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Error-Specific Cognitive Control Alterations in Generalized Anxiety Disorder

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Abstract

BACKGROUND—Trait anxiety is reliably associated with enhanced neural responses following errors: meta analyses have described how the electrophysiological response to errors known as the Error-Related Negativity (ERN) is increased in anxious individuals, particularly in relation to worry. The ERN has been related to a broader class of control signals, particularly via a common theta band denominator, but it is unknown whether worry relates to these alternative medial frontal metrics. Moreover, it is unclear if increased ERN in anxiety relates to altered cognitive control.

METHODS—In this report we examine EEG activities in Generalized Anxiety Disorder (GAD, N=39) and control (CTL: N=52) participants during an executive control task. We leveraged a previously defined theta-band network to examine if an altered control signal in GAD underlies a differential implementation of cognitive control.

RESULTS—GAD and CTL groups were reliably dissociated by both error- and conflict- related neural activity, in both the time and frequency (i.e. theta band) domains. Moreover, we demonstrate that ERN, error-related theta power, and the single trial correlation between theta and response time were unique predictors of GAD status. Overall, we were able to account for nearly 1/4 of the group variance and successfully classify GAD from control participants with 2/3 accuracy.

CONCLUSIONS—Collectively, these findings suggest that multiple neural metrics of error processing may uniquely distinguish clinical anxiety from healthy individuals, and that mechanisms of control also differ in GAD; finally, these error-related neural measures have the potential to be sensitive and specific bio-signatures of anxiety.

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Keywords

Anxiety; ERN; N2; theta; cognitive control; classification

Background

A wealth of studies has identified a reliable correlation between trait anxiety and an enhanced electrophysiological response to motor errors of commission (1; 2). A growing literature has interpreted this error-driven electrophysiological response known as the error-related negativity (ERN) in terms of a generic, primitive, domain-general process that signals the need for increased cognitive control (3–5). This view of the ERN is consistent with the notion that errors are inherently aversive, and motivate behavioral adaptation (6; 7).

Two recent meta-analyses have highlighted the specificity and sensitivity of this relationship. Moser and colleagues detailed how the relationship between anxiety and ERN amplitude appears to be specific to apprehension or worry (1). Cavanagh and Shackman described a related generality, where dispositional anxiety is also associated with larger signals of conflict or punishment (7). Errors, conflict, and punishments all share common psychological significance of inherent aversion and signal the increased need for control. The electrophysiological markers of these events also share a common denominator of mid-frontal theta band activities, which has been advanced as a candidate mechanism for the implementation of cognitive control (8).

Despite the potential functional similarity of errors (i.e., ERN), and conflict (i.e., the N2) and apparent similarity of these ERPs in terms of a common theta-band activity, few studies have systematically evaluated these neural metrics within the same sample. For instance, no study has simultaneously examined ERN and N2—as well as their theta-band representations—in relation to clinical anxiety. In this report we aimed to address the outstanding question of whether clinical anxiety is characterized by increased error-related theta, as well as time- and frequency-domain representations of conflict processing—or alternatively, if clinical anxiety is better characterized by increased ERN alone.

Furthermore, we assessed whether larger theta band related activities have functional consequences and reflect enhanced cognitive control in clinical anxiety. Prior work has revealed that after an error, mid-frontal cortex is transiently theta-band phase-synchronous with other brain regions involved in the exertion of control (see (8) for a review of 11 replications of this finding). Moreover, ERN/theta power amplitude is predictive of the degree of post-error response time slowing following an error (again significant in a meta-analysis(7)). These two findings offer established methods to examine the interactive neural systems involved in the realization and communication of cognitive control. For the first time, we test whether anxious participants have enhanced control (i.e., larger single trial brain-behavior relationship, more phase synchrony) following potentiated neural signals to errors and conflict.

We tested these relationships in a relatively pure sample of clinically anxious individuals high on dispositional worry who met diagnostic criteria for General Anxiety Disorder

(GAD). We not only provide novel evidence for altered features of cognitive control in GAD, but we additionally demonstrate that the use of theta band network activities contribute to better classification of GAD vs controls than event-related potentials alone.

Methods

The current study combined participants from two separate previously-published studies that examined ERN in relation to GAD (9; 10). The current study focused on 39 participants with a diagnosis of GAD (but not comorbid depression), and 52 individuals with no current DSM-IV diagnosis (healthy controls: CTL). All diagnoses were made using the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders (DSM) fourth edition (11). Self-reported symptoms of anxiety and depression were gathered from most participants using the Mood and Anxiety Symptom Questionnaire (MASQ; (12)). The MASQ is a 62-item self-report measure of mood and anxiety symptoms. In one of the papers that previously reported on these data, we found that ERN was related to the General Distress Anxiety symptoms subscales (9); however, no relationship between ERN and MASQ subscales was found in the other study (10).

Table 1 shows demographic and questionnaire information. Between groups, age did not significantly differ for either study or the combined total ($t < 1.18$) but MASQ scores differed in all comparisons for each study and for the combined total ($t > 2.8$). Within each group, the age range of the studies differed but the symptomatology did not (GAD $t < 1$, CTL $t < 1.77$). For additional information on recruiting and patient information, see (9; 10).

An arrow version of the flanker task was administered using Presentation software (Neurobehavioral Systems, Inc., Albany, California, USA). On each trial, five horizontally aligned arrowheads were presented. Half of all trials were compatible (e.g. “< < < <”) and half were incompatible (e.g. “< < > <”). The order of compatible and incompatible trials was random. All stimuli were presented for 200 ms followed by an ITI that varied randomly from 2,300 to 2,800 ms. Participants performed a practice block containing 30 trials. The actual task consisted of 11 blocks of 30 trials (330 trials total) with each block initiated by the participant.

Continuous EEG recordings were collected using an elastic cap and the ActiveTwo BioSemi system (BioSemi, Amsterdam, Netherlands) at 1024 Hz with a 208 Hz low pass filter. Thirty-three electrode sites were used, based on the 10/20 system, as well as two electrodes on the right and left mastoids. The electrooculogram generated from eye movements and eye blinks was recorded using four facial electrodes: horizontal eye movements were measured via two electrodes located approximately 1 cm outside the outer edge of the right and left eyes. Offline, eye blinks were removed using independent component analyses (13). Data were then referenced to the average of the left and right mastoids.

Only participants with six or more errors were included in EEG analyses (14–16), removing one participant from the CTL group and two from the GAD group. All data were analyzed at the FCz electrode. Event-related potentials (ERPs) were low pass filtered at 20 Hz and baseline corrected from –500 to –300 ms (9; 10). The N2 was quantified as the mean

voltage ± 25 ms around 275 ms following stimulus onset; the ERN / CRN was quantified as the mean voltage ± 25 ms around 50 ms following error / correct trials.

The analytic procedure leveraged two existing approaches that previously examined the role of theta power in behavioral control (17; 18). In order to control for trial count and motor activity differences between correct and error epochs, a sub-set of correct trial EEG epochs were selected based on the closest RT match to each error trial (17). Conflict-related analyses were taken from subsets of correct trials where congruent and incongruent trials each followed congruent trials, capitalizing on the Gratton effect where congruent-congruent trial sequences maximally differ from congruent-incongruent trial sequences (18).

All time-frequency and statistical methods used wavelet convolution (for time-frequency plots) and band-pass filter-Hilbert (for frequency-specific analyses) with parameters identical to previously published work (19). Wavelet power was normalized by conversion to a decibel (dB) scale, allowing a direct comparison of effects across frequency bands. Time-frequency plots were cluster thresholded based on 200 permutations of group label using two-sided $p < .05$ criteria. For discriminant analyses, stimulus-locked theta (4–8 Hz) power was taken from a 300–500 ms window; response-locked theta was extracted from 100–250 ms post-response.

All connectivity analyses (detailed in Figure 4) follow the approach used in Figure 4 of Cavanagh, Cohen & Allen, 2009 (17). This approach investigated the 3-way interplay between 1) midfrontal theta power to errors, 2) midfrontal-lateral theta phase synchrony following errors, and 3) post-error RT slowing. Here we investigated if these phenomena and the relationships between each differ between GAD and CTL. Thus, Figure 4 here is arranged as a triangle, with vertices representing brain or behavioral differences and connections between vertices representing the interplay between items 1, 2 and 3 above. The interplay between vertices is taken as evidence of theta-band mediated networks for behavioral adaptation, providing a test of the hypothesis that larger error signals in GAD represent alterations in the implementation of cognitive control.

These single-trial analyses utilized a Laplacian transform of the data, which is necessary to minimize volume conduction prior to connectivity analyses (20). The vertices of the analytic triangle were based on single-trial estimates of: 1) theta power following errors, 2) Inter-site phase clustering over trials (ISPC_trials) which was calculated as the consistency in phase angle difference between FCz and separate dorsolateral electrodes (i.e., F3/4) across error trials, and 3) post-error RT slowing, calculated as the post-error minus error RT difference, although other methods of quantification (i.e., post-error RT alone, post-error minus preceding correct trial (21)) yielded nearly identical findings to those reported here. Note that all single-trial analyses could not be performed on ISPC_trial, as it is a trial average, thus analysis of trial-to-trial synchrony required the computation of ISPC_time which is the consistency in phase angle difference over time (within any single trial). Thus ISPC_time can be computed on single trials, but it was constrained to a scalar value representing the average of phase angle differences over a period 0 and 200 ms (17). Vertex connections were quantified as the following: 1&2) A bar graph of average Spearman's correlations between theta power and ISPC_time in the time period of 0 to 200 ms, 1&3) A time course of

Spearman's correlations between instantaneous theta power and RT slowing, 2&3) A bar graph of average Spearman's correlations between ISPC_time (0 to 200 ms) and RT slowing.

Classification used the LASSO algorithm (Least Absolute Shrinkage and Selection Operator (22)), a penalized logistic regression method as well as a linear support vector machine (SVM). The average of 5000 iterations of CTL and GAD samples were reported from three cross validation methods: 5-times, 10-times, and leave-one-out. The LASSO penalty shrinks regression coefficients in the training stage (setting many to zero) based on tuning parameters, highlighting the smallest subset of important and reliable predictors. LASSO yields multiple solutions to the training set given different tuning parameters that constrain the sparseness of the logistic regression. To select amongst these parameters, the regularization weight with the best accuracy on a validation set was selected; to diminish capitalization on chance, the predictive accuracy of these LASSO weights were assessed on a unique test set (23). Average accuracies on this unbiased second validation set are reported here. The SVM does not select the most influential features, so it did not require a separate validation set and all accuracies were reported based on the sole test set.

Results

Behavior

Analyses of behavioral data were performed separately from trial-count matching and selection procedures implemented for EEG analysis. GAD and CTL participants did not differ in the number of errors committed (GAD: median errors=29, intra-quartile range=14; CTL: median errors=33, intra-quartile range=26; $t(86) = 1.22, p = .23$) or RT for any trial type ($t < 1.6$), see Figure 1. When quantifying post-error slowing as the RT difference between post-error and the preceding error trials, the groups were still highly similar in measures of central tendency ($t(86) = 1.21, p = .23$) and dispersion (standard deviations $t(86) < 1$). Collectively then, the two groups did not significantly differ on any behavioral measure.

ERPs & Time-Frequency

Figure 2 displays the event-related potentials for each group. An Analysis of Variance revealed a significant Group (GAD, CTL) * Type (conflict, congruent) * Event (stimulus, response) 3-way interaction ($F(1,86)=4.5, p=.037$), and a significant Group * Type 2-way interaction ($F(1,86)=9.370, p=.002$). Simple effects contrasts revealed that conflict N2 ($t(86) = 2.22, p = .03$) and ERN ($t(86) = 2.95, p = .004$) were significantly different between groups, whereas congruent N2 ($t = 1.53, p = .13$) and CRN ($t = .25, p = .80$) were not. Figure 3 displays the group differences in the time-frequency spectrographs of total power (phase-locked, like an ERP, as well as non-phase locked) in these conditions. These findings reveal enhanced power in the theta band (4–8 Hz) for GAD vs. CTL participants in both the conflict and error conditions. There were no significant group differences in inter-trial phase consistency in the theta band for any of these conditions, suggesting that theta power is specifically altered in GAD. This is an important distinction, as it has recently been argued that non-phase locked power is more sensitive to conflict (24).

Common and Unique Variance

Table 2 presents correlations between GAD status and all error- and conflict-related neural measures. As expected from Figures 1 and 2, GAD was associated with a larger ERN and N2, as well as increased error- and conflict-related theta. ERN and N2 were uniquely related, whereas error- and conflict-related theta were uniquely related. To examine whether these measures predicted unique variance in GAD status, a stepwise regression analysis predicted GAD status using these four variables. The ERN was the best single predictor of GAD accounting for 9% of the variance in group status ($F(1,86) = 8.69, p < .01$); theta power predicted another 9% ($F(1,85) = 9.17, p < .01$; incremental $F(1,85) = 8.86, p < .01$) and conflict-related variables failed to account for a significant increment (at the .05 level). This finding suggests that error trial activities effectively capture the discriminant variance of interest. While Figure 3 shows a delta-band difference for stimulus-locked activities, delta band activities did not contribute any meaningful variance to the analyses in this report¹. Next we address what these error-specific theta differences may mean for cognitive control in GAD.

Mechanisms of control

In order to test whether larger error signals in GAD are associated with altered cognitive control, an *a priori* analytic suite was leveraged (17). Above, we described how groups did not differ in post-error behavioral adjustment at the group level, but they did differ in the degree of ERN amplitude and mid-frontal theta to errors. Figure 4 details Laplacian-transformed theta-band network findings involved in communicating the error signal to influence post-error behavioral adjustment.

Fig 4a details the recently described finding of enhanced mid-frontal theta power to errors. When the groups were collapsed, there was no main effect for the predictive power of error-related mid-frontal theta power on post-error behavioral adjustment ($t < 1$); however, this was due to a significant difference in the directionality of this relationship between groups ($t(86) = 2.70, p = .008$), with each group having near significant one-sample relationships (Fig 4b: CTL: frontal theta predicted RT *slowing*; $t(36) = 1.91, p = .06$, GAD: frontal theta predicted RT *speeding* ($t(50) = -1.80, p = .08$). This finding in the control group thus replicates prior findings that frontal theta power predicts RT slowing following an error (8; 17). However, this relationship is reversed in GAD—providing the first evidence that enhanced frontal theta in GAD relates to an alteration in cognitive control.

The second facet of this control network was the theta band phase-synchronous relationship between medial and lateral frontal sites following errors (17). Single trial right-lateralized phase connectivity over this time range (ISPC_Time) significantly correlated with the degree of mid-frontal theta power ($t(87) = 18.15, p < .001$), but this measure did not differ between groups ($p = .10$), see Fig 4c. There was a significant increase in medio-lateral phase connectivity (ISPC_trial) immediately following errors in both right and left lateral sides ($t(87) > 5.55, p < .001$; Fig 4d inset). The GAD group had enhanced phase connectivity

¹When stimulus-locked delta power is included in the stepwise-regression models it does not add any unique variance. When network-level analyses were run with delta-band phenomena, none of the connectivity effects were significant as main effects nor as moderated by GAD group.

compared to CTL, but this was only on the right side and only in the error minus correct difference (0 to 200 ms: $t(86) = 2.20, p = .03$), see Fig 4d.

Finally, when collapsed across groups there was no significant main effect of right lateralized ISPC_time predicting post-error behavioral adjustment ($t < 1$), but again this was due to directional differences in the groups ($t(86) = 2.14, p = .04$), where the groups had an inverted dissociation as observed in the midfrontal power findings (CTL: theta synchrony predicted RT *speeding*; $t(36) = -1.27, p = .21$, GAD: theta synchrony predicted RT *slowing* ($t(50) = 1.80, p = .08$), see Fig 4e. This collection of phase synchrony findings complement the interpretation of altered cognitive control in GAD: there was evidence for enhanced phase synchrony in right lateral frontal cortex following errors, and this measure predicted behavioral alteration in the upcoming trial differently for the two groups. The dissociations in brain-behavior relationships suggest that the GAD group exert control differently, but not necessarily more or less efficiently, than the CTL group.

Theta Network Signals Boost Classification of GAD

A second stepwise regression was used to predict GAD, with independent variables from the previous stepwise regression (ERN and theta power) as well as indices of the theta band network features described in Figure 4 (theta ITPC, the theta-RT correlation, theta ISPC, and the ISPC-RT correlation). A three-factor solution accounted for 23% of the group variance (see Table S1). Adding on to the prior step-wise regression findings, the theta-RT correlation was the sole additional variable to account for significant discriminating variance². Across the entire sample, greater anxious symptomatology as measured by the MASQ general anxious distress subscale significantly correlated with larger ERNs ($r_{(78)} = -.24, p = .035$) and the theta-RT relationship ($r_{(78)} = -.32, p = .005$) but not theta power ($r_{(78)} = .03, p = .80$).

Figure S1 displays the accuracies of classification based on different algorithms, cross-validation measures, and feature inputs. In general, findings were robust to algorithms and cross-validation methods. While SVM classification based on power-RT correlations had the highest total accuracy (66.2%), this pattern was not robust when using LASSO (61.3%) and it had unimpressive sensitivity (53.6%). SVM classification based on all three measures (ERN, theta power, and theta-RT β) provided the next highest total accuracy (65.2%), but ERN alone offered compelling stability across algorithmic approaches (standard deviation was at most 50% the size of other feature variance). While these outcomes do not offer an objectively optimal discrimination, together they demonstrate the robustness of these measures.

²All analyses were re-run with a minimum of N=20 errors to check if including participants with low trial count skewed the analyses (GAD: N=26, median errors=34, intra-quartile range=13; CTL: N=35, median errors=43, intra-quartile range=16). The statistical significance of group differences remains unchanged with a few exceptions. Fig 1 error rate was diminished ($p = .01$) and post-error slowing was enhanced ($p = .01$) in the GAD group (but not post-error minus error RT, $p = .84$). The Fig 4 theta-RT relationship is similar but underpowered ($p = .20$). Table 2 theta-RT relationship fails to add significant variance to the prior step-wise regression outputs. Classification accuracy remained at 62–63% across the types of cross-validation methods used. These similarities suggest that these candidate bio-signature findings are robust, even when behavioral differentiation is more volatile.

Conclusions

In the current study, we demonstrated that both error- and conflict-related ERPs (i.e., ERN and N2, respectively) differentiate GAD from controls; moreover, GAD participants were characterized by increased time-frequency representations of these effects (i.e., error- and conflict-related theta, respectively). By leveraging multiple error-specific network activities, we accounted for 23% of the group variance and effectively classified patients with up to 66% accuracy. This more than doubles the amount of variance accounted for in previous work that only utilized the ERN (9; 10).

Mid-frontal theta activities have been proposed to reflect a general mechanism for the realization and implementation of control (8). An enhanced theta response was common across psychological events requiring a need for control (i.e., errors and conflict) in GAD; yet, errors contained all the unique variance required to dissociate GAD from controls. While collinearity is an issue for multiple regressions, pattern classification of stimulus-locked activities were not as powerful as error-related activities³. This error-specific dissociation suggests that the single psychological event of errors captures the common variance of anxiety-enhanced theta to varied signals of the need for control. Mechanistically, this offers an opportunity to probe the relationship between these error markers and the subsequent implementation of cognitive control.

At first glance, it may be surprising that the ERN and theta power accounted for unique variance in GAD compared to controls. However, these are mathematically distinct manifestations that capture both common and unique neural activities. These ERP and time-frequency representations were uncorrelated with each other, and error-related ERPs and theta power accounted for significant independent variance in group differentiation. As an ERP component, the ERN captures temporally specific broad low frequency enhancements, primarily theta and delta, that are phase-locked across trials (19; 24; 25). As the modulus of the complex plane after wavelet convolution, theta power reflects total (phase-locked and non-phase-locked) amplitudes over a temporally smeared but more frequency-specific range. Thus each method is uniquely effective at capturing aspects of a common underlying mid-frontal theta dynamic that is altered in GAD.

Using a stepwise regression, we found that the ERN alone provided the largest effect; however, error-related theta and theta-RT coupling provided additional and unique predictors of GAD status. Thus, multiple neural measures of error processing—derived from the same data—could be leveraged to distinguish GAD from healthy participants. When ERN and error-related theta were combined with theta network activities involved in the implementation of control, classification was powerful (66% total accuracy), stable (high sensitivity and specificity) and robust (across cross-validation procedures, EEG feature selection, and number of errors). Diagnostically, this discriminant specificity of errors paves the way towards a plausible error-based bio-signature for differentiating GAD from controls. Slight differences in algorithmic outputs are partially due to trial count: whereas the LASSO requires a second validation set (diminishing trials available for training), the SVM does not

³All classification accuracies based on stimulus-locked activities were <61.8%

and thus benefits from more training data. These competing approaches are presented here to bolster future patient classification approaches: feature, algorithm, and cross-validation successes are likely to be dependent on each other and it is beneficial to demonstrate the robustness of outcomes across a variety of approaches. A future endeavor may aim to leverage the strengths of varying algorithms to provide a single optimized approach.

Further evidence of specifically altered theta-band activities in GAD was provided by the examination of the medial-lateral frontal network involved in the implementation of cognitive control. The same network shown in Figure 4 has been advanced as a way to operationally define the mechanisms of cognitive control in this task (17). Medio-lateral phase synchrony is a proposed mechanism for the communication of the need for control (8), and the enhancement of this feature in GAD patients suggests an alteration in the implementation of cognitive control, and not just a louder mid-frontal “alarm bell” of ERN/theta power.

The degree of post-error adjustment was used as an objective indicator of cognitive control, and the GAD group indeed had altered brain-behavior dynamics compared to controls. It is highly unlikely that the differential exertion of cognitive control described here could be identified using standard neuropsychological tasks of executive function. Indeed, even behavioral measures on this task could not differentiate the groups; only brain and brain-behavior relationships differentiated GAD from controls. Theta power predicted RT speeding vs. slowing whereas theta band phase synchrony predicted RT slowing versus speeding in GAD vs. controls, respectively. It is hard to provide an *a posteriori* explanation for these dissociated outcomes, particularly since this type of trial-to-trial brain-behavior literature is quite sparse at the moment. Theta power has been shown to predict both post-error slowing (7) and post-error speeding (26) depending on task demands, demonstrating appropriate flexibility in the effective implementation of control. It is possible that this simple task had no overt optimal solution for control, so two systems responded differently to facilitate adjustment: with a reactive medial system and a proactive medio-lateral system trading off in the adjustment of response readiness versus response caution. This is a testable hypothesis using the AX-CPT (27–29) to assess reactive versus proactive control in anxiety.

This report aimed to advance beyond simple group differentiation, which is well-represented in the biological psychiatric literature, towards a mechanistic and diagnostic account of clinical cognitive neuroscience. The next challenge will be to test the ability of error-related EEG measures to sensitively and specifically dissociate GAD from other clinical groups, and to account for demographically relevant moderators such as gender (30). Here we provide evidence that enhanced error signals in clinically anxious individuals are associated with an alteration in the implementation of cognitive control, and these combined features can discriminate patients from controls with impressive power, stability, and robustness. This approach to understanding cognitive control in anxiety is not only mechanistically revealing, but has compelling potential as a simple, fast, and inexpensive bio-signature relevant to psychiatric practice.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Performance in GAD and CTL groups on the flanker task. Error rate was similar between groups. While both groups displayed faster errors and slower post-error trials, there were no significant differences in RTs.

Figure 2. Event-related potentials. As in prior work, GAD had larger conflict (N2) and error-related (ERN) voltage enhancements. Topographical maps are condition differences in the significant time windows, ± 10 uV. Asterisks indicate $p < .05$ between groups.

Figure 3.

Time-frequency plots of GAD minus control. Topographical maps are GAD minus CTL from 300 to 500 ms for stimuli and 100 to 250 ms for responses (Congru/Cor: ± 5 dB, Incong/Err: ± 10 dB). Outlines show statistically significant differences between groups $p < .05$, permutation corrected.

Figure 4.

A theta band network for behavioral adaptation following errors differed between GAD and controls. Horizontal magenta lines indicate group differences ($p < .05$). Vertical blue lines indicate main effects (i.e. above zero), $p < .05$. A) Theta power was larger in GAD following errors (inset is average from 100 to 250 ms), B) Increased theta predicted RT slowing in CTL but RT speeding in GAD (inset shows average from 300 to 500 ms). C) Power correlated with phase synchrony between medial and lateral sites after errors. D) Right sided medio-lateral phase synchrony was increased after errors (see inset, average of 0 to 200 ms) and it was larger in GAD. E) Increased synchrony predicted RT speeding in CTL but RT slowing in GAD.

Table 1

Demographic and questionnaire information for GAD and CTL participants in each study.

	GAD			CTL		
	Total	Study 1	Study 2	Total	Study 1	Study 2
N	39	15	24	52	21	31
% Female	95%	87%	100%	85%	62%	100%
Age (SD)	26.56 (10.07)	31.67 (13.30)	23.38 (5.69)	25.29 (8.46)	30.62 (9.76)	21.68 (4.97)
Has MASQ	82%	93%	75%	92%	100%	87%
MASQ Distress: Depression	27.47 (10.52)	26.71 (6.40)	28.06 (13.03)	18.21 (6.70)	17.38 (7.24)	18.85 (6.30)
MASQ Distress: Anxiety	25.69 (7.43)	25.50 (5.52)	25.83 (8.80)	15.13 (4.26)	13.95 (3.98)	16.04 (4.31)
MASQ Anhedonic Depression	63.91 (14.21)	63.64 (10.00)	64.11 (17.07)	49.90 (12.27)	46.43 (13.80)	52.59 (10.41)
MASQ Anxious Arousal	28.47 (8.00)	26.86 (5.46)	29.72 (9.49)	20.15 (4.29)	19.10 (3.32)	20.93 (4.80)

Table 2

Correlations between clinical group (GAD), the error-related negativity (ERN), N2, error-related theta, and conflict-related theta. All values are two-tailed, all EEG data are linked-mastoids referenced. For ERPs, greater negative values indicate enhanced activity, for power the relationship is isomorphic. Pearson correlations; those between continuous variables are product-moment correlations, while the correlations with the categorical GAD variable is a point-biserial correlation. N=88.

	GAD	ERN	Conflict N2	Error- theta
ERN	-.30 **			
Conflict N2	-.23 *	.55 **		
Error theta	.30 **	-.03	.005	
Conflict theta	.27 *	-.02	.002	.85 **

*
p<.05,

**
p<.01.