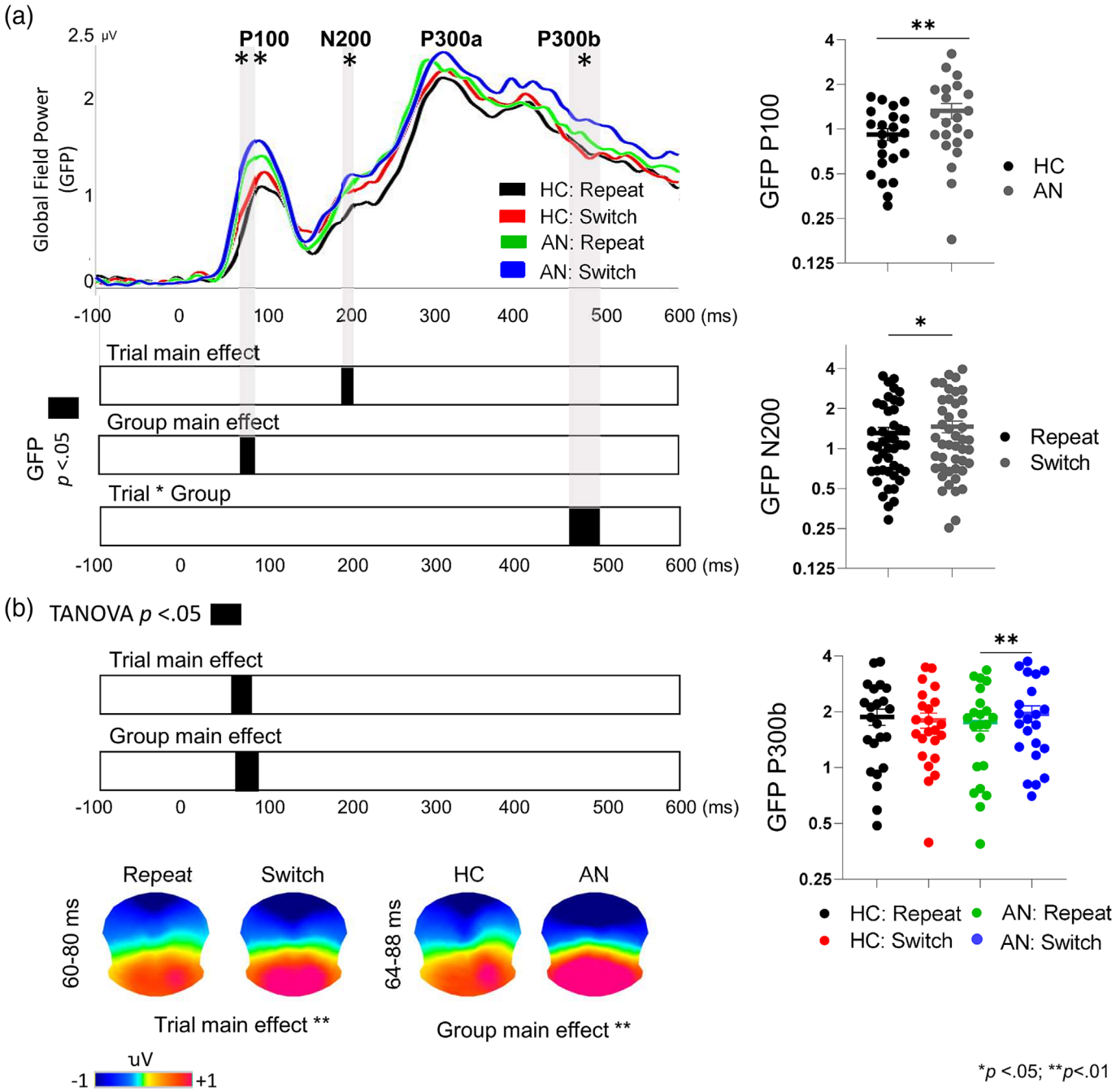

FIGURE 3 Global field power (GFP) and TANOVA results. (a) For each group and experimental conditions, GFP traces are plotted over time. Main effects and interactions effects of the GFP (a) and TANOVA (b) analyses are shown [Trial Type ('Switch', 'Repeat') * Group ('HC', 'AN')]. On the left, results of post hoc analyses on significant GFP effects are shown. On the lower part, topographic maps of the main effect of Group and of the main effect of Trial are plotted. Horizontal grey bars highlight the time interval where significant effects were identified. Statistically significant effects are marked by black vertical square and asterisks (*$p < .05$; **$p < .005$).

trait and state anxiety as separate covariates. These secondary analyses indicated a significant group effect on the switch cost of the P300b after controlling for trait anxiety [F_{quade} (1, 43) = 4.93, $p = .032$, $\eta_p^2 = .103$] and state anxiety [F_{quade} (1, 43) = 4.93, $p = .032$, $\eta_p^2 = .103$], with the AN group showing higher values than HC.

3.5 | Brain imaging results

ERP surface analysis highlighted differences between groups at the P100 (Group main effect) and the P300b (Interaction Trial * Group). Thus, between-group analyses were performed focusing on these components

and on the time windows of the corresponding statistically significant effects.

For the P100 (64–88 ms), the randomization test showed differences between AN and HC in the left post-central gyrus and in the bilateral parietal lobe (all $p_s < .05$) (see Figure 4). Reduced activations were found for adolescents with AN compared with HC in the following brain regions: left post-central gyrus ($t = -2.16$, $p = .03$, $d_{cohen} = .64$); left parietal lobe ($t = -2.90$, $p = .006$, $d_{cohen} = .86$); right parietal lobe ($t = -2.86$, $p = .008$, $d_{cohen} = .85$).

For the P300b (468–500 ms), AN and HC were compared on repeat and switch trials (see Figure 4). For switch trials, the randomization test showed diminished activation in AN patients compared with HC in the bilateral posterior cingulate, right calcarine sulcus and right cerebellum (all $p_s < .05$): left posterior cingulate ($t = -2.21$, $p = .03$, $d_{cohen} = .66$); right posterior cingulate ($t = -2.56$, $p = .01$, $d_{cohen} = .76$); right calcarine sulcus ($t = -2.02$, $p = .04$, $d_{cohen} = .60$); right cerebellum ($t = -2.75$, $p = .008$, $d_{cohen} = .82$).

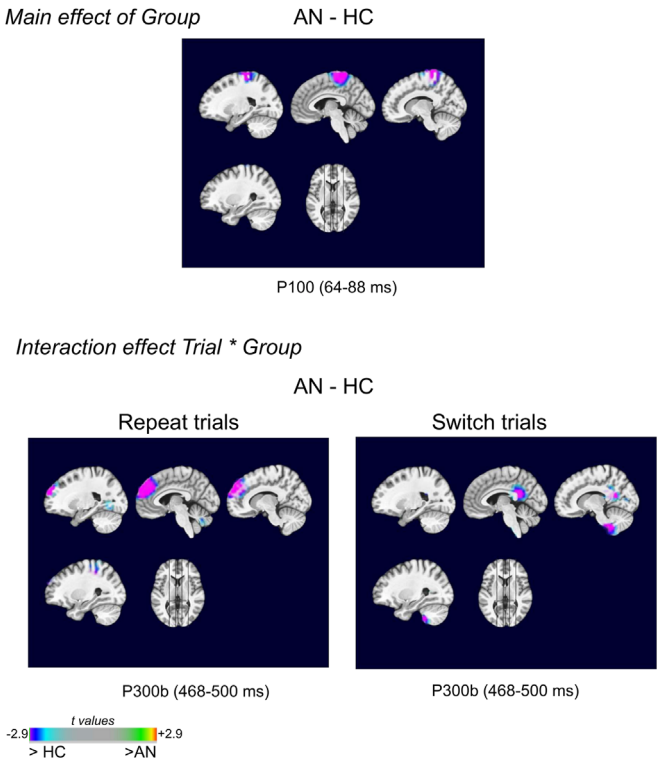

FIGURE 4 Brain imaging results. Randomization tests were performed to compare brain activity between groups. Statistically significant effects are shown for the P100 (Group main effect) for the P300b of switch and repeat trials. *T* values indicate the directions of the contrast: purple/blue > activity in healthy controls (HC), green/red > activity in adolescents with anorexia nervosa (AN). The significance level was set to $p < .05$ (p-values are corrected on regions of interest and on average temporal windows).

For repeat trials, the randomization test showed diminished activation in AN patients compared with HC in the left and right frontal medial lobe, left lingual gyrus, right post-central gyrus, right inferior parietal lobe, right supramarginal gyrus and in the vermis ($p_s < .05$): left frontal medial lobe ($t = -2.75$, $p = .008$, $d_{cohen} = .82$); right frontal medial lobe ($t = -2.24$, $p = .03$, $d_{cohen} = .67$); left lingual gyrus ($t = -2.23$, $p = .03$, $d_{cohen} = .66$); right post-central gyrus ($t = -2.23$, $p = .04$, $d_{cohen} = .66$); right inferior parietal lobe ($t = -2.37$, $p = .02$, $d_{cohen} = .70$); right supramarginal gyrus ($t = -1.98$, $p = .049$, $d_{cohen} = .59$); vermis ($t = -2.47$, $p = .018$, $d_{cohen} = .77$).

3.6 | Correlation analysis

Correlation analyses between trait anxiety, state anxiety and behavioural measures (i.e., accuracy, errors of switch trials and IES switch cost) were conducted within each group. No statistically significant correlations were found in the AN sample ($p_s > .05$), in that the participants' trait and state anxiety seemingly did not affect their accuracy, errors and so on, during the task. However, in the HC group, this analysis highlighted a significant positive correlation between the accuracy of switch trials and trait anxiety ($r_{pb} = .479$, $p = .021$, [.062, .769]). No statistically significant correlations were found with trait anxiety and accuracy of repeated trials, errors of switch trials and IES switch cost in HC ($p_s > .028$). Furthermore, in the HC group, these analyses highlighted a significant negative correlation between errors of switch trial and state anxiety ($r_{pb} = -.481$, $p = .020$, [−.773, −.029]). No statistically significant correlations between state anxiety and accuracy or IES switch cost were identified in HC ($p_s > .039$).

Within the AN sample, no significant correlations were found between ED severity (BMI and EDE-q sub-scale scores) and the following behavioural measures: accuracy, errors of switch trials and IES switch cost ($p_s > .05$). BMI was not correlated with behavioural performance (accuracy, errors of switch trials and IES switch cost) in HC ($p_s > .05$).

Non-parametric series regression analyses were conducted with the average GFP of the P100 (60–80 ms) and of the P300b of switch trials and repeat trials (468–500 ms) as dependent variables and trait and state anxiety as covariates in separate models. Statistical models were implemented independently for each ERP variable to avoid multicollinearity problems. In the AN group, no significant effects were identified on the P100 and P300b (switch trials and repeat trials) and trait anxiety (all $p_s > .05$). Similarly, in the AN group,

no significant effects were identified on the P100 (switch trials and repeat trials) and on the P300b and state anxiety (all $p_s > .05$).

In the HC group, we found a significant effect of trait anxiety on the P100 [$Z = +2.51$; $p = .012$; 95% Conf. Interval (.14, 1.16)]. No other significant effects were found for trait anxiety on the P300b (switch trials and repeat trials) (all $p_s > .05$). No significant effects were found on the P100 and state anxiety (all $p_s > .05$) and on the P300b (switch trials and repeat trials) and state anxiety in HC (all $p_s > .05$).

3.7 | Post hoc tests

In order to assess whether AN comorbidities could have driven ERP effects, further post hoc tests were performed by removing the three adolescents with AN who had a diagnosis of anxiety disorder and depression, that is, between 19 AN patients and 23 HC.

The results of this analysis confirmed group differences in GFP of the P100 (60–80 ms; $p = .018$), with higher amplitude in adolescents with AN than HC, and on TANOVA of the P100 (64–88 ms; $p = .038$). Group effects were also confirmed for GFP of the P300b (Interaction Trial * Group; 468–500 ms; $p = .03$), with the AN group showing higher values in switch trials than repeat trials ($p < .001$).

Furthermore, in order to assess whether an imbalance between clinical subgroups might have biased ERP effects, post hoc tests were performed by removing the four adolescents with AN who had a diagnosis of binge/purge type, that is, between 18 AN restricting-type patients and 23 HC. The results of this analysis confirmed group differences in GFP of the P100 (60–80 ms; $p = .02$), with higher amplitude in adolescents with AN restricting-type than HC, and on TANOVA of the P100 (64–88 ms; $p = .02$). Group effects were also confirmed for GFP of the P300b (Interaction Trial * Group; 468–500 ms; $p = .029$), with the AN restricting-type group showing higher values in switch trials than repeat trials ($p < .001$).

4 | DISCUSSION

To our knowledge, this is the first high-density EEG study to investigate CF in adolescents with AN. The findings of this study suggest that neural abnormalities of task switching are evident in adolescents with AN. This study revealed different behavioural profiles on the DCCS for AN and HC. Specifically, adolescents with AN showed a greater overall accuracy, made fewer errors on switch trials and showed a reduced inverse efficiency switch cost relative to HC. However, this did not hold true after adjusting for anxiety. This suggests that anxiety (both trait and state) plays an important role in behavioural CF in AN. ERP results indicated that augmented early visual orienting processing (P100) and subsequent impaired attentional mechanisms to task switching (P300b) were evident in adolescents with AN compared with HC. These effects were independent of anxiety. Adolescents with AN showed reduced brain activation in task-relevant networks during task switching and task repetition. Findings of this study may support the view that atypical neural-monitoring processes of CF are evident in adolescent AN.

Regarding the behavioural performance, similarly to other studies using the DCCS (Ezekiel et al., 2013; Zelazo, 2006), we found responses on switching trials significantly less accurate, slower and more error prone than responses on repeated trials.

In line with previous evidence in adolescents with AN (Andrés-Perpiña et al., 2011; Bohon et al., 2020; Castro-Fornieles et al., 2019; Fitzpatrick et al., 2012), we found no evidence of impaired encoding of task-relevant information in the AN group. Therefore, our findings seem to suggest that task switching behavioural impairments may reflect chronicity of the disease.

Surprisingly, a slightly enhanced quality of performance (i.e., greater overall accuracy and lower errors in switch trials compared with HC) was observed in the AN group. However, these effects become non-significant after adjusting for trait and state anxiety. This is especially important as trait and state anxiety are associated with AN (Heruc et al., 2018; Raney et al., 2008; Schulze et al., 2009). Anxiety effects on cognitive performance are well recognized (Eysenck et al., 2007; Shi et al., 2019). Under specific circumstances, investing extra effort and resources may help anxious individuals to compensate for aversive effects of anxiety (Eysenck et al., 2007; Kofman et al., 2006; Shi et al., 2019; Ursache & Raver, 2014). Therefore, it is possible that high trait and state anxiety induced greater effort on task execution in AN. Taken together, these findings provide evidence of complex interactions between CF, coping strategies and anxiety in adolescents with AN.

An enhanced quality of performance was also described in a study using a similar task and showing better overall accuracy in adolescents with AN than HC (Bühren et al., 2012). These findings seem to indicate an involvement of additional strategies in adolescents with AN (e.g., enhanced effort and increased use of resources). Several explanations are plausible. One possibility is that perfectionism in adolescents with AN leads to stronger concern over mistakes (Bulik et al., 2003; Dahlenburg

et al., 2019). Furthermore, as suggested by Bühren and co-workers (Bühren et al., 2012), this behavioural profile may be due to a prefrontal cortex that is still developing in young patients. On the other hand, it is also possible that anxiety, as mentioned above, induces an enhanced effort in task execution (Eysenck et al., 2007). In the study of Bühern and colleagues (Bühren et al., 2012), the effects of anxiety on behavioural performance were excluded by performing post hoc correlations analysis between anxiety symptoms and RT, and errors. However, in this study, anxiety was assessed using a global score of symptoms severity (see Essau et al., 2002), and no information about differences between groups were reported. Therefore, it cannot be excluded that stable characteristics or temporary states of anxiety had a substantial impact on participants' performance.

In this last respect, we only found significant correlations between accuracy/errors of switch trials and trait and state anxiety, respectively, within the HC group. Surprisingly, lack of effects of trait and state anxiety were observed in the AN group. These findings suggest that an increased vigilance helped HC to better focus on task switching. Notably, HC exhibited lower trait and state anxiety than AN. In this respect, we may hypothesize that the behavioural strategy adopted by adolescents with AN (i.e., an enhanced cognitive effort) aims at reducing and controlling their high levels of anxiety. This would explain the lack of significant correlations within the AN group. Nevertheless, the current results should be interpreted in light of the fact that behavioural flexibility, anxiety and associated coping strategies may not be completely separate when investigating CF in adolescents with AN.

To further extend our findings, behavioural data were investigated using switch costs analysis. Switching between tasks requires reconfiguration of mental resources and produces a decrease in performances (Monsell, 2003). A greater asymmetric 'switch cost' is often elicited by easier tasks (Ellefson et al., 2006; Schneider & Anderson, 2010), likely as a reflection of the strength of top-down control applied (Yeung & Monsell, 2003). Our analysis highlighted a reduced inverse efficiency switch cost in AN compared with HC. This effect may indicate that adolescents with AN maintained readiness for task switching (even when this was not necessary). However, group effects become non-significant after adjusting for trait and state anxiety. Performance efficiency is negatively affected by anxiety (Eysenck et al., 2007; Shi et al., 2019). Remarkably, enhancement of stress has been found to be associated with reduced switch costs (Kofman et al., 2006). This suggests that differences between AN and HC on 'switch cost' were anxiety driven. Furthermore, these data can be interpreted as suggesting that trait and state anxiety facilitate performance efficiency differently in HC and AN.

Furthermore, correlation analyses within the AN group demonstrated that the behavioural patterns observed in the AN group were independent of active symptomatology and BMI. This finding seems to exclude an impact of the severity of the illness on behavioural performance in adolescents with AN.

It is also important to note that our sample was more representative of AN-restrictive type. Because adolescents with AN restricting-type are generally expected to show higher perfectionistic tendencies (Nilsson et al., 2008), this may suggest an association with high-standard performance. However, current evidence seems to exclude differences in levels of perfectionism between AN subtypes (Dahlenburg et al., 2019). Furthermore, neuropsychological research suggests shared underlying mechanisms of CF between restricting and binge purge subtypes of AN (Tchanturia et al., 2004; Vall & Wade, 2015; Van Autreve et al., 2013). Based on available evidence, it appears reasonable to assume that clinical dimensions related to AN subtypes had a limited impact on our behavioural findings.

ERP was investigated using global-spatial methodologies. Across participants, task switching induced changes in the topography of the P100 and an augmented global response at the latency of the N200.

Contrary to our expectations, P100 abnormalities in AN were identified in both experimental conditions. P100 responses are thought to reflect automatic recruitment of visual spatial attention (Di Russo & Spinelli, 1999) but also cognitive modulation of sensory processing (Kaiser et al., 2020). For the P100, source imaging analysis indicated differences between AN and HC in the left post-central gyrus and the bilateral parietal lobe. The posterior parietal cortex plays a key role in the sensorimotor integration of visually guided movements (Buneo & Andersen, 2006) and perceptual and categorical decisions (Zhou & Freedman, 2019). The observed decrease in activation could indicate that adolescents with AN were quicker to automatically orient visual attention to task requests than HC.

No group differences were identified for the N200. This finding provides evidence of an intact neural mechanism of conflict monitoring in adolescents with AN.

For the P300b, we found an augmented global response in the AN group to switch trials than repeat trials. The P300b is a marker of executive functioning (Downes et al., 2017), and augmented amplitudes of this component have been found to be associated with lower CF (Peltz et al., 2011). Our findings suggest a poorly

modulated attentional mechanism of task switching in adolescents with AN.

During task switching, source imaging analysis of the P300b showed reduced activations in AN in a network involving the bilateral posterior cingulate, the right calcarine sulcus and the right cerebellum. The posterior cingulate cortex is deactivated during cognitive tasks involving externally directed attention (Singh & Fawcett, 2008), and the calcarine sulcus is involved in visuo-spatial attention (Noesselt et al., 2002). The cerebellum, among other cognitive regulatory functions, is also involved in set-shifting and perseveration (Schmahmann, 2019). Previous findings have indicated reduced activations in AN in visual and cerebellar regions during visuo-spatial shifting tasks (Castro-Fornieles et al., 2019). The reduced activity of this network is suggestive of a hypo-functioning executive system during task switching in AN.

During repeated trials, source imaging analyses of the P300b indicated lower activations in adolescents with AN compared with HC in the bilateral medial prefrontal lobe, left lingual gyrus, right post-central gyrus, right inferior parietal lobe, right supramarginal gyrus and in the vermis. Parietal, visual and cerebellar regions are typically involved in task repetitions (Loose et al., 2017). Previous evidence has documented altered activity of the medial prefrontal cortex in AN (Bronleigh et al., 2022; Fuglset et al., 2016), which is a region that has a top-down regulatory role (Narayanan & Laubach, 2017), and is necessary to modulate prepotent responses (Uddin, 2021). The findings of this study seem to highlight a decreased prefrontal/posterior functionality most probably implicated in motor response suppression.

Previous studies have documented poorer self-reported CF in adolescents with AN than HC (Lao-Kaim et al., 2015; Miles et al., 2020). Although these findings appear to be inconsistent with many behavioural studies (Andrés-Perpiña et al., 2011; Bohon et al., 2020; Dmitrzak-Weglarz et al., 2011; Fitzpatrick et al., 2012), neurobiological evidence appears, instead, to support CF neural abnormalities in adolescent AN (Castro-Fornieles et al., 2019). One possible explanation is that the tendency of ED patients to be highly self-critical (Duarte et al., 2016) may lead to perceiving themselves as inflexible and rigid. On the other hand, it is also possible that real-life complex situations interfere more with CF functioning among young patients. Because neurobiological findings of our study seem to confirm the presence of abnormalities in neural mechanisms behind CF, this last hypothesis seems to be the most plausible.

Contrary to our hypothesis, dysfunctional P100 and P300b amplitudes were not significantly related to trait anxiety in the AN group. Correlation analyses indicated only a positive association between trait anxiety and the P100 in HC suggesting a reduced visual attentive arousal mechanism in AN. Furthermore, it is also important to note that between-group differences in ERP remained significant after adjusting for anxiety.

Taken together, our findings suggest that anxiety may not account for ERP abnormalities in AN, ruling out a possible link between anxiety symptoms and CF neural dysfunctions in young patients.

Analyses on the AN-restrictive subgroup versus HC confirmed statistically significant differences in the P100 and P300b, excluding that an imbalance between clinical subgroups might have biased the results.

This study has some limitations. The main limitation is the small sample size, which could have limited the statistical power of our analysis. The small sample size did not allow to investigate subtypes of AN, because of a lack of power to reliably assess differences between AN subtypes. A limitation of this study is also its cross-sectional nature; this does not allow to draw any conclusions regarding CF mechanisms in AN after recovery. Furthermore, one participant taking psychotropic medication was included; however, we did assess the possible influence of comorbidities. Further studies should include a larger sample size in order to replicate our findings.

5 | CONCLUSION

This study sheds novel insights into potential mechanisms mediating behavioural characteristics and neural vulnerabilities for mental inflexibility in adolescents with AN. Our work suggests different behavioural strategies and altered neural responses in adolescents with AN.

Our behavioural findings indicate an enhanced cognitive effort on task execution in AN. However, this pattern was mainly driven by differences between groups on trait and state anxiety. Thus, our findings suggest that a different behavioural profile in task switching in AN might be secondary to differences in coping behaviours associated with anxiety.

Neural abnormalities of the P300b might be characteristic of adolescent AN during task switching and perseveration. Neural abnormalities are localized in large-scale brain networks, including the posterior cingulate, the medial prefrontal cortex, several posterior regions and the cerebellum. Our findings also indicate that abnormal CF neural patterns were not related to trait and state anxiety in AN. The association between anxiety and CF is no doubt complex in adolescents with AN and will require further investigation. To gain a deeper understanding of CF mechanisms in AN, the

findings of our study emphasize the importance of complex models focusing upon behavioural and biological dimensions.

Insights generated by this study increase our neurobiological understanding of adolescent AN. Future research should investigate the efficacy of cognitive training in AN to rebalance the brain networks of CF.

AUTHOR CONTRIBUTIONS

Cristina Berchio: Conceptualization; data curation; formal analysis; funding acquisition; investigation; methodology; project administration. **Ynes Bouamoud:** Data curation. **Nadia Micali:** Conceptualization; data curation; formal analysis; funding acquisition; investigation; methodology; project administration.

ACKNOWLEDGEMENTS

This work was supported by the Foundation Gertrude von Meissner and the Fondation Ernst et Lucie Schmidheiny (to Cristina Berchio and Nadia Micali). Open access funding provided by Universite de Geneve.

CONFLICT OF INTEREST STATEMENT

All authors disclose any potential sources of conflict of interest.

DATA AVAILABILITY STATEMENT

Raw data are available upon reasonable requests to the corresponding author.

PEER REVIEW

The peer review history for this article is available at https://publons.com/publon/10.1111/ejn.15921.

ORCID

Cristina Berchio https://orcid.org/0000-0002-4573-5997

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How to cite this article: Berchio, C., Annen, L. C., Bouamoud, Y., & Micali, N. (2023). Temporal dynamics of cognitive flexibility in adolescents with anorexia nervosa: A high-density EEG study. *European Journal of Neuroscience*, *57*(6), 962–980. https://doi.org/10.1111/ejn.15921