Archival Report

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The Balance N1 Is Larger in Children With Anxiety and Associated With the Error-Related Negativity

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ABSTRACT

BACKGROUND: The error-related negativity (ERN) is a brain response evoked by mistakes in cognitive tasks that is enhanced with anxiety and can predict the subsequent onset or exacerbation of anxiety in children and adolescents. A physical disturbance to standing balance evokes a brain response called the balance N1 that resembles the ERN in scalp topography and in response to a variety of moderating factors. We recently found that the balance N1 and ERN correlate in amplitude across small samples of adults.

METHODS: In the current study, we tested the effect of anxiety on the balance N1 in children (ages 9–12 years) with and without diagnosed anxiety disorders (38 children with generalized anxiety, social anxiety, and/or obsessive-compulsive disorder and 50 children without these disorders). We measured the balance N1 in response to sudden release of support from a forward leaning posture, the ERN in response to mistakes on a Go/NoGo task, and anxiety symptoms using child- and parent-report forms of the Screen for Child Anxiety and Related Emotional Disorders.

RESULTS: Both the balance N1 and the ERN were larger in the anxious group. The balance N1 was also associated with both the ERN and parent report of child anxiety symptom severity across individuals.

CONCLUSIONS: The higher measurement reliability of the balance N1 than the ERN and greater experimental control over errors suggest that balance paradigms may provide a more powerful method for investigating individual differences in error-related brain activity related to anxiety.

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The error-related negativity (ERN) is a neural response to errors that is exaggerated with anxiety. The ERN is a negative voltage deflection observable at frontocentral electroencephalography (EEG) electrodes after errors such as clicking the wrong response button (1). The ERN is thought to reflect brain activity in the anterior cingulate cortex or supplementary motor area (2,3) that may contribute to cognitive and emotional evaluation of mistakes (4), with a possible role in adaptation (5). The ERN shows potential as a biomarker for anxiety that is larger with anxiety and obsessive-compulsive disorder (6–8) and can predict the subsequent onset or worsening of anxiety in children and adolescents (6,9).

The balance N1 is evoked by a physical disturbance to standing balance and resembles the ERN. The balance N1 is a negative voltage deflection observable in frontocentral EEG after sudden, unpredictable disturbances to body posture (10) that typically require a reaction to avoid falling. The balance N1 has been localized to the supplementary motor area (11,12) with concurrent activation of the anterior cingulate cortex (13,14). We previously suggested that the balance N1 and ERN may reflect activation of a common error detection system based on similar modulation with motivation, perceived consequences, perceptual salience, expectation, development, and aging (15). We also found that the balance N1 and ERN correlate in amplitude across small nonclinical adult samples (16). Unlike tasks that evoke the ERN, physical disturbances to balance do not depend on spontaneous mistakes and offer control over the number, timing, type, and sequencing of balance errors (17,18). Furthermore, balance recovery is largely involuntary (19) and does not require any particular understanding or interpretation of the stimulus. The balance N1 increases with induction of state anxiety (20) but has not been assessed for group or individual differences in anxiety.

Anxiety and balance control have reciprocal relationships in neural circuitry, behavior, disorder presentation, and response to treatment. A sudden loss of balance can evoke acute anxious arousal, mediated in part by overlapping neural circuits for anxiety and balance control (21). For example, vestibular sensory input activates neural circuits that mediate avoidance, anxiety, and conditioned fear responses via the parabrachial nucleus (21), and norepinephrine outputs from the locus coeruleus may mediate effects of alerting and vigilance on vestibulo-motor circuits (22). State anxiety influences standing balance (20,23) and the ability to use vestibular inputs for balance (24). Anxiety and balance disorders are frequently comorbid (21,22) and appear to maintain or exacerbate one another over time (25,26). Balance clinics may refer patients to psychotherapy (27), particularly when fear and avoidance prevent the necessary exposure to adapt balance control (25).

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Although psychotherapists do not typically refer clients who report dizziness or fear of falling to balance clinics, balance training can reduce anxiety in children (28), suggesting reciprocal crossover benefits of treatment. Therefore, work to bridge these fields may yield novel treatment strategies for both populations.

We compared the balance N1 to the ERN in children with and without anxiety disorders. We hypothesized that the balance N1 would share the ERN's relationship to anxiety and would therefore be larger in children with anxiety and associated with both the ERN and anxiety symptoms across individuals. We measured the balance N1 in response to sudden releases of support from a forward leaning posture, the ERN in response to mistakes on a Go/NoGo task, and anxiety symptoms using the Screen for Child Anxiety and Related Emotional Disorders (SCARED) (29–31).

METHODS AND MATERIALS

Power Analysis

Group comparisons were powered by a medium effect (Cohen's d = 0.60) of anxiety on the ERN in children (32), requiring 36 children per group for power = 0.80 and alpha = 0.05 for a 1-sided t test. Correlations were powered by a large effect ($r > 0.60$) for the association between the balance N1 and ERN in adults (16), requiring 28 individuals for power = 0.80 and alpha = 0.05 (33).

Inclusion/Exclusion Criteria

Anxious group eligibility required generalized anxiety disorder, obsessive-compulsive disorder, and/or social anxiety disorder, which have been associated with larger ERNs (6,7). Attention-deficit/hyperactivity disorder or depression, which may be associated with smaller ERNs (34,35), warranted exclusion from both groups. Other eligibility criteria included the following: ages 9 to 12 years, cognitive and English language ability to consent/assent, and ability to stand unassisted. Children were also excluded if they had a history of severe psychopathology (e.g., psychosis).

Eligibility Interviews

Parents/guardians were interviewed by phone (by AMP) using relevant sections (attention-deficit/hyperactivity disorder, depression, and all anxiety sections) of the DSM-5 version of the Kiddie Schedule for Affective Disorders and Schizophrenia interview (36).

Participants

A total of 87 children completed the lab visit (37 children with anxiety and 50 control children). Parents and children were informed before providing written consent/assent. All procedures for this study were approved by the Institutional Review Board of Florida State University.

Overview of Experimental Procedures

Children performed a balance task to elicit the balance N1 and a Go/NoGo task to elicit the ERN (Figure 1, detailed below, order counterbalanced). Because these data were collected in association with an ongoing clinical trial (NCT05503017), children with anxiety were subsequently randomized to a brief computerized intervention before repeating the tasks. The current analyses involve only baseline pre-intervention data. To assess anxiety symptoms, children and attending parents/guardians separately completed the SCARED (29–31).

Balance Task

A lean-and-release method (37) was used to disturb balance (Figure 1). In 30 to 35 trials, children were released suddenly from a forward leaning posture, and they typically stepped forward to recover balance. The leaning posture was supported by a cable attaching the back of a safety harness to a point at the same height on the wall behind them (detailed below). A research assistant remained behind the child to ensure safety and reattach the cable between trials.

Children were asked to cross their arms, stare into a fixation cross, lean into the harness, keep their weight on their heels, and relax into the harness before release. Children practiced the lean (without release) until they understood the task and to determine foot placement for a load force of 5% of body weight. Children were told to do anything necessary to recover balance but to try to maintain visual fixation and crossed arms for 1 second after each release.

Brain and muscle activity, load force, and head accelerations (detailed below) were monitored live to ensure a steady baseline before releases. When a steady baseline was not readily achieved, children were reminded to relax or were coached on how to maintain a steady lean. Children were frequently reminded that they could take rest breaks at any time. Excluding breaks longer than 2 minutes, the balance task lasted 16 $\pm$ 6 minutes, with intertrial intervals (release to release) of 29 $\pm$ 15 seconds.

The first 20 trials were excluded from analyses as practice trials, and the remaining 10 were kept for analyses. Practice trial exclusion was decided a priori to allow initial habituation and adaptation of the balance N1 and behavior (12,38,39). Up to 5 additional trials were added to replace any of the last 10 trials that included unusual load forces (e.g., movement just before release). Offline trial selection was determined exclusively by force data before processing brain activity.

Technical Details

The leaning posture was supported by a flexible cable (1/8-inch polyvinyl chloride–coated stainless steel cable with looped ends) attaching the dorsal-D ring of a full-body safety harness to a metal clip (TP425 aluminum archery release aid; Topoint Archery) rigidly mounted to a height-adjustable wall mount through the body of a force sensor (LSB302 300 lb tension and compression load cell; Futek) that measured the load force (tension) in the cable. The force sensor output was routed through a signal conditioner (IAA100 full bridge strain gauge signal conditioning voltage amplifier; Futek) into the EEG amplifier (detailed below).

The cable was released by pressing a button (from an adjacent room) on a circuit that connected AA batteries to a solenoid actuator (DC push-pull type solenoid electromagnet,

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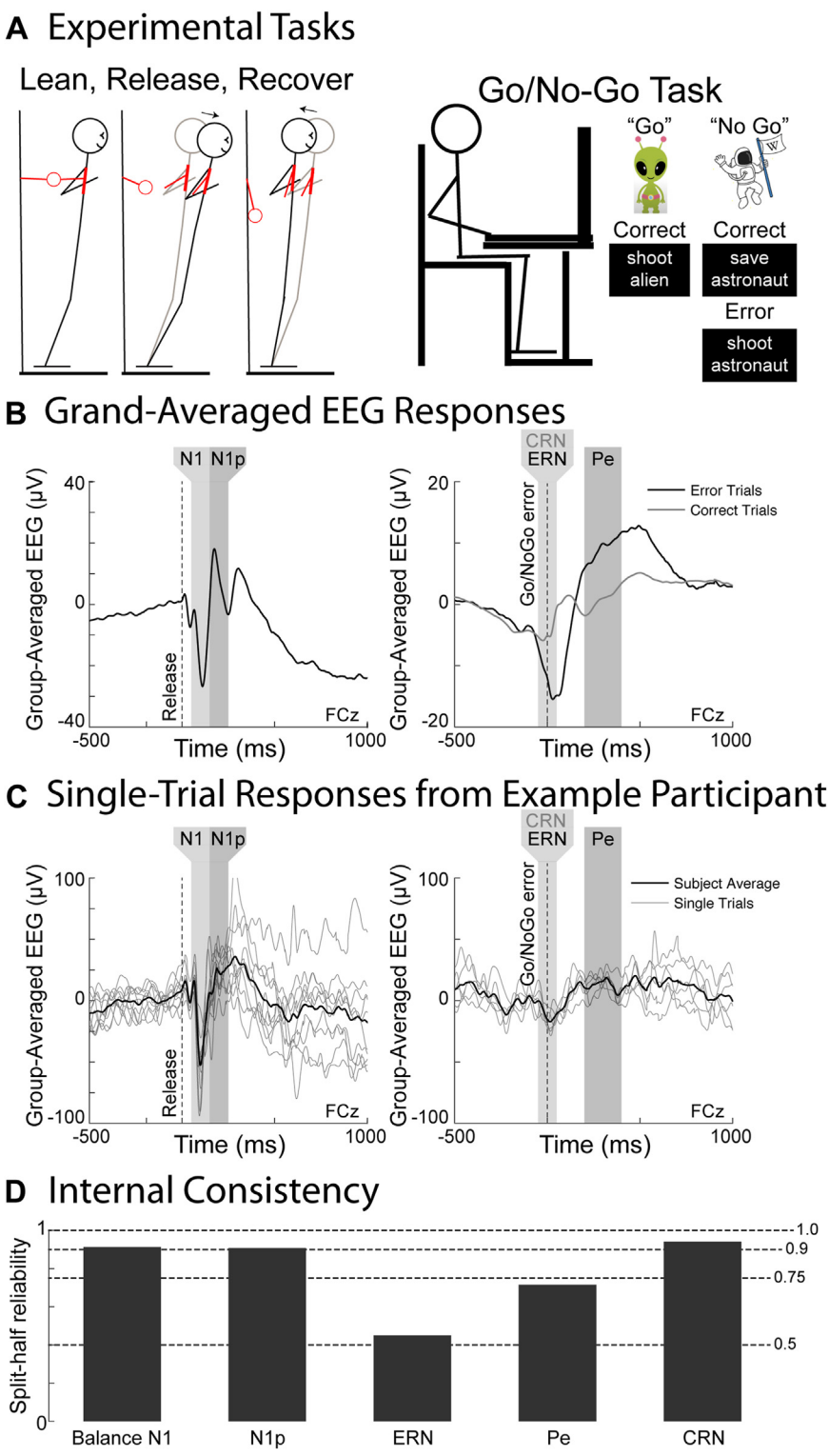

Figure 1. Tasks and outcome measures. **(A)** Children performed a lean-and-release balance task and a seated Go/NoGo task. **(B)** Grand-averaged electroencephalography (EEG) waveforms are shown for the balance task (left) and for both error (black) and correct (gray) trials in the Go/NoGo task (right). **(C)** Single-trial data (gray) are shown from an example participant (black). **(D)** The bar plot shows the split-half reliabilities for each of the brain responses. CRN, correct-related negativity; ERN, error-related negativity; N1p, N1 positivity; Pe, error positivity.

DC 5 V 18 W 0.8 N 10 mm open frame linear motion; UXCell) that mechanically pulled a sensitive trigger to unclip the cable. Metal-on-metal collisions within the clip and solenoid actuator made a quiet but audible clicking sound on release that was dampened by foam padding at contacts, foam earplugs, and a white noise machine.

Go/NoGo Task

A Go/NoGo task (40) elicited error-related brain activity (Figure 1). Children were instructed to blast aliens (75% of stimuli) by clicking a mouse button as quickly as possible and to save astronauts (25%) by not responding. Stimuli were displayed for 500 ms and replaced with a fixation cross for 1000–2000-ms intertrial intervals. To ensure that children understood the task, an experimenter observed a block of 5 practice trials, which could be repeated if necessary. Children then completed 2 blocks of 100 trials alone. Between blocks, text appeared on the screen based on accuracy: "Good job! Now go faster" (accuracy $>$ 95%), "Good job! SAVE the astronaut!" (accuracy $<$ 85%), or "Great job!" (otherwise).

Data Recording

An ActiCHamp plus amplifier (Brain Products) recorded data during tasks. Active EEG electrodes (actiCAP snap electrodes; Brain Products) were placed at Fz, FCz, Cz (international 10-20 system), and behind each ear (1–2 cm above mastoid bone) for offline rereferencing, with an active ground at AFz. Eye activity (electrooculography) was recorded by passive electrodes (E220X electrodes; Brain Products) above and below 1 eye with a forehead reference. Muscle activity (electromyography) was recorded by passive electrodes (B18 multitrodes; Brain Products) in a neck muscle (sternocleidomastoid) to indicate startle and from opposing ankle muscles (tibialis anterior and medial gastrocnemius) to indicate baseline relaxation and balance recovery behavior. Electrodermal activity was recorded by sensors on the palm of one hand (thenar and hypothenar eminences, GSR Sensor kit; Brain Products). A 3-dimensional acceleration sensor (Brain Products) was taped to the head to measure accelerations in 2 dimensions (forward-backward and up-down).

Impedances $<$25 kΩ (recommended for active electrodes) were generally obtained through a combination of abrasive and nonabrasive electrode gels (ABRALYT HiCl, High-Chloride Abrasive Electrolyte Gel, and SuperVisc High Viscosity Electrolyte Gel for active electrodes; Brain Products). Alcohol preparation pads were used to prepare skin for electrode placement.

Data Preprocessing

ActiCHamp plus data were collected with a 280-Hz anti-aliasing low-pass hardware filter, sampled at 1000 Hz, and recorded using BrainVision Recorder (Brain Products). Subsequent processing occurred in MATLAB (The MathWorks, Inc.). EEG and electrooculography data were filtered offline using consecutive forward and backward passes of a 0.1-Hz third-order Butterworth high-pass filter and then with a similarly designed 25-Hz low-pass filter (filtfilt.m). EEG data were then rereferenced to averaged mastoids, epoched separately for each task (described below), and corrected for eye movement and blink artifacts using the Gratton and Coles algorithm (41). Analyses focus on FCz, where both brain responses were largest.

Balance Task Data Processing

Balance task data were time-locked to releases, which were automatically identified by sudden loss of the load force and verified by visual inspection. Twenty practice trials were excluded from analyses, and 10 of the remaining trials were selected for analyses based on force data to minimize load force variability and the deviation from 5% body weight. EEG data were epoched into 2-second intervals, centered around release, and averaged across the selected trials within individuals.

The balance N1 was measured as the mean voltage 50 to 150 ms after release relative to a baseline of 50 to 150 ms before release. This time window is consistent with 100-ms peak latencies from lean-and-release in adults (37) and captures the balance N1 observed in the current sample (Figure 1B). Because the anxiety effect clearly extended beyond the balance N1 (Figure 2), we also measured the subsequent positivity (N1p, 150–250 ms) for exploratory analyses that were not associated with a priori hypotheses.

Head accelerations were used to assess group differences in behavior or the potential for movement artifacts. Accelerations were combined into non-negative vector magnitudes, averaged across trials, and measured as the mean within 3 time windows, including a baseline (500 ms prior to release) and windows spanning the measured brain activity (0–150 ms and 0–250 ms).

Muscle activity was used as an additional method to test for potentially confounding group differences in behavior. Muscle activity was filtered using a 35-Hz third-order Butterworth high-pass filter, demeaned, rectified, and low-pass filtered at 40 Hz (39). Muscle activity was then visually inspected to exclude individuals without a clear evoked response. This method was intended to remove cases with detached or poorly placed electrodes but may excessively exclude data from the tibialis anterior muscle (analyzed in 62% of individuals) because it is not required for balance recovery in this task. In contrast, most children were included in analyses of the medial gastrocnemius (91%) and sternocleidomastoid (78%). Muscle activity was then averaged across trials and measured as the mean within 3 time windows (500 ms before release, 0–150 ms, and 150–350 ms).

Electrodermal activity was used to assess autonomic arousal (42). Electrodermal activity was filtered between 0.1 and 1 Hz and visually inspected to exclude individuals whose sensors became detached during the experiment (retaining 84% of individuals in analyses). Electrodermal activity was then averaged across trials and measured as the mean response (2–4 seconds) relative to a baseline of 2 seconds before release.

Go/NoGo Task Data Processing

Go/NoGo task data were time-locked to responses (button press) in 1.5-second trial epochs beginning 0.5 seconds before response. Trials were automatically rejected from analyses if they contained unusual EEG activity ($>$50 μV between consecutive samples, a voltage range of 300 μV, $>$3 standard deviations above the individual's average, or $<$0.5-μV cumulative difference) within a 1-second window centered around response. After trial rejections, 2 individuals (1 from each group) were excluded from ERN analyses for having fewer than 6 artifact-free error trials (43). Data were then averaged within response types (error, correct) and baseline subtracted (300–500 ms before response).

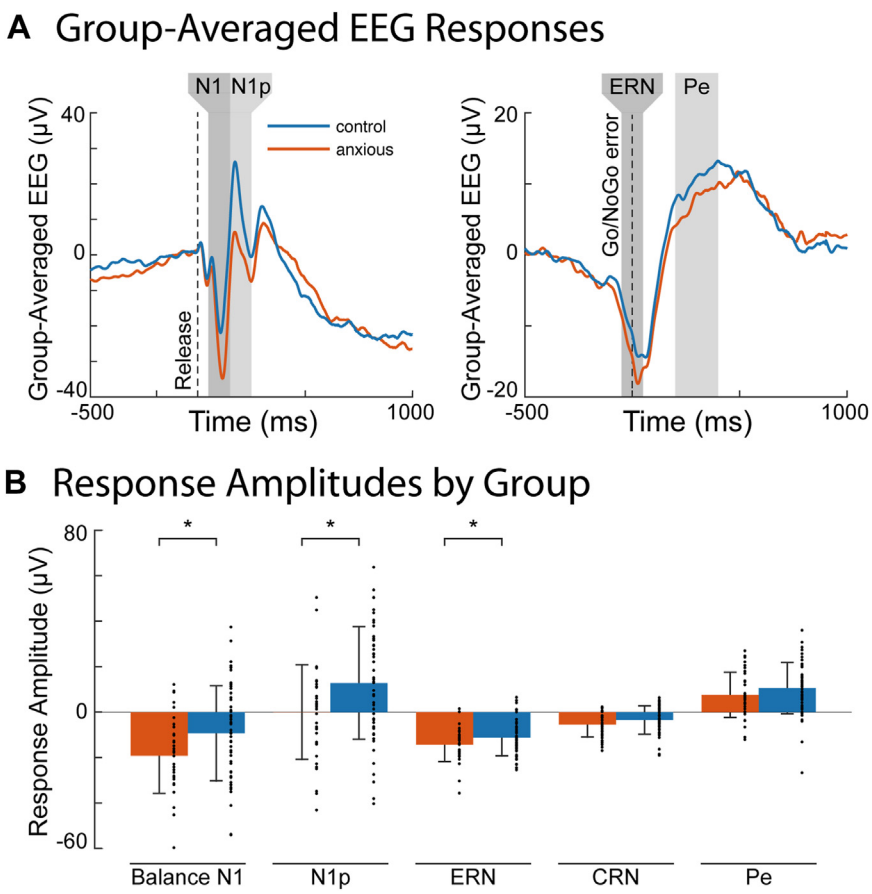

Figure 2. Evoked brain responses by group. **(A)** Group-averaged waveforms are shown for the balance task (left) and the Go/NoGo task (right) for the anxious (orange) and control (blue) groups at FCz, where responses were the largest. **(B)** Bar plots show the measured amplitudes for each brain response by group. Asterisks indicate a significant group difference at $p < .05$ (1-tailed). CRN, correct-related negativity; EEG, electroencephalography; ERN, error-related negativity; N1p, N1 positivity; Pe, error positivity.

The ERN and correct-related negativity (CRN) were measured within trial types as the mean of 100 ms centered around response. The error positivity (Pe, 200–400 ms after error responses) (44) was also measured to assess whether nonspecific factors, such as individual differences in impedance or signal quality, could drive associations between the balance N1 and ERN. Any analyses that included the Pe were exploratory and not associated with a priori hypotheses.

Statistical Analyses

Internal consistencies were assessed by split-half reliability and dependability metrics. Specifically, each brain response was compared across even and odd trial subsets using Pearson's correlation coefficient with the Spearman-Brown correction (45,46). Overall and participant-level dependability of the balance N1 and ERN were further assessed using the ERP Reliability Analysis Toolbox version 0.5.2 (47).

The directionally specific hypothesis that the balance N1 would be larger in the anxious group was tested by a 1-tailed *t* test (*p* values for 1-tailed tests are denoted by p_1). Other brain responses were similarly analyzed to assess the specificity of the anxiety effect. Additionally, the ERN was included with either the balance N1 or N1p as simultaneous predictors of group in logistic regression analyses to assess whether the 2 tasks provided unique or overlapping predictors of anxiety.

Two-tailed *t* tests were used for descriptive group comparisons that were not associated with hypotheses (age, height, weight, SCARED child report and parent report, muscle activity, head accelerations, and parent income) or Fisher's exact test (sex, race, ethnicity, and parent marital status). For the *t* tests, the Satterthwaite correction was applied to cases with unequal variances.

The hypotheses that the balance N1 would be associated with the ERN and anxiety symptom severity (SCARED child-report and parent-report form) were tested with simple linear regression analyses. To assess the specificity of the correlation between the balance N1 and ERN, we also tested for correlations with other brain responses (CRN, Pe, N1p). SCARED scores were normalized before regressions by a Box-Cox transformation (boxcox.m), which takes a natural log raised to a power determined by the skewness of the original distribution. Scatter plots display original data with statistics from transformed data. Analyses were performed using SAS software (SAS Enterprise Edition, release 3.81; SAS Institute).

Brain responses were also compared across sexes with 2-tailed *t* tests and tested for associations with age using simple linear regression analyses. Because the N1p differed

across sexes, tests involving the N1p were repeated in models that accounted for sex differences. Primary analyses were also repeated while excluding individuals with obsessive-compulsive disorder, and any changes to outcomes are reported.

RESULTS

Participant Characteristics

Children in the anxious group had higher anxiety symptom severity according to both the SCARED child- (p = .006) (Table 1) and parent-report ($p < .001$) forms. Groups were otherwise similar in age (p = .606), sex (p = .195), height (p = .653), weight (p = .613), race (p = .680), ethnicity ($p > .99$), parent marital status (p = .340), parent income (p = .836), and parent education (p = .309).

Task Behavior

In the Go/NoGo task, children responded correctly on 96% ± 5% of alien trials and incorrectly on 32% ± 15% of astronaut trials. Reaction times were shorter for errors ($p < .001$, error 321 ± 53 ms, correct 371 ± 37 ms). Groups did not differ in accuracies or reaction times (all ps $> .19$). After trial exclusions, 2 individuals were excluded from ERN analyses for having too few artifact-free error trials. The remaining individuals (36 with anxiety, 49 control) had 16 ± 8 error trials for analyses.

Two individuals in the anxious group were excluded from measurement of the balance N1 because an issue with the live force display resulted in larger balance disturbances. The remaining individuals (35 with anxiety, 50 control) had 10 lean-and-release trials entered into analyses. Prerelease load forces were 5.1% ± 0.6% body weight and did not differ across groups in magnitude (p = .62) or variability (p = .28). Groups did not differ in head accelerations (all ps $> .19$), muscle activity (all ps $> .64$), or electrodermal activity (p = .53).

Brain Activity

The balance N1 was more internally consistent than the ERN (Figure 1D). Internal consistencies were excellent for the

Table 1. Participant Characteristics

	Control Group, n = 50[a]	Anxious Group, n = 37[a]	p Value
Age, Years	10.6 ± 1.1	10.7 ± 1.2	.606
Sex, Female/Male	28/22	15/22	.195
Race[a]			.680
Black	8	9	
Multiracial	5	3	
White	37	25	
Ethnicity[a]			>.99
Hispanic	6	4	
Non-Hispanic	44	33	
Parent Marital Status			.340
Married	28	20	
Not married	22	9	
Parent Education, Years Beyond High School	4.4 ± 2.3	4.9 ± 2.5	.309
Parent Income, $/Year	100,000 ± 58,000	97,000 ± 43,000	.836
SCARED, Child Report	25.4 ± 13.3	35.5 ± 18.0	.006
SCARED, Parent Report	8.7 ± 6.7	33.4 ± 13.1	.001
KSADS Diagnoses, Number of Children With Threshold (or Subthreshold)			
Generalized anxiety disorder	0 (0)	16 (6)	
Obsessive-compulsive disorder	0 (0)	17 (1)	
Social anxiety disorder	0 (0)	22 (1)	
Selective mutism	0 (0)	4 (2)	
Separation anxiety disorder	0 (0)	5 (3)	
Panic disorder	0 (0)	1 (0)	
Agoraphobia	0 (1)	0 (3)	
Specific phobia	2 (7)	8 (1)	
Major depressive disorder	0 (0)	0 (0)	
Persistent depressive disorder	0 (0)	0 (2)	
Attention-deficit/hyperactivity disorder	0 (4)	0 (11)	

Values are presented as n or mean ± SD. p Values for sex, race, and ethnicity are from Fisher's exact tests. Other p values are from 2-tailed 2-sample t tests, with Satterthwaite correction for unequal variances when applicable.

KSADS, Kiddie Schedule for Affective Disorders and Schizophrenia; SCARED, Screen for Child Anxiety and Related Emotional Disorders.

[a]ns include individuals with missing data. Specifically, error-related negativities could not be measured in 2 individuals who committed too few errors. Balance N1 data were excluded from 2 individuals who received larger balance disturbances due to an issue with the live display of the force data.

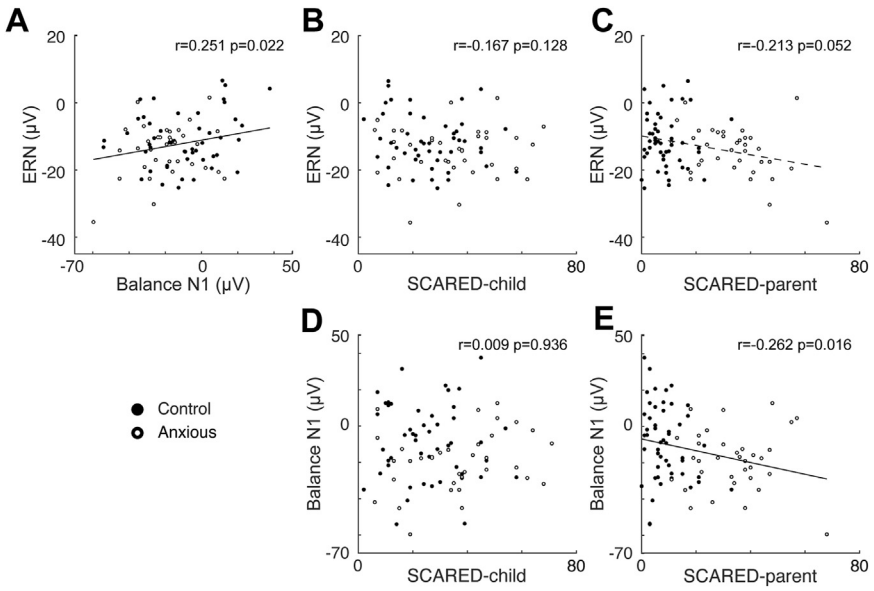

Figure 3. Correlations between outcome measures. In the top row, error-related negativity (ERN) amplitudes are plotted against **(A)** the balance N1, and child anxiety symptom severity according to the **(B)** child or **(C)** parent report. In the bottom row, the balance N1 is plotted against **(D)** child report and **(E)** parent report of child anxiety symptom severity. In all panels, children with anxiety disorders are represented by open circles. Plots display original data values with statistics from transformed data when appropriate. SCARED, Screen for Child Anxiety and Related Emotional Disorders.

balance N1 (0.91) and N1p (0.90), poor for the ERN (0.45), moderate for the Pe (0.71), and excellent for the CRN (0.94). The overall dependability of the balance N1 was 0.91 (95% CI, 0.88–0.93), with dependability above 0.9 for all individuals. Overall dependability of the ERN was 0.58 (95% CI, 0.45–0.70), with dependability below 0.8 for all individuals.

Evoked brain responses were more negative for the anxious group (Figure 2). The anxious group had a more negative balance N1 (p_1 = .010, Cohen's d = 0.530) and N1p (p_1 = .007, d = 0.559) in the balance task. In the Go/NoGo task, the anxious group had a more negative ERN (p_1 = .040, d = 0.391) and a trend for a more negative CRN (p_1 = .052, d = 0.365), but the Pe was similar across groups (p_1 = .107, d = 0.277).

When combined in a logistic regression analysis, neither the balance N1 (p = .061) nor the ERN (p = .234) remained significant predictors of anxiety group membership. However, the N1p (p = .025) did predict group membership in a model that also included the ERN (p = .108).

The balance N1 was positively correlated with the ERN (r = 0.251, p = .022) (Figure 3) and CRN (r = 0.282, p = .010), with a trend for a similar association with the Pe (p = .077). The N1p was positively correlated with the balance N1 (r = 0.671, $p < .001$) and Pe (r = 0.338, p = .002), but not with the ERN (p = .76). Parent report of child anxiety symptoms was correlated with the balance N1 ($r = -0.262$, p = .016), with a similar trend for the ERN ($r = -0.213$, p = .052), but neither were associated with child report of child anxiety (balance N1 p = .936, ERN p = .128).

The N1p was larger in girls (p = .0001), but no other brain responses were associated with sex (all other ps $> .15$) or age (all ps $> .64$). The N1p lost significance (p = .051) when predicting group membership in combination with both the ERN and sex, but no other N1p outcomes changed when sex was accounted for. When individuals with obsessive-compulsive disorder were excluded, no primary outcomes changed except the association between ERN and parent-reported child anxiety (r = 0.292, p = .016).

DISCUSSION

This is the first study to demonstrate that the balance N1 is larger in a clinically anxious population and associated with anxiety symptom severity. A previous study showed that the balance N1 increased with state anxiety (i.e., perceived threat and fear of falling) within individuals (20), but within-subject effects do not necessarily transfer to individual differences (48,49). The current results show that the effect of anxiety on the balance N1 extends to group and individual differences in trait anxiety. These findings suggest that the balance N1 may provide a useful tool to investigate relationships between balance control and anxiety. It may be informative to assess how the balance N1 relates to different anxiety constructs or differs across anxiety disorders in larger or more homogeneous anxious populations. Additionally, although this study focused on anxiety disorders with an enhanced ERN, the balance N1 may also be informative in anxiety disorders with more frequent vestibular-related balance complaints, such as panic disorder and agoraphobia (50–52). These findings also raise the question of whether anxiety factors into relationships between the balance N1 and balance ability (53,54).

Our results suggest that the balance N1 provides a more reliable measure of error-related brain activity than the ERN. The larger ERNs in our children with anxiety disorders and the trend with parent-reported anxiety symptoms are consistent with systematic reviews and meta-analyses that have found that ~85% of studies detect an increased ERN in clinically anxious samples (8), and ~50% of studies detect correlations with trait anxiety (55). This study also provides a larger replication in children of the correlation between the ERN and balance N1 that we previously reported in younger and older

adults (16), suggesting this association is robust across the life span. Although the balance N1 was similarly correlated with the ERN and CRN, it is unlikely that these associations are driven by systematic biases, such as individual differences in impedance, because the ERN was not correlated with the N1p, and the balance N1 was not correlated with the Pe. Because measurement reliability limits observable effect sizes (56), it is possible that the balance N1 could provide greater statistical power for assessing effects of anxiety than the ERN. Balance tasks are also feasible in populations that have difficulty with cognitive tasks, such as toddlers (57) and elderly populations with Parkinson's disease (54).

We make no attempt to identify underlying neural processes and only provide evidence that these brain responses yield similar information about anxiety as outcome measures. The balance N1 and ERN both involve recruitment of the anterior cingulate cortex and supplementary motor area (3,14), but their localizations differ slightly (~1 cm) under single-source assumptions (11). However, even if these potentials shared no overlapping cortical neurons, they might still share inputs broadly conveying cognitive and/or emotional biases on information processing in both tasks. It would be interesting to explore whether manipulations of the ERN (58) transfer to the balance N1 or whether manipulation of the balance N1 impacts anxiety. The group difference in the balance N1 clearly extended into the N1p, about which there is almost no previous literature (59,60), but it may provide a unique predictor of anxiety based on the lack of correlation with the ERN. Given the fixed latency of involuntary balance recovery behavior, the evoked brain activity could be equally considered stimulus- or response-locked, and so this positivity could be related to both stimulus-locked P300 and response-locked Pe, which partially overlap in cognitive tasks (44).

This study has several methodological limitations. The practice trials were intended to allow initial habituation and adaptation of the balance N1 and behavioral response (12,38,39) but also proved necessary for children to learn to relax into the harness for a consistent sensory stimulus (i.e., release) across trials. Clinical applications may prefer a method with a more natural starting position (e.g., applying rather than removing a load) to require fewer practice trials. Additionally, behavior may have differed across groups in ways that we failed to detect, particularly across the earliest practice trials (61), which were too variable to assess fairly. We also cannot rule out the possibility that the balance N1 was affected by the assistant who remained in the room during the balance task. However, there is no evidence to suggest that the effect of an observer (who witnesses a participant's mistakes) on the ERN (62) would transfer to a similar effect of an observer (who ensures safety while a participant is subjected to external forces) on the balance N1. Finally, we note that children in the control group reported unusually high levels of anxiety symptoms (63), and group assignments may have differed if children had been included in eligibility interviews. Such differences are unlikely to strengthen the observed effects because the brain responses were related more to parent-reported than to child-reported anxiety symptoms. However, children should not be disregarded as useful informants because discrepancies between parent and child reports can yield meaningful information about child and adolescent psychopathology (64), and different experimental tasks may yield outcomes that are more related to the child report (65).

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AMP contributed to conceptualization, methodology, statistical analyses, eligibility interviews, data collection and management, funding acquisition, manuscript and figure preparation, and manuscript revision. NBS provided resources, supervision, and mentorship and reviewed and approved the manuscript. AM provided supervision and mentorship and reviewed and approved the manuscript. GH contributed to conceptualization and methodology, reviewed manuscript drafts, and provided supervision, mentorship, funding, and resources.

The processed single-trial and subject-averaged waveforms for the 2 tasks at Fz, FCz, and Cz electrodes and a spreadsheet of relevant subject data are freely available through Open Science Framework (https://doi.org/10.17605/OSF.IO/VX5UM).

All authors report no biomedical financial interests or potential conflicts of interest.

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