Frontal EEG asymmetry and symptom response to cognitive behavioral therapy in patients with social anxiety disorder

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ABSTRACT

Although previous studies have shown that socially anxious individuals exhibit greater relative right frontal electroencephalogram (EEG) activity at rest, no studies have investigated whether improvements in symptoms as a result of treatment are associated with concomitant changes in resting brain activity. Regional EEG activity was measured at rest in 23 patients with social anxiety disorder (SAD) before and after cognitive behavioral therapy (CBT). Results indicated that patients shifted significantly from greater relative right to greater relative left resting frontal brain activity from pre- to posttreatment. Greater left frontal EEG activity at pretreatment predicted greater reduction in social anxiety from pre- to posttreatment and lower posttreatment social anxiety after accounting for pretreatment symptoms. These relations were specific to the frontal alpha EEG asymmetry metric. These preliminary findings suggest that resting frontal EEG asymmetry may be a predictor of symptom change and endstate functioning in SAD patients who undergo efficacious psychological treatment.

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1. Introduction

Social anxiety disorder (SAD) is characterized by extreme fear and avoidance of social and performance situations (American Psychiatric Association, 2000). With a lifetime prevalence estimated to be as high as 12%, SAD is the third most common psychological disorder (Kessler et al., 2005). SAD has an early onset, a chronic course, and a debilitating impact on quality of life in interpersonal, emotional, and occupational domains. Numerous controlled trials have demonstrated that cognitive behavioral therapy (CBT) is efficacious in the treatment of SAD (Moscovitch et al., 2009; Smits & Hofmann, 2009), with approximately 50–70% of patients experiencing significant symptom reduction after 12 weeks of therapy (Swinson et al., 2006).

Despite the proven efficacy of CBT in reducing symptoms of social anxiety, little is known about the impact of CBT interventions on the neural mechanisms underlying social anxiety. Imaging studies on the neurobiological effects of CBT among patients with depression as well as those with anxiety disorders have shown significant treatment-related changes across broadly distributed brain regions (Porto et al., 2009; Roffman et al., 2005). However, only one previous study has investigated the association between symptom improvement during CBT and corresponding brain changes in patients with a principal diagnosis of SAD (Furmark et al., 2002), and this study used positron emission tomography (PET).

The search for neural mechanisms underlying CBT interventions for SAD is of considerable theoretical and practical importance. Intervention studies provide us with a means by which to examine how environmental influences impact brain–behavior relations. Studies may also be useful in terms of identifying aspects of neuronal function at pretreatment that may predict endstate functioning, thereby leading to improvements in the prognosis and treatment of SAD.

Of particular interest to the present study are models that link frontal brain asymmetry to individual differences in affective style and emotion processing. Davidson and Fox have theorized that the frontal lobes are differentially involved in positive versus negative affective states and corresponding motivated behaviors (Davidson, 2000; Fox, 1991), with left frontal areas of the brain mediating the experience of positive emotions (e.g., joy, happiness, etc.) and approach behaviors, and right frontal areas of the brain mediating the experience of negative emotions (e.g., fear, sadness, etc.) and withdrawal behaviors. Moreover, patterns of frontal electroencephalogram (EEG) asymmetry may serve as an index of risk for
a variety of emotion-related disorders, including depression and anxiety. A number of studies over the past three decades have supported these theoretical predictions (see Coan and Allen, 2004). Non-clinical samples of adults selected for high levels of shyness (Schmidt, 1999) and social anxiety (Beaton et al., 2008), or clinically diagnosed with SAD (Davidson et al., 2000) have been shown to exhibit significant relative elevations in right frontal brain activity when assessed during resting states or periods of acute emotional provocation.

Although no studies to date have investigated patterns of change in frontal EEG activity among individuals with SAD before and after CBT, preliminary research has demonstrated that frontal EEG asymmetry can be balanced following therapeutic interventions. For example, in a recent randomized controlled study, patients with posttraumatic stress disorder who received CBT exhibited decreased right frontal EEG activity from pre- to posttreatment relative to those assigned to a waitlist control condition (Rabe et al., 2008).1 Across a number of other studies, significant modifications of EEG alpha asymmetry (increased relative left or decreased relative right frontal EEG activity) have been observed in depressed adolescents who received massage and music therapy (Jones and Field, 1999), in healthy individuals (Davidson et al., 2003) as well as those with a history of suicidal depression (Barnhofer et al., 2007) exposed to an 8-week mindfulness meditation program, and in depressed individuals undergoing repetitive transcranial magnetic stimulation treatment (Funk and George, 2008; but see Spronk et al., 2008). Moreover, the administration of acute cortisol and prednisone, which generate anxiogenic effects, has been shown to increase right frontal EEG alpha activity among healthy participants (Schmidt et al., 1999; Tops et al., 2005). Finally, a study that examined frontal asymmetry in young adults at two time points 1 year apart (Blackhart et al., 2006) found that greater relative right frontal EEG resting activity at time 1 was significantly associated with higher levels of trait anxiety (controlling for symptoms of depression) at time 2, suggesting that EEG asymmetry may predict the development of future anxiety symptoms. Similarly, Beaton et al. (2008) found that a nonclinical sample of socially anxious undergraduate students demonstrated greater relative right frontal EEG activity at rest, but only after accounting for symptoms of depressed mood.

We investigated whether CBT was associated with changes in frontal EEG asymmetry in individuals with SAD and whether pretreatment EEG had utility in terms of predicting posttreatment functioning. Patients with a principal diagnosis of generalized SAD completed 12 sessions of standardized group CBT. At pre- and posttreatment, resting regional EEG activity was recorded as part of a larger study (see Miskovic et al., 2011). We hypothesized that: (i) at pretreatment, SAD patients would exhibit greater right than left frontal resting EEG activity; (ii) patients would shift significantly from greater right to greater relative left frontal brain activity from pre- to posttreatment; and (iii) greater pretreatment left frontal EEG asymmetry would uniquely predict greater treatment-related decreases in symptoms of social anxiety. Given the documented links between depression and frontal EEG asymmetry (e.g., Henriques and Davidson, 1990; Schaffer et al., 1983), the high rate of comorbidity between SAD and depressive disorders (Brown et al., 2001), and the significant degree of overlap observed in clinical samples between symptoms of social anxiety and depression (Brown and Barlow, 2009), we accounted for symptoms of depression in our analyses.

2. Method

2.1. Participants

Thirty three outpatients were recruited for this study. Eight of these individuals discontinued treatment and their participation in the study. Two participants had experienced adverse effects on EEGs (spectral power >3 SD from mean) and were thus excluded from the analysis, leaving 23 (12 male) eligible Caucasian, right-handed participants for the current analysis. All participants were assessed and treated at the Anxiety Treatment and Research Centre (ATRC), an urban Canadian anxiety treatment clinic (Hamilton, Ontario), and each received a principal DSM-IV-TR (1) diagnosis of SAD (generalized subtype), as determined by trained clinicians on the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders – 4th edition (SCID-IV: First et al., 2001). Clinical severity ratings (CSR) ranged from 4 to 7 (M = 5.57, SD = .90), with CSRs of 4 and above representing significant interference and distress associated with the principal diagnosis. Seventeen participants (69.6% of the sample) received at least one current comorbid DSM-IV diagnosis secondary to their SAD, with major depressive disorder (single episode or recurrent) and generalized anxiety disorder the most common comorbid diagnoses (n = 7 each), followed by specific phobia (n = 6), and obsessive compulsive disorder (n = 5). Participants ranged in age from 19 to 73 years (M = 35.74 years, SD = 15.0). Of the 23 patients, 15 had never been married, 5 were currently married, and 3 were previously married; 19 had a high school education or greater; 11 earned an income of $40,000 (CDN) or more per year.

Exclusionary criteria included current mania, psychosis, significant suicidal ideation, and/or organic brain disorders. Individuals with current substance abuse/dependence were excluded if the patients could not agree to refrain from using substances prior to and during treatment and experimental protocols; and/or were deemed by the clinical team to be unsuitable for group CBT targeting SAD (2 participants were included in the present study with secondary diagnoses of alcohol and cannabis dependence). Finally, participants were excluded if they consumed 0.6-blockers within 3 days of the EEG testing; and alcohol, marijuana or antihistamines within 12 h of EEG testing. Patients were requested to maintain stable medication type and dosage throughout the study, from 1 month prior to their first EEG assessment, until after their final testing session and were required to report their medication adherence at each testing and treatment session. As participants were recruited from a community outpatient psychiatric clinic situated within a hospital, 65% of them were taking psychotropic medications at the beginning of treatment, with an average of 2.79 (SD = 2.3) medications per patient. Some patients (39%) were taking selective serotonin reuptake inhibitors (SSRI) alone or in combination with another medication such as benzodiazepines and antipsychotic medications. Participants who reported taking psychotropic medications pro re nata (PRN, or “as needed”) were also required to maintain dose stability throughout the course of the study. Among all patients taking psychotropic medication, reported duration of medication treatment at their stable dose prior to their first study session ranged from 12 to 56 weeks (M = 28.7 weeks). Four participants reported on their weekly adherence forms that they had either begun a new psychotropic medication or increased their dose of a pre-existing prescription during the previous week. Our data analytic approach with respect to these participants is described below.

2.1.1. Clinical treatment procedures

Participants completed a 12-session standardized composed of group CBT for SAD at the ATRC in St. Joseph’s Healthcare in Hamilton, Ontario. Sessions lasted approximately 2 hours each and were administered by 2-3 qualified therapists, with 7-9 patients per group. Therapists followed a manualized protocol based on Antony and Swinson (2008) in their delivery of treatment, which included psychoeducation, cognitive restructuring, in-session and between-session exposure exercises and social skills training.

2.1.2. Experimental procedures

Participants were tested in the Child Emotion Laboratory at McMaster University. All laboratory procedures were approved by the participating university and hospital ethics committees and written consent was obtained from participants prior to testing. This study was part of a larger one (Miskovic et al., 2011), in which patients were assessed across four separate visits: two before, one during, and one after treatment. For the purpose of the present study, we examined asymmetry at pretreatment (initial visit) and posttreatment only in order to determine (a) whether CBT was associated with pre-to-post changes in frontal EEG asymmetry and (b) whether pretreatment EEG had utility in terms of predicting posttreatment symptoms and endstate functioning. Regional EEG was recorded for a 6-min resting period, which alternated 1-min condition blocks (eyes-open/eyes-closed segments) at both pre- and posttreatment. Each participant’s pre- and posttreatment assessments were scheduled to occur within a window of approximately 2 weeks before and after the first and final group CBT session.

1 Other studies have reported a variety of treatment-related electrocortical effects in patients with anxiety disorders other than SAD, including changes in event-related potentials during CBT for spider phobia (Leutgeb et al., 2009) and changes in EEG gamma activity during CBT for generalized anxiety disorder (Oathes et al., 2008).
2.1.3. Measures

Clinical measures. At pre- and posttreatment, both an independent clinician blind to the purpose of the study and patients' treatment status and one of the group therapists rated each participant's illness severity on the Clinical Global Impression Severity Scale (CGI; Guy, 1976). The CGI Severity Scale has been used in previous research as a valid and reliable measure to evaluate the efficacy of CBT for SAD (Zaidi et al., 2003).

Self-report measures. At the end of each EEG session, we examined self-report responses on the Social Phobia Inventory (SPIN; Connor et al., 2000) and the Beck Depression Inventory – Second Edition (BDI-II; Beck et al., 1996) at pre- and posttreatment. The SPIN is a 17-item inventory measuring symptoms of social fear, avoidance and arousal. The BDI-II is a 21-question self-report inventory for measuring the severity of depression, and is composed of items relating to cognitive, emotional and physical symptoms of depression. Change scores for the SPIN and BDI-II were computed by subtracting posttreatment from pretreatment total scores. Using the regression method, residual SPIN scores were also computed separately for pre- and posttreatment by partitioning out BDI-II scores from SPIN scores at each time point.

2.1.4. EEG recording and data reduction

EEG recording. EEG was recorded using a StressCap (Electro-Cap, Inc., Eaton, OH) with electrodes positioned according to the international 10/20 Electrode Placement System (Jasper, 1958). Each electrode site was filled with a small amount of electrolyte gel (Omni-prep) and gently abraded. Electrode impedances below 10kΩ per site were considered acceptable, as were impedances of up to 500Ω between homologous sites. EEG was recorded from F3/F4, C3/C4, F7/F8, O1/O2, but only data from the left and right mid-frontal (F3, F4), central (C3, C4), and parietal (P3, P4) regions are presented here. All electrodes were referenced to the central vertex (Cz). EEG was amplified by individual SA Instrumentation Bioamplifiers. The filters were set at 1 Hz (high pass) to 100 Hz (low pass), with a 60Hz notch filter. The data were digitized on-line at a sampling rate of 512Hz.

EEG data reduction and analysis. The EEG data were visually scored for artifact due to eye blinks, eye movements, and other motor movements when amplitudes exceeded ±50μV, using the software developed by James Long Company (EEG Analysis Program; Caroga Lake, NY). This program removes data from all channels if artifact is present on any one channel. All artifact-free EEG data were analyzed using a discrete Fourier transform (DFT), with a Hanning window of 1-s width and 50% overlap of epochs. Power (microwatts squared) was derived from the DFT output in three alpha frequency bands (alpha-I: 8–10 Hz; alpha-II: 10–12 Hz; full alpha: 8–13 Hz). Spectral power was estimated for the different subbands because previous work has demonstrated both the lower and higher Hz components of the alpha band may have separate factor structures (Goncharova and Davidson, 1995) and that each of these alpha bands may be related to shy and socially anxious behaviors (Schmidt and Fox, 1994). A natural log (ln) transformation was performed on the EEG power data to normalize the distributions. Resting EEG was collapsed across eyes-open and eyes-closed. Resting data from eyes-open are highly related to those from eyes-closed and are typically averaged (Hagemann and Naumann, 2001). Indeed, in the present study, pretreatment, r-values >.96, p-values <.001, and posttreatment, r-values >.82, p-values <.001, frontal alpha EEG data for the eyes open and eyes-closed condition were highly correlated. Separate EEG asymmetry scores were computed for the mid-frontal, central, and parietal regions (e.g., ln(right) − ln(left)). Because EEG alpha power is inversely related to scalp-recorded cortical activity, positive asymmetry scores are thought to reflect greater relative left EEG cortical activity (Allen et al., 2004a; Davidson, 1988).

3. Results

3.1. Effectiveness of CBT on symptom change

Paired t-tests (pre- versus posttreatment) on patients' CGI severity scores indicated that illness severity decreased significantly from pretreatment to posttreatment as rated by both the independent, blind clinician, t(18) = 3.98, p = .001 (M = 5.05 ± 0.78 versus 4.37 ± 1.11), and the group therapist, t(22) = 6.10, p < .001 (M = 5.30 ± 0.82 versus 3.83 ± 1.15). As expected, patients’ self-reported social anxiety and depression symptoms at pre- versus posttreatment also indicated a significant decrease in both the SPIN, t(22) = 5.68, p < .001 (M = 45.74 ± 12.2 versus 30.30 ± 15.3) and BDI-II, t(22) = 3.34, p = .003 (M = 24.30 ± 9.0 versus 17.26 ± 11.8) total scores after receiving treatment.

3.2. Resting EEG asymmetry

An ANOVA with Time (pretreatment, posttreatment) and Region (frontal, central, parietal) was performed to examine changes in resting EEG asymmetry before and after CBT separately for alpha I, II, and full alpha. For alpha II (i.e., 10–13 Hz), a significant Time by Region interaction, F(2,44) = 3.85, p = .03, ηp² = .15, indicated that there was a change in EEG asymmetry from pre- to posttreatment in the frontal region, t(22) = 2.60, p = .02, but not in the central, t(22) = 0.10, p = .92, or parietal, t(22) = 0.16, p = .13, regions (see Fig. 1). As predicted, individuals displayed a shift from relatively greater right to left frontal EEG asymmetry with treatment.2

3.3. Correlations between frontal EEG asymmetry and self-report measures

Table 1 presents the results of two-tailed Pearson correlations between resting EEG asymmetry and self-report measures. As predicted, pretreatment resting frontal alpha EEG asymmetry was negatively related to posttreatment SPIN and BDI-II scores such that greater relative left frontal resting EEG asymmetry at pretreatment was associated with lower social anxiety and depression symptoms after treatment, as shown in Fig. 2A and B. Of particular interest, results demonstrated that greater relative left frontal EEG asymmetry was correlated with greater decreases in CGI severity scores from pretreatment to posttreatment (p = .13).

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2 There was a Time by Region interaction on resting frontal EEG asymmetry in the 8–13 Hz frequency band, F(2,42) = 6.95, p = .005, ηp² = .25, but a follow-up paired t-test indicated there was no significant change in frontal EEG asymmetry from pre- to posttreatment (p = .13).
asymmetry at pretreatment was associated with greater decrease (pre- minus posttreatment scores) in social anxiety and depression symptoms during treatment, as shown in Fig. 2C and D.Overall, the model accounted for 50.2% of the variance in posttreatment SPIN scores, \( F(3,19) = 6.39, p = .004 \). Pretreatment SPIN scores did not account for a significant amount of variance in posttreatment SPIN scores, \( R^2 = 6.2, F(1,21) = 1.38, p = .25 \). After accounting for depression, pretreatment SPIN scores and resting frontal alpha EEG asymmetry accounted for a significant amount of variance in posttreatment SPIN scores, \( \Delta R^2 = 44.1, \Delta F(2,19) = 8.40, p = .002 \), with each variable accounting for a significant amount variance (pretreatment SPIN: partial \( r = .58 \), semi-partial \( r = .50 \), \( p = .01 \); pretreatment EEG asymmetry: partial \( r = -.51 \), semi-partial \( r = -.42 \), \( p = .02 \)).

We also examined the potential predictive utility of pretreatment resting frontal EEG asymmetry over and above patients' pretreatment social anxiety symptoms. Accordingly, a similar regression analysis was performed with pretreatment BDI-II scores entered on the first step, pretreatment SPIN scores entered second step, and pretreatment frontal alpha EEG asymmetry entered on the third step. Pretreatment BDI-II scores did not significantly predict posttreatment social anxiety symptoms, partial/semi-partial \( r = .25 \), \( p = .25 \). Both pretreatment SPIN scores, partial \( r = .53 \), semi-partial \( r = .52 \), \( p = .01 \), and pretreatment frontal alpha EEG asymmetry, partial \( r = -.51 \), semi-partial \( r = -.42 \), \( p = .02 \), were still significant predictors of posttreatment social anxiety symptoms.

### 4. Discussion

We investigated changes in the pattern of frontal EEG asymmetry among patients with SAD who completed 12 sessions of group CBT. In conjunction with symptom amelioration from pre- to posttreatment, significant electrocortical changes occurred that were characterized by a shift from greater relative right to left frontal alpha EEG asymmetry at rest. These findings are consistent with the growing literature demonstrating that frontal EEG asymmetry...
is sensitive to the effects of therapeutic interventions across various types of samples (e.g., Barnhofer et al., 2007; Davidson et al., 2003; Jones and Field, 1999; Rabe et al., 2008).

At pretreatment, SAD patients exhibited greater relative right frontal alpha EEG asymmetry at rest. This finding is consistent with the predictions of existing EEG asymmetry–emotion models (Davidson, 2000; Fox, 1991) and with empirical reports of elevated right frontal brain activity during rest or emotional provocation among nonclinical and clinical samples of socially anxious individuals (Beaton et al., 2008; Davidson et al., 2000; Schmidt, 1999). Of particular interest, the present study demonstrated that the nature and degree of symptom change that occurred as a result of treatment could be predicted by pretreatment resting frontal EEG asymmetry. Pretreatment frontal alpha EEG asymmetry (less right-sided or more left-sided activity) at rest was associated with greater reductions in social anxiety from pre- to posttreatment and lower posttreatment symptoms of social anxiety, even after accounting for pretreatment symptoms of social anxiety and depression. The strongest changes in resting frontal EEG asymmetry from pre- to posttreatment were observed in analyses of the high alpha range (i.e., 10–13 Hz). Although some have argued that alpha subbands are structurally (Goncharova and Davidson, 1995) and functionally (Klimesch, 1999) heterogeneous, most recent evidence does not support the suggestion that the subbands are differentially sensitive to predicting variation in temperament traits (Shackman et al., 2010).

The frontal EEG asymmetry–emotion model would predict that greater relative right frontal asymmetry would be related to increased social anxiety symptoms atpretreatment among patients with SAD. However, pretreatment resting frontal EEG asymmetry in the present study was not related to pretreatment symptoms of social anxiety. There are several possible explanations for this apparently puzzling finding. One important factor may have been the restricted range of SPIN scores among SAD patients at pretreatment, with most participants’ scores clustering between 40 and 60. Conversely, pretreatment BDI-II scores, which were significantly correlated with greater pretreatment right frontal EEG asymmetry, were distributed with greater variance in the sample (spread evenly across participants between 10 and 40). However, this explanation is limited by the finding that posttreatment asymmetry was also not significantly associated with posttreatment anxiety symptoms, despite the less restricted range of SPIN scores across the study sample at the completion of therapy. A second possibility is that although both the SPIN and BDI-II are considered valid and reliable measures in so far as they adequately capture participants’ core negative emotional experiences associated with social anxiety and depression, respectively, the SPIN (unlike the BDI-II) might not effectively measure the type of avoidance or withdrawal behavior that is purportedly related to EEG measures of frontal asymmetry (see Coan and Allen, 2004). Indeed, previous studies have also failed to support hypotheses and find significant associations between frontal EEG asymmetry and self-report measures of behavioral inhibition (Coan and Allen, 2003; Harmon-Jones and Allen, 1997). Investigators of these studies have concluded that the relation between EEG asymmetry, withdrawal behavior, and negative affectivity is likely to be multifaceted and complex (see Coan and Allen, 2003).

It is possible that the observed predictive relation between increased relative left hemisphere activity at pretreatment and enhanced CBT response might reflect superior left-sided verbal processing abilities that better support the core verbal therapeutic techniques used in CBT, such as cognitive restructuring (see for example Bruder et al., 1997). Unfortunately, we did not assess patients’ verbal skills in order to test this hypothesis directly, nor is it clear why such effects would be confined to left frontal EEG activity and not extend to central and even parietal areas. An alternative interpretation is the putative involvement of the left orbitofrontal frontal cortex in evaluating the affective salience of stimuli as well as the extinction of conditioned fear responses. Consistent with this suggestion, Brody et al. (1998) reported that patients with OCD who had higher pretreatment levels of activity in their left orbital frontal cortex ultimately responded best to behavioral therapy.

Several limitations of the present study should be noted. First and foremost, the study design did not include a comparison group of clinical or healthy controls, or an alternate treatment or waitlist condition against which the effects observed in patients with SAD who underwent CBT could be evaluated. Previous well-controlled studies have clearly established that individuals with SAD experience significant clinical gains during CBT that are not observed among patients assigned to corresponding waitlist conditions (Ponnhia and Hollon, 2000), and that resting frontal EEG alpha asymmetry in adult clinical samples remain relatively stable over significant time intervals in the absence of treatment (e.g., Allen et al., 2004b; Jetha et al., 2009; Vuga et al., 2006). Nevertheless, because we did not include a control group in our design, we cannot conclude definitively that changes in alpha asymmetry were due to the specific effects of CBT per se. Additional research is required before it is possible to infer causality in the observed relation between the effects of CBT and changes in frontal EEG asymmetry.

Second, as is common in outpatient psychiatric settings, the majority of CBT participants in the present study were also taking medication. As a result, we were unable to disentangle whether changes in EEG asymmetry were associated specifically with the effects of CBT, the effects of medication, or the combined effects of therapy and medication. It is possible that some combination of CBT and the type and/or dosage of medication were related to treatment outcome and/or the link between pre-treatment EEG and treatment response. The literature provides no clear indication of the relative efficacy of CBT plus medication versus CBT alone in the acute treatment of SAD, as findings from clinical trials examining this question have been mixed (see Blanco et al., 2010; Davidson et al., 2004; Haug et al., 2003; Pontoski and Heimberg, 2010). Although well-controlled future studies are needed to determine whether differential changes in frontal EEG asymmetry occur in patients with SAD as a result of “pure” psychological versus pharmacological treatment, it is important to note that findings from studies based on such highly selected samples of patients undergoing tailored treatment protocols may not generalize readily to the types of patients typically treated in most clinical practice settings.

Third, it is important to note that our study was limited by its reliance on a purely subjective measure of medication adherence. Using this measure, a small minority of participants (n = 4) reported that they increased their medication during the study. However, analyses that excluded these participants from the overall sample produced only slight attenuations in the strength of our results, which remained relatively robust and in the same direction as the observed effects when all participants were included in the analyses. The slight attenuation in the strength of our results in the absence of these participants may suggest that the observed changes in frontal asymmetry were driven by the combined therapeutic effects of both medication and CBT, or may simply reflect a decrease in statistical power related to removing four participants from a relatively small sample.

A prospect for future research involves determining the extent to which the present results are driven by characteristics that are unique to social anxiety and CBT per se. An interesting question concerns the extent to which similar effects might be observed in other types of affective psychopathology and in response to other types of interventions specifically designed to target and reduce negative affect and behavioral avoidance. Given the established association between individual differences in frontal EEG asymmetry, on one
hand, and broadly measured indicators of negative versus positive affect and approach–withdrawal behaviors, on the other (Coan and Allen, 2004), it is likely that the pattern of results observed here would generalize from SAD to related emotional disorders that are also characterized by high levels of emotional distress and withdrawal behaviors (e.g., generalized anxiety disorder, depression, etc.), and from CBT to any intervention that is designed specifically to reduce these emotional and behavioral symptoms.

Notwithstanding its limitations and the need for future replication and extension of the preliminary findings, our study suggests that the frontal alpha EEG asymmetry metric may be a useful assessment tool to help clinicians gauge how patients might respond to treatment. While it is too early to conclude that frontal EEG asymmetry is a biological marker of affective psychopathology and has any prognostic value for its treatment, our findings are consistent with the view that the effects of successful treatment for SAD can be measured biologically and that symptom reduction is associated with reliable changes in EEG asymmetry. Future studies are needed to determine the sensitivity and specificity of EEG asymmetry in predicting individual differences in treatment outcome. Such studies would represent a crucial next step toward enhancing our understanding of how EEG asymmetry might be related to symptom changes for individual patients, moving beyond the group-level correlations reported here. With such additional supporting evidence, future innovations in CBT treatment might incorporate EEG asymmetry measures in the routine assessment of emotional disorders, leading potentially to more precise diagnosis and the development of interventions customized to the specific clinical needs and pretreatment symptom profiles of individual patients.

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