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Changes in EEG Cross-Frequency Coupling During Cognitive Behavioral Therapy for Social Anxiety Disorder

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Abstract

Coupling between EEG delta and beta oscillations is enhanced among anxious and healthy individuals during anticipatory anxiety. EEG coupling patterns associated with psychotherapy have not yet been quantified in socially anxious individuals. In this study, we used a double baseline, repeated measures design, in which 25 adults with a principal diagnosis of social anxiety disorder completed 12 weekly sessions of standardized group cognitive behavioral therapy and four EEG assessments: two at pretreatment, one at midtreatment, and one at posttreatment. Treatment was associated with reductions in symptom severity across multiple measures and informants, as well as reductions in delta-beta coupling at rest and during speech anticipation. Moreover, the clinical group exhibited greater coupling at pretreatment than did post hoc control participants with low social anxiety. The EEG cross-frequency profiles in the clinical group normalized by the posttreatment assessment. These findings provide evidence of concomitant improvement in neural and behavioral functioning among socially anxious adults undergoing psychotherapy.

Keywords

emotions, EEG, CBT, cross-frequency coupling, social anxiety disorder, psychopathology

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Studies examining the neural correlates of psychological treatment are far outnumbered by studies investigating the neural correlates of pharmacological interventions, despite evidence that cognitive behavioral therapy (CBT) and medications have equal short-term clinical efficacy for the treatment of anxiety (Otto, Behar, Smits, & Hofmann, 2009), with psychotherapy being more cost-effective over the long term (Heuzenroeder et al., 2004). A promising line of research involves tracking brain activity before and after the delivery of evidence-based psychological treatments that are standardized and proven to reduce symptoms. One example of such a treatment is CBT, a highly structured and collaborative form of psychotherapy that helps patients identify and modify the maladaptive, interactive patterns of thoughts and behaviors underlying their emotional dysfunction (Barlow, 2008).

Currently, the majority of research on brain changes in response to psychotherapy consists of nuclear and magnetic imaging studies involving comparison activations before and after treatment (Roffman, Marci, Glick, Dougherty, & Rauch, 2005). Rather than producing regionally isolated alterations, psychotherapeutic interventions seem to produce changes in the dynamic interactions between cortical and subcortical brain regions. For example, psychotherapy for obsessive-compulsive disorder reduces correlated activity in cortico-striatal-thalamic circuits (Schwartz, Stoessel, Baxter, Martin, & Phelps, 1996), and depression treatment affects the balance of fronto-limbic activity (Goldapple et al., 2004).

Compared with studies using functional MRI and PET scans, studies quantifying psychotherapy-related electrocortical changes are rare (for exceptions, see Leutgeb, Schafer, &...
Schienle, 2009; Oathes et al., 2008; Rabe, Zoellner, Beauducel, Maercker, & Karl, 2008). Brain electrical measures (EEGs) reveal the oscillatory characteristics of neuronal mass activity, and unlike blood-oxygenation levels (Attwell & Iadecola, 2002), they provide a direct index of synchronous dendritic potentials in real time. Accordingly, EEG measures may be uniquely suited to examining the interactive brain dynamics that are assumed to accompany successful clinical treatments.

The macroscopic brain oscillations revealed by scalp-recorded EEG measures cover a broad frequency spectrum, ranging from slow wave (SW; delta and theta) to fast wave (FW; beta and gamma) activities. It has been suggested that the frequency of oscillations reflects recirculation time in neuronal pathway loops (Bressler & Tognoli, 2006), such that SW oscillations integrate neural processes spanning large distances, and FW electrical signatures relate to synchronization of topographically restricted regions. Several independent lines of evidence from human and animal studies support the suggestion that subcortical structures play a stronger role in SW generation, and unlike blood-oxygenation levels (Attwell & Iadecola, 2002), they provide a direct index of synchronous dendritic potentials in real time. Accordingly, EEG measures may be uniquely suited to examining the interactive brain dynamics that are assumed to accompany successful clinical treatments.

Patterns of delta-beta coupling in the clinically diagnosed prevalent anxiety disorder (Kessler, Chiu, Demler, & Walters, 2005) that is marked by excessive fears of interpersonal situations in which people feel they may be harshly evaluated. Previous work has shown that the anticipation of public speaking produced significant increases of prefrontal delta-beta coupling in a nonclinical sample of adults with high social anxiety (Miskovic et al., 2010). In the study reported here, we were interested in extending these findings to a clinical sample by examining whether psychotherapeutic treatment of SAD led to concomitant decreases in delta-beta coupling. Discovering a reliable neural correlate of treatment may potentially provide novel insights into the psychophysiology of social anxiety.

In this study, we measured regional EEG activity at rest and prior to an anticipated public speech in a sample of 25 adults diagnosed with SAD (generalized subtype). EEG measures were collected at four visits (two pretreatment, one midtreatment, and one posttreatment) in a double baseline, repeated measures design, in which participants served as their own controls. Treatment consisted of 12 standardized group CBT sessions. Additionally, the EEG profiles of the clinical group at pretreatment and posttreatment were compared with the EEG profiles of a nonclinical sample of adults with high and low social anxiety (Miskovic et al., 2010).

We hypothesized that measures of cross-frequency power coupling, which may index cortico-subcortical dynamics not immediately available in functional neuroimaging (Schutter et al., 2006), would be sensitive to treatment-related reductions in clinical symptomatology. On the basis of prior studies, we expected reductions in correlated delta-beta power to appear largely in electrodes overlying the prefrontal cortex. Patterns of delta-beta coupling in the clinically diagnosed SAD participants were expected to be similar to patterns in the post hoc control group with high social anxiety at the pretreatment assessment and analogous to patterns in the group with low social anxiety by posttreatment.

**Method**

**Participants**

Thirty-three outpatients from a large anxiety-treatment clinic at an urban hospital in Hamilton, Ontario, were recruited for the study. All participants were diagnosed with principal SAD (generalized subtype) by trained clinicians using criteria from the fourth edition of the Structured Clinical Interview for DSM-IV-TR Axis I Disorders—Patient Edition (First, Spitzer, Gibbon, & Williams, 2001). Because of attrition, the final sample size was 25 (12 females, 13 males; see Fig. 1). Clinical severity ratings (CSRs) were assigned on a scale from 0 to 8, with CSRs of 4 and above representing an increasing level of clinically significant interference and distress associated with the principal diagnosis. The CSRs for SAD diagnoses in the study sample ranged from 4 to 7 ($M = 5.54, SD = 0.90$). All participants were Caucasian, right-handed, and ranged in age from 19 to 73 years ($M = 35.9$ years, $SD = 15.18$).
Figure 1 summarizes the study design (see also Fig. S1 in the Supplemental Material available online). Participants were requested to maintain the type and dosage of their medication throughout the study. Seven patients reported changes in either medication type or dosage during the study.

Exclusion criteria for the study consisted of current mania, psychosis, significant suicidality, and organic brain disorders. Individuals with current substance abuse or dependence were excluded if they did not agree to refrain from use prior to and during treatment and experimental protocols, were deemed by the clinical team to be unsuitable for group CBT targeting SAD, or required initial treatment for substance use. Participants who self-reported consuming beta blockers within 3 days of the EEG testing and alcohol, marijuana, or antihistamines within 12 hours of EEG testing were also excluded.

**Clinical treatment procedures**

All of the participants completed 12 weekly, 2-hr sessions of group CBT for SAD at the Anxiety Treatment and Research Centre, in St. Joseph’s Healthcare Hamilton. The structure for the group CBT sessions was based on a standardized protocol (Antony & Swinson, 2009). Core components of group CBT included psychoeducation, cognitive restructuring, in-session and between-session exposure exercises, and social-skills training. Group CBT sessions were administered by 2 or 3 qualified therapists with 7 to 9 patients per group. Sessions were administered in consecutive weeks, with a 1-week break between the sixth and seventh sessions to allow for midtreatment assessments.

**Experimental procedures**

EEG testing prior to a standardized speech task was administered in the laboratory twice at pretreatment, once at midtreatment, and once at posttreatment. Procedures were explained to participants, and they provided written informed consent for the psychophysiological portion of the study. A different set of speech topics was generated for each of the four EEG laboratory assessments, but otherwise procedures remained identical. Following EEG application, participants were given several minutes to acclimate to the testing environment. Participants’ EEG measures were then collected continuously during 6 min of rest, which consisted of three eyes-open and eyes-closed epochs alternating for 1 min each. Following the resting recording, participants completed state-anxiety measures for the second time. Next, participants were provided with a set of three predetermined speech topics on controversial issues (e.g., capital punishment, same-sex marriage, funding of religious schools) and performed the public-speaking task on any one or more of these topics for a maximum of 3 min. Each participant’s posttreatment assessment was scheduled to occur within a window of approximately 2 weeks following the final (12th) group CBT session. All laboratory procedures were conducted under the supervision of trained research staff and approved by the Hamilton Health Sciences Ethics Committee.

**Clinical and self-report measures**

We administered several measures to track treatment efficacy and obtain measures of state anxiety during the EEG recording.
visits. Independent, trained clinicians blind to the study purpose and participants’ treatment status, as well as one of the group therapists, rated each participants’ illness severity on the Clinical Global Impression (CGI) scale (Guy, 1976) at pretreatment and posttreatment. Previous work has established the validity of the CGI in evaluating treatment efficacy for SAD (Zaider, Heimberg, Fresco, Schneier, & Liebowitz, 2003). Participants also completed the following self-report scales: the Illness Intrusiveness Ratings Scale (IIRS; Devins, 1994), a 13-item questionnaire measuring the extent to which an illness, its treatment, or both interfere with daily life; the SPIN (Connor et al., 2000), a 17-item inventory measuring symptoms of social fear, avoidance, and arousal; the self-report version of the Liebowitz Social Anxiety Scale (LSAS-SR; Baker, Heinrichs, Kim, & Hofmann, 2002), a 24-item measure that assesses fear and avoidance in a broad range of social interactions; and the second edition of the Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996). Participants’ state anxiety during the speech at each EEG visit was measured using the Subjective Units of Distress Scale (SUDS; Wolpe & Lazarus, 1966), which ranges from 0 (no anxiety) to 100 (highest possible level of anxiety), both before and following the speech anticipation.4

**Results**

**Treatment efficacy**

We conducted separate repeated measures ANOVAs, using time (pretreatment, posttreatment) as the within-subjects factor on both the clinician-rated measure (CGI illness severity) and patient-rated measures (IIRS, SPIN, LSAS-SR, and BDI-II). As expected, there was a main effect of time (ps < .02) across all of the dependent variables, as assessed by clinicians and patients (see Table 1).

**Findings for EEG cross-frequency coupling**

To study the effects of group CBT on brain electrical activity, we computed a set of Pearson correlation coefficients (r) between ln delta and ln beta band power separately for each electrode at each laboratory assessment and for each experimental condition. We used Fisher’s r-to-Z transformation to normalize the distribution of correlation coefficients and then performed a Steiger test for dependent comparisons. Because preliminary analyses indicated no significant differences in delta-beta coupling magnitude between the two pretreatment visits (ps > .13), these data were averaged into a common pretreatment measure to reduce the number of comparisons. In addition to analyzing the electrode sites overlying the prefrontal cortex (F3 and F4), we performed exploratory analyses incorporating the central, parietal, and occipital leads (see Fig. 2). Two-tailed p values were used for all comparisons.

**Table 1. Mean Ratings for Multiple-Symptom Severity Measures**

<table>
<thead>
<tr>
<th>Scale</th>
<th>Pre-treatment</th>
<th>Post-treatment</th>
<th>p</th>
<th>(\eta^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinician rated:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CGI illness severity</td>
<td>5.00 (0.17)</td>
<td>4.29 (0.26)</td>
<td>&lt; .001</td>
<td>.47</td>
</tr>
<tr>
<td>(independent clinician)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CGI illness severity</td>
<td>5.28 (0.17)</td>
<td>3.76 (0.23)</td>
<td>&lt; .001</td>
<td>.64</td>
</tr>
<tr>
<td>(group therapist)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Patient rated:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IIRS total score</td>
<td>59.48 (2.70)</td>
<td>51.33 (4.12)</td>
<td>.017</td>
<td>.25</td>
</tr>
<tr>
<td>SPIN total score</td>
<td>46.92 (2.50)</td>
<td>31.83 (3.21)</td>
<td>&lt; .001</td>
<td>.59</td>
</tr>
<tr>
<td>LSAS-SR total score</td>
<td>97.42 (4.65)</td>
<td>67.13 (6.56)</td>
<td>&lt; .001</td>
<td>.54</td>
</tr>
<tr>
<td>BDI-II total score</td>
<td>23.96 (1.89)</td>
<td>17.42 (2.37)</td>
<td>.003</td>
<td>.33</td>
</tr>
</tbody>
</table>

Note: Standard errors are given in parentheses. BDI-II = second edition of the Beck Depression Inventory (Beck, Steer, & Brown, 1996); CGI = Clinical Global Impression (Guy, 1976); IIRS = Illness Intrusiveness Ratings Scale (Devins, 1994); LSAS-SR = self-report version of the Liebowitz Social Anxiety Scale (Baker, Heinrichs, Kim, & Hofmann, 2002); SPIN = Social Phobia Inventory (Connor et al., 2000).
Fig. 2. Strength of delta-beta correlations ($r$) collapsed across left- and right-hemisphere electrodes within the four tested regions at each measurement occasion. Results are shown separately for (a) resting conditions and (b) speech-anticipation conditions.
Resting. In the resting condition, there was a significant decrease in delta-beta coupling from pretreatment to midtreatment, both in the left midfrontal electrode (Z = 2.64, p = .008) and in the right midfrontal electrode (Z = 2.12, p = .03). The comparison between pretreatment and posttreatment likewise revealed a significant reduction in delta-beta coupling bilaterally (F3: Z = 2.74, p = .006; F4: Z = 2.65, p = .008). By contrast, there was no difference in coupling from midtreatment to posttreatment in either of the frontal electrodes (ps > .66).

Extended analyses indicated that reductions in delta-beta coupling from pretreatment to midtreatment during the resting condition also reached significance at other electrode locations (C4, P4, and O2; ps < .03). Likewise, the comparison from pretreatment to posttreatment revealed reductions in delta-beta correlations across all of the regions in the left hemisphere (C3, P3, and O1; ps < .03). There were no differences in coupling from midtreatment to posttreatment (ps > .33). Although the findings lacked regional specificity, the strongest differences were generally located in electrodes overlying the frontocentral cortex, as illustrated in Figure 3.

Speech anticipation. In the speech-anticipation condition, there was no decrease in frontal delta-beta coupling from pretreatment to midtreatment (ps > .10). As expected, there was a decrease in delta-beta coupling from pre- to posttreatment (F3: Z = 3.35, p = .001; F4: Z = 3.04, p = .002). There were no significant changes in frontal oscillatory coupling from midtreatment to posttreatment.

Extended analyses showed that the coupling changes were not specific to the frontal region. Delta-beta coupling was reduced from pretreatment to midtreatment in all of the central, parietal, and occipital electrodes (ps < .02), except for O2. Similarly, coupling was reduced (ps < .04) at all of the recording sites from pretreatment to posttreatment. There were no differences from midtreatment to posttreatment (ps > .25). Overall, the strongest treatment-related differences emerged for electrodes covering the frontocentral cortex (Fig. 3).

Medication confounds. It is important to note that all of the results reported remained significant following the exclusion of 7 patients who did not maintain their medication type or dosage over the course of the study.5

Findings for EEG spectral power

To test for treatment-related changes in individual frequency band power, we ran separate repeated measures ANOVAs for delta and beta bands, using region (frontal, central, parietal, occipital), hemisphere (left, right), condition (resting, speech anticipation), and time (pretreatment, midtreatment, posttreatment) as within-subjects factors.

In contrast to the coupling patterns, analyses revealed no main or interaction effects involving time for delta spectral power (ps > .11) or beta spectral power (ps > .23). Main or interaction effects involving other independent variables are not reported here because they do not directly inform treatment-related hypotheses.

Post hoc control comparisons

Post hoc control comparisons were confined to the midfrontal electrodes (F3 and F4) because our previous study indicated that there was significant delta-beta coupling solely for these sites in a speech-anticipation task (Miskovic et al., 2010; see Fig. 4).

Pretreatment. The clinical group at pretreatment had greater delta-beta coupling in the right midfrontal electrode during speech anticipation than did the group with low social anxiety (p < .01). There were no other significant between-group comparisons.

Posttreatment. There was a trend for the clinical group at posttreatment to exhibit diminished delta-beta coupling relative to the group with high social anxiety in the right midfrontal electrode (p = .06). By posttreatment, the clinical group and the group with low social anxiety did not differ in delta-beta coupling magnitude (ps > .37).

Discussion

The findings reported here indicate that effective group CBT for SAD was associated with changes in brain electrical activity and suggest a potential neural correlate of psychotherapy. Reductions in correlated delta-beta EEG spectral power (i.e., cross-frequency coupling) were observed during both resting and speech-anticipation conditions and were detectable by the midtreatment assessment, although the strongest differences emerged for the pretreatment to posttreatment comparisons. It is interesting that at pretreatment, the clinical group showed increased delta-beta coupling in the right midfrontal electrode compared with a post hoc control group with low social anxiety. However, there were no significant differences in coupling between these two groups when the clinical group was assessed at posttreatment, and this suggests that brain electrical activity was normalized in patients following the completion of therapy.

Reductions in delta-beta coupling following treatment for SAD appear to reflect a brain spectral profile associated with diminished anxiety, an interpretation that is in agreement with therapists’ and patients’ perceptions of improvement. Because there were no changes in delta and beta spectral power independently of one another, it is possible that CBT may have specifically altered the degree of temporally coherent SW-FW energy distribution. Previous work has suggested that correlated activity in delta and beta bands may be an electrophysiological index of cortico-subcortical interactions (Schutter et al., 2006; Velikova et al., 2010). A plausible argument is that the decreased information transfer (reflected in low delta-beta correlations) is associated with reduced bottom-up...
transmission of threat-related signals conveyed by subcortical regions to the neocortex (e.g., van Honk & Schutter, 2007). Although this interpretation remains highly speculative, it provides a heuristic framework that is generally convergent with previous metabolic imaging findings suggesting that psychotherapy for mood disorders and anxiety disorders is associated with changes in reciprocal cortico-subcortical dynamics rather than with regionally isolated changes (Goldapple et al., 2004; Schwartz et al., 1996).

It is worth emphasizing that significant EEG changes were evident by the midtreatment assessment in the present study. Previous neuroimaging investigations of brain changes during psychotherapy have relied on pretreatment versus posttreatment comparisons only (Roffman et al., 2005). Demonstrating changes in brain physiology by midtreatment may carry interesting implications for the clinical concept of sudden gains, in which considerable symptom improvements occur for some patients in the initial portions of therapy (Hofmann, Schulz, Meuret, Moscovitch, & Suvak, 2006).

The present study had some limitations that preclude causal generalizations. A major limitation was the lack of waiting-list or healthy control groups that were assessed at multiple time points. Previous randomized control studies have clearly established that CBT for SAD is associated with clinical improvements that are not observed among waiting-list control participants (Ponniah & Hollon, 2008), and ethical review boards at treatment institutions have begun to discourage
investigators from delaying treatment to patients in need by assigning them to waiting-list control conditions. Thus, rather than use waiting-list control participants, we utilized a double-baseline approach that involved two pretreatment visits in addition to post hoc comparisons of our clinical group with two distinct nonclinical groups of adults with high and low social anxiety. It is notable that there were no significant differences in coupling strength between the two pretreatment visits; this argues against a simple interpretation of nonspecific reductions in coupling strength or habituation effects. Moreover, EEG differences between the clinical group and post hoc control groups were consistent with the notion that treatment-related changes in spectral correlations reflected a measure of neural activity that was normalized by intervention. However, to attribute the changes in oscillatory coupling strength specifically to treatment, future studies also need to examine the long-term stability of EEG spectral profiles in SAD adults randomly assigned to a control condition in which they do not undergo treatment.

A second limitation is that, for some participants, psychotherapeutic and medication treatments were combined. Because some patients were also treated with medication, it is questionable whether the results may be interpreted as a consequence of psychotherapy. Rather than exclude these treatment-seeking patients from the study, we asked participants to keep their medication type and dosage stable throughout the study. It is important to note that the EEG results remained significant after excluding individuals who reported changes in medication regimen. In the future, it may be instructive to move from quantification of treatment-related brain changes to randomized controlled trials testing the active ingredients of psychotherapy that are necessary and sufficient for mediating changes in brain function. Such knowledge would assist in designing more effective forms of psychological treatment for specific disorders.

Overall, this study suggests that predictable changes in mass activity of the brain accompany completion of CBT for SAD, and that these changes parallel improvements resulting from clinical intervention. There is both theoretical and practical value to discovering neural correlates associated with clinical symptom reduction. Theoretically, these findings are informative with regard to the sorts of distributed patterns of brain activity that may underlie the presentation of social anxiety as well as their capacity for environmental modification. At a practical level, identifying neural correlates of psychotherapy may suggest possible treatment markers and may have inherent value as a treatment tool in itself through the development of neurofeedback techniques.

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Declaration of Conflicting Interests
The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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Supplemental Material
Additional supporting information may be found at http://pss.sagepub.com/content/by/supplemental-data

Notes
1. The considerable design differences among these studies include the type of therapy, the extent to which comparisons were primarily within groups or between groups (employing waiting-list or healthy control groups), and the nature of the neural signals recorded.
2. For brevity, the term cross-frequency coupling is used throughout this article to denote the zero-order correlation of EEG power between two frequency bands. For all of the delta-beta coupling measures in this study, we quantified coupling across participants. An alternate use of the term relates to estimates of the strength of phase or power synchronization between two frequency bands within individual participants.
3. Generalized subtype indicates that anxiety is prevalent in most social situations, rather than being restricted to very specific contexts.
4. To ensure that the speech-anticipation task was experienced as stressful, we ran four separate repeated measures ANOVAs on SUDS scores. There was a main effect of condition (resting, speech anticipation; ps < .001). Overall, participants provided higher SUDS ratings (i.e., reported more anxiety) during the speech-anticipation condition (grand M = 60.97, SD = 9.03) than during the resting condition (grand M = 44.05, SD = 5.14).
5. Planned analyses were also performed for the small subset of patients (n = 8) who were medication free, but these analyses were restricted to the midfrontal electrodes. There was a trend (p < .07) for reduced right midfrontal (F4) delta-beta coupling from pretreatment to posttreatment in the speech-anticipation condition only. There were no other significant or trend-level effects for the resting condition or the left midfrontal (F3) site. However, the medication-free subgroup reported significantly less severe social anxiety on the SPIN and had lower clinician-rated illness severity ratings (p < .06) than did patients who took medication. These results held even at pretreatment, and this indicated that the medication-free subgroup was overall a less clinically impaired sample than the medicated subgroup.

References


