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Anxiety Symptoms in Young Children Are Associated With a Maladaptive Neurobehavioral Profile of Error Responding

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ABSTRACT

BACKGROUND: Childhood anxiety symptoms have been linked to alterations in cognitive control and error processing, but the diverse findings on neural markers of anxiety in young children, which vary by severity and developmental stage, suggest the need for a wider perspective. Integrating new neural markers with established ones, such as the error-related negativity, the error positivity, and frontal theta, could clarify this association. Error-related alpha suppression (ERAS) is a recently proposed index of post-error attentional engagement that has not yet been explored in children with anxiety.

METHODS: To identify neurobehavioral profiles of anxiety in young children by integrating ERAS with the error-related negativity, error positivity, frontal theta, and post-error performance indicators, we employed K-means clustering as an unsupervised multimetric approach. For this, we first aimed to confirm the presence and scalp distribution of ERAS in young children. We performed event-related potentials and spectral analysis of electroencephalogram data collected during a Go/NoGo task (Zoo Task) completed by 181 children (ages 4–7 years; 103 female) who were sampled from across the clinical-to-nonclinical range of anxiety severity using the Child Behavior Checklist.

RESULTS: Results confirmed ERAS, showing lower post-error alpha power, maximal suppression at occipital sites, and less ERAS in younger children. K-means clustering revealed that high anxiety and younger age were associated with reduction in ERAS and frontal theta, less negative error-related negativity, enlarged error positivity, more post-error slowing, and reduced post-error accuracy.

CONCLUSIONS: Our findings indicate a link between ERAS, maladaptive neural mechanisms of attention elicited by errors, and anxiety in young children, suggesting that anxiety may arise from or interfere with attention and error processing.

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Anxiety is the most prevalent mental health issue during early development, affecting 19.3% of preschoolers and 31.9% of adolescents (1,2). Early-life anxiety significantly increases the risk of later mental health problems (3–6), underscoring the importance of developing quantifiable, mechanism-based markers for early detection, intervention, and prevention (7).

The etiology of anxiety, potentially linked to the abnormal development of error-processing mechanisms crucial for learning, remains unclear (8,9). These mechanisms, which strengthen during childhood—a critical time for learning and adaptation (10–13)—coincide with the initial emergence of psychopathologies, including anxiety disorders.

Neurophysiological responses to errors can be quantified via the error-related negativity (ERN), an established marker of performance monitoring and a Research Domain Criteria subconstruct of cognitive control. Studies have revealed that compared to healthy individuals, adolescents and adults with anxiety disorders show an enlarged ERN amplitude (9,14). The ERN, a negative electroencephalogram (EEG) amplitude deflection that occurs 100 ms post-error and emanates from the midline prefrontal cortex (e.g., anterior cingulate cortex) (15,16), has been associated with neural mechanisms of attention elicited by errors (16–18). Another relevant index of error processing is the error positivity (Pe), which is observed as a positive amplitude deflection in the centroparietal region that occurs between 200 and 500 ms following an error. The Pe component is thought to indicate awareness of an error (19,20), but its exact role and its relationship with anxiety disorders remain ambiguous (21–23). In the time-frequency domain, frontal theta activity (4–7 Hz), which emanates primarily from the medial frontal cortex, is increased immediately after errors (24) and has been associated with the monitoring and correction of errors (24,25) as well as the modulation of attention and implementation of cognitive control in challenging situations (26). Collectively, the ERN, Pe, and frontal theta are considered to represent elements of a broad

SEE COMMENTARY ON PAGE 547

response-monitoring system. This system is designed for identifying response errors, orienting to these and other novel events, and ultimately indicating the necessity for adjustments in behavior (27–30).

The ERN, Pe, and frontal theta have been associated with anxiety disorders (25,31–33). However, the functional significance of an enhanced brain response to errors in anxiety disorders remains unclear, with various theories having been proposed. For example, some have suggested that a larger ERN in participants with anxiety indicates heightened affective sensitivity to errors (31,32), while others view it as a compensatory mechanism for adaptive control to overcome symptoms (34) and/or the distracting effects of anxious thoughts on cognitive performance (9). These varied findings, especially across different levels of anxiety severity and developmental stages, highlight the importance of a broader view. Incorporating new neural markers of error monitoring, along with the ERN, Pe, and frontal theta, has the potential to provide a clearer understanding of the association between error processing and anxiety symptoms in young children.

Error-related alpha suppression (ERAS), characterized by reduced alpha power (8–14 Hz) 200 to 500 ms post-error at parieto-occipital sites (35,36), has emerged as a potential marker of neural mechanisms of attention elicited by errors. Existing research shows that ERAS is not correlated with the ERN or Pe (35), which is indicative of divergent or parallel cognitive systems. Specifically, ERAS may reflect a shift in attention from internal to external sources following error commission. This interpretation is consistent with studies that have shown increased alpha power during inward attention (37,38) and decreases for external focus (39,40), with alpha power also being linked to mind wandering (41).

Alpha activity is associated with the default mode network (DMN), which is active during internal processes but decreases for external tasks (42–45). Thus, ERAS may reflect DMN activity reduction, refocusing on the task and adjusting performance. Atypical DMN function is associated with anxiety because individuals often struggle to shift attention from internal worries even when aware that the worries are excessive (34,46). Thus, in patients with anxiety disorders, abnormal DMN engagement during cognitive tasks suggests that an inability to deactivate the DMN may contribute to worry, thereby affecting task performance. In healthy youths, age-related increases in DMN and cognitive control network interactions during task and rest (47–51) are believed to enhance cognitive task performance and emotional self-regulation. Early anxiety symptoms may disrupt this developmental trajectory, with children with anxiety less capable of shifting attention from internal worries to external tasks and goals (34).

The Current Study

The overarching goal of this study was to delineate neurobehavioral profiles of anxiety in young children by employing a multimetric approach for the integration of ERAS as a novel marker of neural mechanisms of attention elicited by errors, along with established ones (ERN, Pe, frontal theta) and post-error performance indicators. First, we aimed to confirm the presence and scalp distribution of ERAS, previously demonstrated in adults (35,36), in children performing an error-eliciting Go/NoGo task (Zoo Task) (10). We hypothesized that, similar to adults, alpha power would be significantly reduced following errors compared to correct responses, with the greatest suppression at occipital recording sites. Additionally, given the progressive efficiency of cognitive control and error processing with age (10–12), we anticipated that older children would exhibit more ERAS than their younger counterparts.

Furthermore, we explored the association between ERAS and anxiety symptoms as measured by the DSM-oriented anxiety problems subscale of the Child Behavior Checklist (CBCL), hypothesizing that reduced ERAS may reflect the anxiety-driven focus on internal stimuli despite external task requirements. In this way, higher levels of anxiety symptoms were expected to be correlated with more post-error alpha power, indicating less ERAS.

Lastly, we employed a data-driven approach using the unsupervised machine learning technique K-means clustering to identify distinct clusters involving ERAS, other well-described error-related neurophysiological markers of cognitive control (ERN, Pe, frontal theta), and post-error performance metrics (post-error reaction time, post-error accuracy) and determine how the clusters were associated with anxiety severity, age, and sex.

METHODS AND MATERIALS

Participants

A total of 181 children (ages 4–7 years, 56.9% female) were sampled from the community and the University of Michigan Child and Adolescent Psychiatry Clinic. EEG data from 56 (52), 30 (53), and 32 of these participants (54) have been included in previous reports on the ERN but have never been combined into a single larger sample or used for spectral analysis. Missing data (Table S1) were handled using the machine learning imputation method missForest (55).

Eligibility criteria included no history of head injury, serious medical conditions, neurodevelopmental delays, or central nervous system–affecting medications. Informed consent was obtained from parents, with assent from children. Participants had an average age of 5.6 years (SD = 1.1, range = 4.0–7.0), 11% were African American non-Latino, 20% were of other races or ethnicities, including Latino, and 69% were White non-Latino (Table 1).

All the experiments were carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans. The University of Michigan Medical School's Institutional Review Board approved these studies.

The Zoo Task

Children were led to a child-friendly EEG booth where they watched cartoons during setup. To maintain motivation and reduce fidgeting during EEG recordings, children received animal stickers and brief breaks between Zoo Task blocks. Participants performed the Go/NoGo Zoo Task (10), where they helped a zookeeper by pressing a button to capture loose animals (Go trials) but refrained from responding to orangutans (NoGo trials) (Figure 1). The task comprised 8 blocks, each with

Table 1. Demographic Characteristics and Zoo Task Performance of Study Participants, *N* = 181

Characteristic	Value
Sex, *n* (%)	
Female	103 (56.9%)
Male	78 (43.1%)
Race/Ethnicity, *n* (%)	
Asian or Pacific Islander	5 (2.8%)
Biracial	26 (14%)
Black or African American	20 (11%)
Hispanic or Latino	5 (2.8%)
Native American	0 (0%)
Other	1 (0.6%)
White Non-Hispanic or Latino	124 (69%)
Age, Months	
Mean (SD)	73 (15.5)
Range	45–116
Miss Error Rate	
Mean (SD)	0.04 (0.04)
Range	0.0–0.3
False Alarm Error Rate	
Mean (SD)	0.08 (0.04)
Range	0.0–0.2
Post-Error Reaction Time	
Mean (SD)	525.2 (61.2)
Range	340.9–684.0
Post-Error Accuracy	
Mean (SD)	0.9 (0.1)
Range	0.7–1.0
CBCL-Anxiety	
Mean (SD)	56.6 (9.1)
Range	50.0–88.0

CBCL, Child Behavior Checklist.

CBCL-Anxiety indicates anxiety problems T score from the CBCL; post-error reaction time indicates the reaction time for correct responses after errors of commission; miss error rate indicates omissions (Go errors) error rate (considering all trials); and false alarm error rate indicates commissions (NoGo errors) error rate (considering all trials).

30 Go and 10 NoGo trials totaling 320 trials. Trials began with a fixation cross (200–300 ms), followed by an animal image (750 ms), and then a blank screen (500 ms). Responses were allowed during presentation of the image and blank screen. Each block featured a new set of animal images, balanced in size, color, and species. Before EEG data collection, children practiced with 12 trials, including 3 orangutan and 9 other animal trials, repeating until they mastered the task. Eprime software (Psychology Software Tools, Inc.) facilitated task presentation.

Correct trials involved pressing the button for nonorangutan animals (Go trials) and not responding to orangutans (NoGo trials). Errors comprised button presses during NoGo trials (false alarms, errors of commission) and nonresponses in Go trials (misses, errors of omission).

Performance Metrics

Post-error slowing was measured using an approach described previously (56). Trials with reaction times (RTs) under 100 ms were excluded to eliminate anticipatory responses. We identified specific 5-trial sequences of hit (button press in a Go trial), hit, false alarm, hit, hit, and calculated post-error RT from the first hit following a false alarm.

Post-NoGo error accuracy was calculated by dividing the correct post-NoGo error responses by the total post-NoGo error trials. False alarm and miss rates were computed separately by dividing the total count of each error type by the total number of trials.

Electrophysiological Recording, Data Reduction, and Analysis

EEG data were recorded using the ActiveTwo system (BioSemi Inc.) with 17 Ag/AgCl scalp electrodes (Figure 1). BrainVision Analyzer version 2.2.2 (Brain Products GmbH) was used for analysis. EEG data were sampled at 1024 Hz, bandpass-filtered (0.05–30 Hz) using zero-phase shift Butterworth filters, and referenced to averaged mastoid electrodes. Ocular movement artifacts were corrected via a regression-based algorithm (57). Data were response locked to correct Go and incorrect NoGo trials. As previously described (10,52–54), incorrect Go and correct NoGo trials were excluded due to the absence of a button response needed for EEG linkage in this task. Epochs were rejected if voltage steps were >50 μV in 200 ms or trial voltage variation/channel was >150 or <0.5 μV.

ERN and Pe were quantified using mean amplitude measurements relative to a preresponse baseline −200 to −100 ms. The mean amplitude of the ERN and Pe were computed 0 to 50 ms and 300 to 500 ms after NoGo errors, respectively. As in previous work (10,52), we selected the channels with the highest mean amplitude (ERN at FCz: mean ± SD = 3.70 ± 5.25; Pe at Pz: 13.6 ± 9.16).

Power spectra were processed using a continuous wavelet, computed for single trials, and subsequently averaged. Data from 1 to 30 Hz were transformed using a complex Morlet wavelet with a Morlet parameter *c* of 3.5 applied in 30 frequency steps distributed on a logarithmic scale. To apply a percentage change baseline correction of power, the response-locked amplitude was first averaged to a preresponse baseline on a trial-by-trial basis (−200 to −100 ms). Computed average alpha (8–14 Hz) from channels O1, O2, and Oz and theta power (4–7 Hz) from channel FCz were extracted 200 to 500 ms and 100 to 300 ms postresponse, respectively. Selection of time windows was guided by visual examination of spectrograms (Figure 2).

Anxiety Assessment

Child anxiety was measured by parent report on the CBCL DSM-oriented anxiety problems subscale (CBCL-Anxiety; $\alpha = 0.79$) (58).

Statistical Analysis

A detailed description of statistical procedures can be found in Table S2. For data that were not normally distributed according to the Shapiro-Wilk test results, nonparametric tests were used. We applied Wilcoxon signed-rank tests with false discovery rate correction for multiple testing to compare response-locked alpha power (incorrect No/Go vs. correct Go

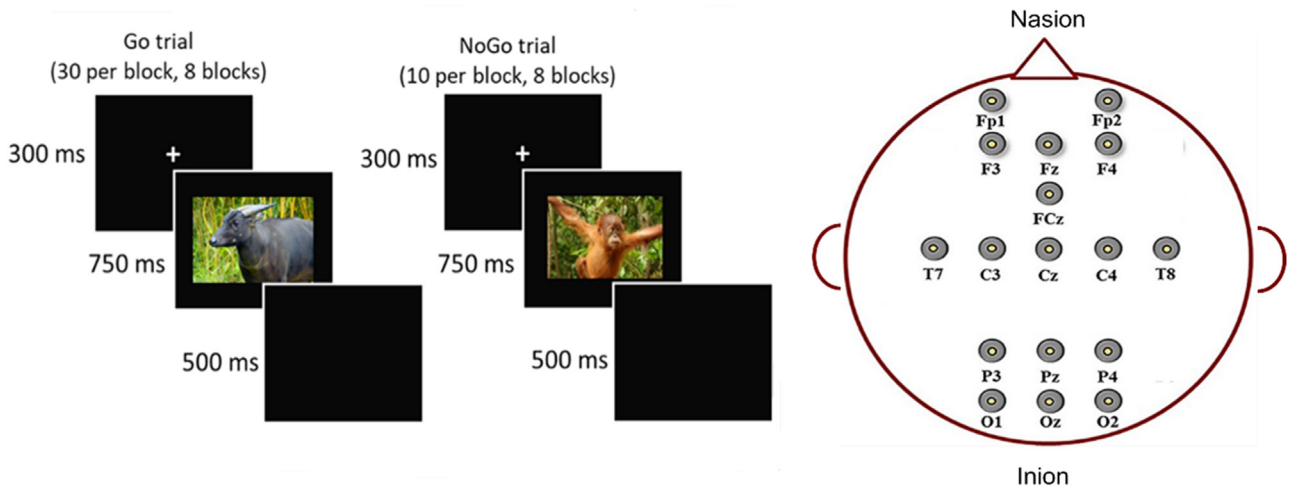

Figure 1. Experimental setup. Left panel: visual depiction of the Go/NoGo Zoo Task. Children could respond during the stimulus presentation or during the 500-ms blank screen. Right panel: representation of electroencephalograph electrode placement.

trials) across occipital electrodes (O1, O2, Oz). Correlations between variables were computed using Spearman's rho.

K-means clustering was followed by Mann-Whitney *U* tests to confirm the significance of cluster differences. To assess cluster robustness, 1000 bootstrap samples underwent K-means clustering, with cluster stability assessed by modal cluster assignment frequency. A χ^2 test was used to compare the distribution of sex across clusters, and a Welch's *t* test was

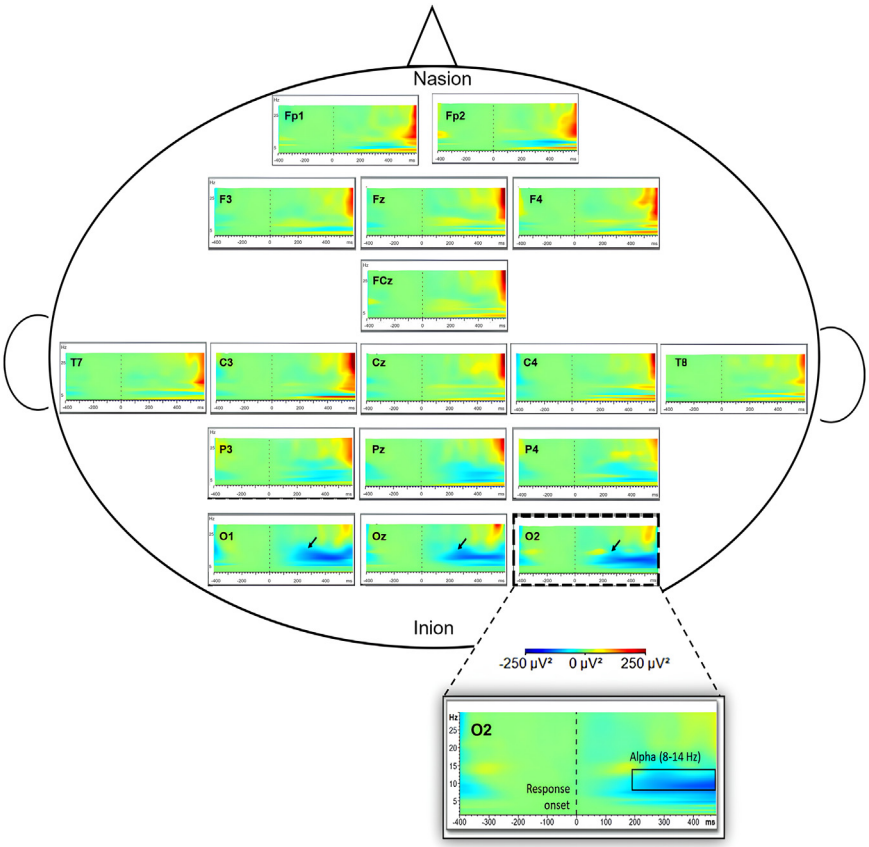

Figure 2. Response-locked wavelet frequency-decomposed spectra for all channels. Each spectrogram represents a scalp electrode shown in its corresponding position. Spectrograms show the difference between errors minus correct responses, and colder colors represent a suppression in alpha power (black arrows). Electrode O2 is a close-up to show that time 0 represents response onset and that occipital alpha power for errors was maximally suppressed during the 200- to 500-ms time window (black square).

conducted to compare age in months between clusters. Age in months was used to achieve finer precision to detect subtle developmental changes.

All analyses were conducted using R version 4.3.2 and Jamovi version 2.3.21.0 (www.jamovi.org).

RESULTS

Participant Characterization and Zoo Task Performance

As presented in Table 1, participants showed an average CBCL-Anxiety T score of 56.6 (SD = 9.1, range = 50.0–88.0), indicating variation in anxiety from normative to clinically significant. There were no differences in anxiety T scores by sex at birth (U = 3637, p = .261). Similarly, no correlation between age and anxiety was found (Spearman's rho = −0.128, p = .086). Regarding performance, the mean post-error RT for correct trials was 525.2 ms (SD = 61.2, range = 340.9–684.0), and post-error accuracy was 0.9 (SD = 0.1, range = 0.7–1.0).

Confirmation of an Occipital ERAS in Young Children

Drawing from adult studies using similar cognitive tasks (35,36), we hypothesized lower post-error than post-correct alpha power, with maximal alpha suppression at occipital sites. This was validated by analyzing wavelet frequency-decomposed spectra and confirmed at occipital electrodes (Figure 2). Wilcoxon tests on occipital electrodes (O1, O2, Oz) showed significantly lower alpha power post-error than post-correct (O1: W = 13,723, p < .001; O2: W = 14,104, p < .001; Oz: W = 13,664, p < .001) (Table 2). That is, we confirmed the presence of ERAS in young children.

We found a negative correlation between the average post-error alpha power and age in months (Spearman's rho = −0.346, p < .001) (Figure 3).

Association Between ERAS and Anxiety Symptoms and Identification of Specific Patterns in This Relationship

The correlation analysis showed that higher post-error alpha power was associated with higher CBCL-Anxiety scores (Spearman's rho = 0.217, p < .003) (Figure S1). This indicates that less ERAS was associated with more anxiety symptoms, as hypothesized. Additionally, post-error alpha power was correlated with post-error RT (Spearman's rho = 0.255, p < .001) and post-error accuracy (Spearman's rho = −0.206, p = .005). Similarly, CBCL-Anxiety was also negatively correlated with post-error accuracy (Spearman's rho = −0.188, p = .011) (Table S3).

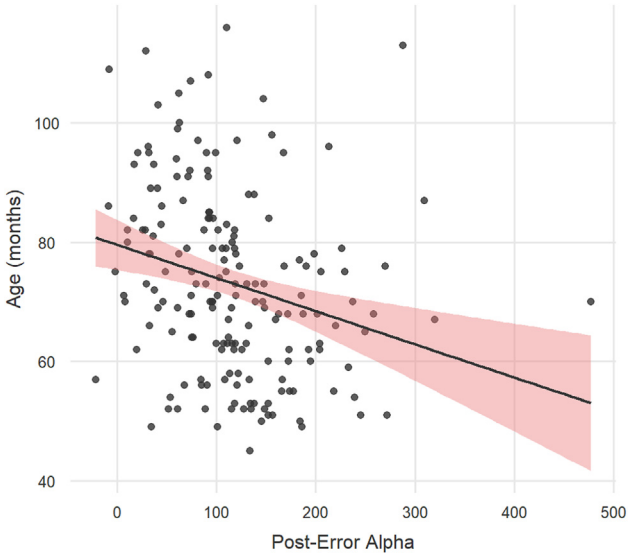

Figure 3. Correlation between post-error alpha power and age. The scatterplot illustrates the correlation between the average occipital alpha power following NoGo errors of channels O1, O2, and Oz and age in months (Spearman's rho = −0.346, p < .001).

Using K-means clustering including the variables post-error alpha, ERN, Pe, post-error frontal theta, post-error RT, and post-error accuracy, we identified 2 clusters (cluster 1, n = 89; cluster 2, n = 92). A bootstrap analysis for cluster consistency highlighted the efficacy of the clustering model (Figure S2). As shown in Figure 4, compared to cluster 2, cluster 1 was characterized by higher post-error alpha power (reduced ERAS), less negative amplitude of the ERN, more positive amplitude of the Pe, lower post-error frontal theta power, more post-error RT, and less post-error accuracy. A Mann-Whitney U test confirmed significant differences between clusters for all variables (all adjusted ps < .05) (Table S4). Additionally, a Welch's t test indicated that there was a significant difference in age between clusters (t_{174} = −6.86, p < .001, Cohen's d = −1.02), with more younger children in cluster 1. We did not observe sex differences between clusters (χ^2_1 = 1.20, p = .272).

Lastly, when comparing dimensional anxiety levels between clusters (Figure 5), a Mann-Whitney U test indicated that individuals from cluster 1 had higher CBCL-Anxiety scores than

Table 2. Comparison of Alpha Power Following Correct and Incorrect Responses for All Occipital Electrodes

Comparison	Statistic, Wilcoxon W	p Value	Mean Difference	SE Difference	Effect Size[a]	Adjusted p Value[b]
O1 Alpha: Correct vs. Error	13,723	<.001	54.5	6.98	0.666	.001
O2 Alpha: Correct vs. Error	14,104	<.001	56.3	6.46	0.713	.001
Oz Alpha: Correct vs. Error	13,664	<.001	54.2	7.06	0.659	.001

Comparison of post-correct and post-error alpha power (Wilcoxon signed-rank test) for each occipital electrode (O1, O2, Oz). The mean difference in the table refers to the average difference between the paired measurements. A positive mean difference indicates that the first measurement in the pair is higher (alpha correct).

[a]Rank biserial correlation effect size.

[b]False discovery rate correction for multiple testing.

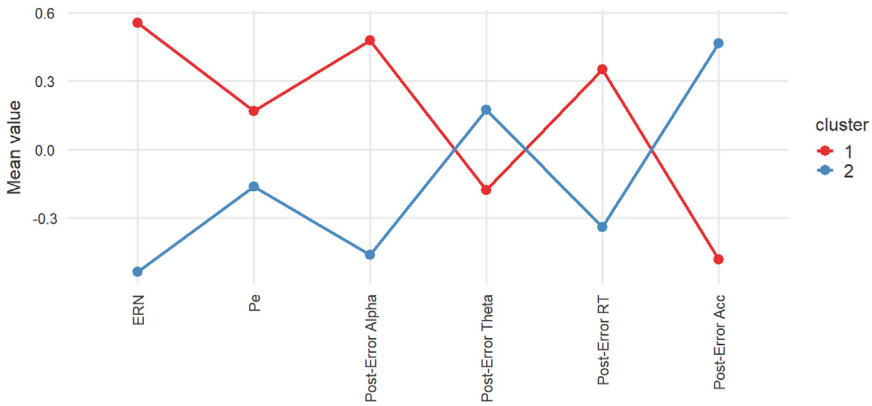

Figure 4. Distinct profiles in relation to error-related alpha suppression, error monitoring, and task performance. The plot shows the standardized means across clusters obtained by K-means clustering. Compared to cluster 2, participants from cluster 1 were characterized by less negative error-related negativity (ERN), more positive error positivity (Pe), higher post-error alpha power (average occipital alpha power [8–14 Hz] following NoGo errors of channels O1, O2, and Oz [200–500 ms]), lower post-error theta power (theta power [4–7 Hz] at channel FCz [100–300 ms postresponse]), slower post-error reaction time (RT) (RT for correct responses after errors of commission), and less post-error accuracy (Acc).

their counterparts from cluster 2 (U = 3171, p = .007, rank biserial correlation effect size = 0.225).

DISCUSSION

Our study had 2 primary aims: 1) to test for the presence of ERAS in young children, and 2) to identify neurobehavioral profiles of anxiety in this population by integrating ERAS with established markers of cognitive control and neural mechanisms of attention elicited by errors (ERN, Pe, frontal theta) and post-error performance indicators.

Confirmation of ERAS in Young Children and Occipital Scalp Distribution

As hypothesized, we confirmed lower post-error alpha power than post-correct (Figure 2, Table 2), as well as its occipital distribution, demonstrating that ERAS is a robust and reproducible phenomenon that is detectable in children as young as 4 years. ERAS may signal increased attention to external stimuli post-error, because alpha rhythm suppression aligns with heightened visual attention (59–61). Conversely, alpha power increases with internal focus, possibly indicating external attention inhibition and DMN engagement (62–66). However, alpha suppression also follows infrequent task stimuli, not just errors, which suggests that it may signal orienting toward task demands (67,68).

Additionally, we found that post-error alpha power was negatively correlated with age, indicating enhanced ERAS in older children. This suggests a developmental improvement in shifting attention from internal to external stimuli, possibly indicating growing inhibitory and attentional control mechanisms (Figure 3).

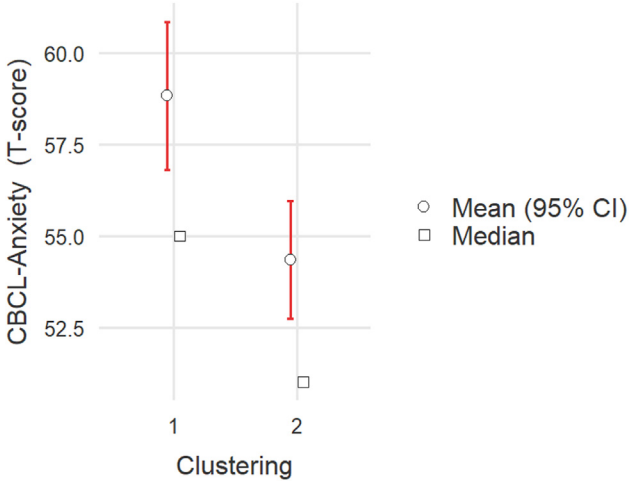

Figure 5. Comparisons of anxiety levels between clusters. A Mann-Whitney U test indicated that individuals from cluster 1 had higher Child Behavior Checklist (CBCL)-Anxiety scores than their counterparts from cluster 2 (U = 3171, p = .007, rank biserial correlation effect size = 0.225).

Identification of Neurobehavioral Profiles of Error Responding and Anxiety

Our results reveal a complex link between error processing and anxiety: Higher anxiety was correlated with lower ERAS, which implies that children with anxiety may have difficulty disengaging from internal worries, which in turn affects their attentional shift in tasks. Consistent with studies indicating that high anxiety impacts attention control and DMN engagement, research has shown abnormal DMN activity in adults with anxiety/depression during focus tasks, signaling intrusive performance worries (69–73). Patients with anxiety exhibit altered DMN connectivity at rest, associated with persistent, irrelevant worries (74–79). The disrupted ERAS in children with anxiety may reflect DMN interference, which impairs their shift from internal concerns and is related to the DMN's link to increased alpha power.

The K-means clustering revealed 2 distinct profiles, each indicative of varying degrees of cognitive control at different levels of anxiety severity (Figure 4). Cluster 1 showed a profile suggesting less effective cognitive control in the context of errors compared to cluster 2. The participants from cluster 1 presented higher post-error alpha power (reduced ERAS), less negative amplitude of the ERN, larger Pe, lower post-error frontal theta power, slower post-error RT, and less post-error accuracy. The children from cluster 1 also exhibited higher anxiety (Figure 5) and younger age than those from cluster 2. There were no sex differences between clusters, which suggests that cognitive maturation is sex independent, which contrasts with previous findings on early sex differences in anxiety (80).

The reduced post-error alpha suppression in children from cluster 1 may reflect a sustained attentional engagement with internal stimuli, consistent with difficulties in disengaging from the DMN. This persistent inward focus is consistent with our hypothesis that children with higher anxiety face challenges in redirecting their attention from internal states of worry, thus impacting their task performance.

The reduced ERN in this cluster may indicate an underactivation of the neural systems associated with error detection, error correction (16,17), or attention allocation (18). Moreover, the heightened Pe amplitudes in cluster 1 suggest increased conscious error awareness (19,20). While greater awareness of errors can facilitate corrective behavior, in the context of anxiety, it may also lead to exacerbated ruminations, potentially creating a cycle where increased error-related concern amplifies anxiety and further preoccupation with errors, as has been suggested in previous work (81). The Pe was not correlated with any adaptive behavioral metrics and only positively correlated with anxiety in bivariate analyses (Table S3). Similarly, participants from cluster 1 also presented reduced frontal theta, which could be interpreted as indicative of a compromised capacity for cognitive control and attentional modulation (18,26). These findings are consistent with previous research showing that the oscillatory dynamics of medial frontal theta and posterior alpha are implicated in the modulation of attention and cognitive control (26).

Our study also revealed a notable difference in post-error RT and post-error accuracy between clusters. Cluster 1 showed more post-error RT and less post-error accuracy than cluster 2. Post-error accuracy has been linked to an adaptive control system associated with the ERN and Pe (30). While post-error slowing is often seen as compensatory (80,82,83), research indicates that changes in speed and accuracy post-error may be independent (35,84,85), with some suggesting a nonadaptive orienting response to errors (86). This is consistent with the lack of correlation between post-error RT and accuracy (Table S3), indicating that the duration of behavioral adjustment post-error did not directly improve accuracy. While more anxious and younger children (cluster 1) may take more time to react after errors, they appear to be less able to utilize this longer response period to improve their accuracy in subsequent tasks. In this context, anxiety and younger age appear to modulate the ability to benefit from post-error slowing, potentially by exacerbating or prolonging cognitive and emotional disturbances initiated by errors.

Taken together, the K-means cluster analysis results suggest a combination of early disengagement from external stimuli to correct mistakes along with later perseveration on the conscious, emotional, or embodied representation of the mistake that may accompany the tendency to slow down the reaction after errors and make multiple mistakes in a row. This suggests the presence of less efficient cognitive mechanisms when dealing with errors, particularly in individuals prone to anxiety.

Our study, which was exploratory in nature, sets the stage for future research to elucidate the causal links between cognitive processes and anxiety. Future investigations should utilize predictive analytics and longitudinal designs to achieve a deeper understanding of these cognitive dynamics across childhood and adolescence. The variation observed in our results indicates the importance of within-participant, or trial-by-trial, analyses. Such detailed within-individual examination will clarify the dynamic relationship between cognitive processes and anxiety, aiding in identifying the development patterns of anxiety disorders. Additionally, this approach may reveal critical intervention points, facilitating early and targeted therapeutic strategies.

Limitations

While our findings offer valuable insights into the relationship between error processing and anxiety in children, it is important to note methodological limitations that affect their interpretation. A technical constraint inherent to our Go/NoGo task is the inability to obtain response-locked EEG data for omission errors and correct NoGo trials due to absence of button press, as previously described (10,52–54). Future research could explore alternative techniques to detect event-related neural activity without motor responses, which would enable a more thorough investigation of neural mechanisms that underlie both correct and incorrect inhibitory task trials.

We recognize the limited racial and ethnic diversity in our sample, which potentially impacts the generalizability of our findings. Nonetheless, our results offer valuable insights into cognitive and clinical processes within the studied population. Future studies with more diverse samples could investigate how cultural and ethnic differences influence the relationship between ERAS and anxiety in children.

Conclusions

In conclusion, mirroring adult research, our study shows that young children also exhibit occipital ERAS. Our clustering analyses indicated an association between anxiety and maladaptive error processing. Specifically, anxiety was associated with a reduction in ERAS and frontal theta, less negative ERN, an enlarged Pe, longer post-error RT, and reduced post-error accuracy. This suggests an initial disengagement from external stimuli for error correction, followed by prolonged focus on the conscious, emotional, or physical aspects of the mistake, leading to slower post-error reaction and more errors. These findings could imply either that anxiety stems from these attentional and error-related patterns or that it disrupts attention and error processing. Future research should explore this as a potential reciprocal reinforcement loop.

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