Does respiratory sinus arrhythmia (RSA) predict anxiety reduction during cognitive behavioral therapy (CBT) for social anxiety disorder (SAD)?

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Abstract

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Modifying dysfunctional emotion regulation is an important goal in psychological treatments for social anxiety disorder (SAD). Antecedent-focused strategies learned in cognitive behavioral therapy (CBT), such as cognitive reappraisal, have proven more effective in reducing social anxiety than response-focused strategies, such as expressive suppression. Still, not all patients with SAD respond well to CBT. Medications and physiological factors may also influence the clinical response. The purpose of the present study was to examine the role that these factors play in determining treatment response following CBT for SAD. Using multilevel modeling, we examined associations across four separate laboratory visits between change in self-reported anxiety and indices of reappraisal, suppression, medication status, and resting respiratory sinus arrhythmia (RSA), a proxy measure of self-regulatory capacity, in 23 socially anxious adults during a 12-week program of CBT. Most participants were ultimately classified as responders to CBT (n = 15), but in some, anxiety levels remained unchanged (n = 8). Medication use explained substantial variance related to individual differences in anxiety among participants. When modeled separately, reappraisal, suppression, and RSA each accounted for significant variance related to anxiety. However, the best-fitting model included reappraisal and RSA. Moreover, RSA reactivity (change in RSA levels over time) was more important for predicting anxiety reduction than were baseline levels of RSA. These findings suggest that reappraisal and parasympathetic responsiveness may be important in reducing anxiety in adults with SAD who respond well to CBT.

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1. Introduction

Social anxiety disorder (SAD) is a common psychological disorder, with an estimated lifetime prevalence of about 12% (Kessler et al., 2005). Its core features include difficulty in regulating negative emotion (Kashdan, 2007; Kashdan et al., 2006) and deficient experience of positive emotion (Hofmann et al., 2012; Kashdan et al., 2006). In SAD, strong fears of negative evaluation can lead to high anxiety, avoidance of social interactions, and suppression of the expression of behaviors that might lead to embarrassment or social rejection (Moscovitch, 2009; Spokas et al., 2009; for a review, see Aldao et al., 2010). A critical goal for psychological treatment of SAD is to help individuals learn to regulate negative emotions such as anxiety more effectively. Despite the general efficacy of treatments such as cognitive behavioral therapy (CBT) in ameliorating emotional distress (Smits and Hofmann, 2009), many individuals with SAD who receive CBT do not experience significantly improved social and emotional functioning (e.g., Moscovitch et al., 2012). Understanding the variables that influence treatment response remains a major objective for researchers and clinicians alike.

1.1. Cognitive strategies for coping with SAD

Specific techniques have been developed to help patients learn to manage strong emotions in everyday situations. In one such strategy –

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cognitive reappraisal – participants learn to construe anxiety-provoking situations in a less negative way, in order to circumvent the development of anxious feelings before they become established (Gross, 1998). Because of its pre-emptive nature, cognitive reappraisal is an example of an antecedent-focused regulation strategy. Changing one’s interpretation of anxiety-provoking situations “up front” alters the course of the entire emotional response, thereby reducing the subjective experience of distress (Goldin et al., 2009; Gross, 1998).

In a contrasting strategy – expressive suppression – participants inhibit the behavioral responses precipitated by strong emotions after the emotional reaction has already been initiated. While this approach is intended to minimize the likelihood of rejection by others and is often a strategy that is favored by patients before they are exposed to therapy, suppression is likely to enhance, rather than reduce, subjective and physiological responses associated with distress (Campbell-Sills et al., 2006; Gross and John, 2003). Not surprisingly, suppression has been associated with poor social functioning and well-being, whereas the reverse is true for reappraisal (Gross and John, 2003). In addition, the use of suppression has been shown to increase sympathetic arousal, blood pressure, and stress in suppressors and also their social partners (e.g., Butler et al., 2003; Campbell-Sills et al., 2006; Harris, 2001; Hofmann et al., 2009; Moore et al., 2008). Although suppression may be expedient when time is short (Gross and John, 2003) or high-level performance is at stake (Richards and Gross, 2000), over the long term, suppressors are likely to incur significant emotional, social, and physiological costs.

1.2. Parasympathetic regulation and anxiety

The contribution of physiological variables to treatment response in clinical cohorts is a relatively understudied but growing area of research. Physiological measures may provide objective indices of affective and self-regulatory responses that potentially corroborate and augment the data gleaned from self-reports. One index that may have important implications for self-regulation and social behavior is high-frequency heart rate variability (HF-HRV), or its natural log equivalent, respiratory sinus arrhythmia (RSA). Both measures are commonly used to index phasic, vagal cardiac control and account for a large proportion of the variability in heart rate (Bernston et al., 2007; Grossman and Taylor, 2007; Porges, 1995).

The intrinsic heart rate (the rate at which the heart beats when it is not under autonomic control) is faster than the resting heart rate. During rest, heart rate is tonically inhibited by actions of the parasympathetic nervous system (Levy, 1990). Parasympathetic effects occur on a time scale of milliseconds, whereas sympathetic effects are an order of magnitude slower; as such, the parasympathetic system is responsible for instantiating moment-by-moment cardiac responses to changing environmental contingencies. The ability to spontaneously disengage and re-engage the “vagal brake” (Porges, 2007), reflected in a) high resting HF-HRV or b) greater vagal reactivity to challenges, has long been linked to behavioral flexibility and adaptive functioning (e.g., Fabels and Eisenberg, 1997; Thayer and Lane, 2000). Higher resting HF-HRV indexes not only more robust cardiovascular health (DeMeersman and Stein, 2007; Liao et al., 1996), but also more efficient cardiovascular adaptation to the challenges of daily life (Park et al., 2007), including greater self-regulatory behavior (Thayer and Lane, 2000), emotional regulation (e.g., Brosschot et al., 2007; Geisler et al., 2010), and attentional control (e.g., Hansen et al., 2003; Mathewson et al., 2010; Porges, 1992; see Thayer and Brosschot, 2005; Thayer et al., 2009 for reviews). Conversely, lower parasympathetic control has been associated with less adaptive emotion regulation (e.g., Friedman and Thayer, 1998; Rechlin, 1994; Udupa et al., 2007).

In typical adults, higher resting HF-HRV has been linked to greater spontaneous use of reappraisal for managing negative emotions (Volokhov and Demaree, 2010). However, when covariation among limbic regions that normally act in concert to process emotional information is decreased, the consequences appear to include lower levels of HRV, increased sympathetic activation, and higher trait anxiety (Mujica-Parodi et al., 2009). Comparable findings have been demonstrated in groups sensitive to social rejection (e.g., Ahls et al., 2009; Friedman and Thayer, 1998; Gyurak and Ayduk, 2008). Thus, HF-HRV may be lower in anxious adults than their non-anxious peers (e.g., Brosschot et al., 2006; Friedman and Thayer, 1998).

1.3. Influence of medications

Psychotropic medications are effective for reducing anxiety, but they are also known to affect autonomic functioning (e.g., heart rate and blood pressure; O’Brien and Oyebode, 2003), with specific, unfavorable effects on parasympathetic activity (Licht et al., 2009, 2010; Roelofs and van der Bijl, 1994). As anxiety-lowering medications may also influence cognitive operations related to learning, memory or executive control, their use may have important implications for individuals with social anxiety who are learning to reappraise difficult social situations. About two-thirds of the socially anxious participants in this study were prescribed psychotropic medications to regulate anxiety and/or depression. Therefore, we examined the influence of stable medication use on anxiety during CBT, in conjunction with parasympathetic regulatory capacity.

1.4. The present study

The primary purpose of this study was to investigate whether RSA would aid in predicting the clinical response of adults with SAD to CBT, both in its own right and in interaction with emotion regulation strategies and/or pharmacological treatment. Specifically, we used multilevel modeling to examine whether reappraisal, suppression, medication status, or RSA (or combinations of these variables, described below) best predicted pretreatment anxiety levels and change in anxiety symptoms over time in 23 treatment-seeking adults with a principal diagnosis of SAD. Self-reports of anxiety, emotion regulation strategies, and resting RSA derived from ECG recordings were collected on four occasions: at two pretreatment sessions, midtreatment, and posttreatment. Among psychotropic medication users, type and dosage of medication were held constant for the duration of the study; thus, the presence or absence of medication use was treated as a covariate.

In this particular cohort, using reappraisal uniquely distinguished those who ultimately responded well to therapy (responders) from those who did not (nonresponders), as reported elsewhere by Moscovitch et al. (2012). As an effective strategy for managing anxiety, cognitive reappraisal was expected to predict lower levels of anxiety over time. Because suppression tends to enhance subjective distress, suppression was expected to predict higher levels of anxiety over time. Specifically, we anticipated that cognitive reappraisal and suppression would each explain significant variance in anxiety during the study, but in opposite directions. We also anticipated that resting RSA, reflecting an individual’s capacity for physiological self-regulation, would explain significant variance in anxiety. Reappraisal and RSA were likely to have similar effects on anxiety, whereas the effects of suppression and RSA were likely to be dissimilar. Therefore, we predicted that the combination of reappraisal and RSA would explain more variance in anxiety levels than would the combination of suppression and RSA.

2. Method

2.1. Participants

Individuals seeking outpatient treatment at the Anxiety Treatment and Research Centre (ATRC) in a large urban healthcare center in Hamilton, Ontario, Canada, were assessed by graduate-level clinicians using the Structured Clinical Interview for the Diagnostic and Statistical Manual
of Mental Disorders—4th Edition (SCID, First et al., 1996). Diagnoses were confirmed during weekly consensus case conference meetings, chaired by a psychologist with over 10 years of experience in SCID administration and training. Individuals with a principal diagnosis of SAD were invited to participate in the study, provided they did not have active psychosis, mania, comorbid substance dependence or abuse, were not suicidal, and were willing to maintain the same medication type and dose for the entire duration of the study, beginning 1 month prior to their first laboratory assessment until after their final testing session. As participants were recruited from a psychiatric clinic situated within a hospital, 64% of the sample was taking psychotropic medications (reported in Moscovitch et al., 2012) to regulate anxiety and depression. Other exclusion criteria included taking beta-blockers 3 days prior to testing, as well as the use of alcohol, marijuana, or antihistamines within 12 h of the testing sessions. Participants were required to report their medication adherence in writing at each testing and treatment session.

Initially, 33 outpatients (18 males, M = 36.3 years, SD = 15.1; 15 females, M = 33.4 years, SD = 11.2) agreed to participate. Eight discontinued their participation during the study for the following reasons: personal reasons (n = 5), time constraints (n = 1), did not attend posttreatment assessment (n = 1), or missed more than four treatment sessions (n = 1). Data from two additional participants were also excluded because the medication dosage for one person was changed (augmented) during the study, and one significantly older participant’s cardiac data were unreliable. Thus, data from 23 participants (11 males, M = 32.8 years, SD = 11.9; 12 females, M = 33.3 years, SD = 12.3) were analyzed.

All 23 participants received a principal (i.e., most interfering or distressing) DSM-IV diagnosis of SAD. Nineteen (83%) participants received at least one current comorbid diagnosis, with major depressive disorder (single episode or recurrent; n = 9) and generalized anxiety disorder (n = 6) as the two most common secondary diagnoses. Other secondary diagnoses included obsessive–compulsive disorder (n = 1), bipolar disorder (n = 1), PTSD (n = 1), and substance abuse (n = 1).

2.2. Procedure

Participants were introduced to the laboratory and briefed about procedures before they provided written consent. Three self-report measures were administered individually on four occasions: 2 weeks and 1 week before group therapy commenced (M = 7.7 days apart), at the treatment midpoint (within 1 week of completing week 6), and at the end of therapy (within 2 weeks of completing week 12). At each visit, 6 min of regional electroencephalogram (EEG) and electrocardiogram (ECG) were continuously recorded via a lyca stretch cap (Electro-cap, Inc., Eaton, OH) and two disposable ECG electrodes placed on the medial forearms, while the participant sat quietly in a comfortable chair. Each participant then prepared and delivered a short, video-taped speech, forearms, while the participant sat quietly in a comfortable chair. Each participant then prepared and delivered a short, video-taped speech, with different speech topics assigned at each visit to eliminate practice effects. The EEG data were collected as part of a larger study (reported in Miskovic et al., 2011). Participants received $20 (Canadian dollars) in remuneration at the end of each visit. All laboratory procedures were approved by the participating university and hospital ethics committees.

2.3. Self-report measures

Participants completed the Social Phobia Inventory (SPIN, Connor et al., 2000), a 17-item self-report instrument measuring symptoms of social anxiety (social inadequacy, low self-esteem, fear of losing bodily control, feelings of social inferiority, and fear of being the center of attention). The SPIN constituted the primary clinical measure during treatment and has demonstrated good internal consistency (SPIN, α = .82 to .94, Connor et al., 2000). In the present study, internal consistency was strong (α = .86 to .95) across the four testing sessions.

Patients also completed the Emotion Regulation Questionnaire (ERQ, Gross and John, 2003), a 10-item instrument assessing customary use of two contrasting emotion regulation strategies. Six items indexed reappraisal (e.g., I control my emotions by changing the way I think about the situation I’m in) and four items indexed suppression (e.g., I control my emotions by not expressing them). Both subscales assessed regulation of positive as well as negative emotions and have demonstrated adequate internal consistency (reappraisal: α = .75 to .82; suppression: α = .68 to .75; Gross and John, 2003). In the present study, internal consistency was good for reappraisal at each of the four visits (α = .83 to .85), and modest but satisfactory for suppression (α = .57 to .67), except at Visit 2 (α = .39).

2.4. Physiological index

Measures of resting RSA are reported to be stable in adults with SAD (Schmidt et al., 2012) and typically developing children (El-Sheikh, 2005). In the present study, the internal consistency for RSA across four measurement occasions was strong (Cronbach’s α = .93). Therefore, we used resting RSA as a trait-like measure representing characteristic individual differences in the capacity for flexible, parasympathetic regulation of heart rate.

ECG signals were amplified by an individual SA Instrumentation Bioparameter, filtered between 0.1 Hz (high pass) and 1000 Hz (low pass), and digitized at a sampling rate of 512 Hz. Signals were visually edited for artifact (missing or spurious R-waves) using software developed by the James Long Company (BI Analysis Program; Caroga Lake, NY). A file of interbeat intervals collected during the resting condition was created for each participant. Heart period data were detrended using a high-pass filter with a period of 10 s. A discrete Fourier transform analysis, with a 32-second Hanning window and 50% consecutive overlap, was applied to quantify the amount of high-frequency variability (ms²) in the signal (0.12 to 0.40 Hz) (Bernston et al., 1997). High-frequency power values underwent a natural log transformation to normalize the distribution, yielding estimates of RSA (Task Force, 1996).

In the present study, respiration rate was not covaried from our measure of RSA. While statistical control of respiration is one way to ensure that RSA measures are not influenced by respiration rate (e.g., Grossman and Taylor, 2007), during seated rest, natural respiration rates are slow enough that they are unlikely to significantly affect RSA (Berntson et al., 1997; Denver et al., 2007).

2.5. Group CBT treatment

Treatment consisted of weekly 2-hour sessions of CBT for 12 consecutive weeks delivered in a group setting (7–9 patients per group), with a 1-week break between sessions 6 and 7 to allow for midtreatment assessments. Sessions were led by a senior clinician with extensive experience in administering CBT, and conducted by 2–3 trained clinicians using a standardized, evidence-based treatment manual (Antony and Swinson, 2008). Components included psychoeducation, cognitive restructuring, exposure exercises, social skills training, and skills consolidation. All clinicians had graduate or postprofessional training in CBT. The costs of participants’ treatments were covered by provincial health coverage, available to all permanent residents of Ontario.

2.6. Reliable Change Index of treatment response

Moscovitch et al. (2012) reported that not all participants with SAD showed significant improvement as a result of the therapeutic intervention. Participants who were deemed to be either recovered or significantly improved were considered treatment responders, according to the Reliable Change Index (RCI, Jacobson and Truax, 1991), calculated for each participant from test–retest reliability data on the SPIN (Antony et al., 2006). On completion of treatment, four male participants reached
a posttreatment absolute SPIN score that was below the cut-off for clinical significance (22.5, calculated according to Formula A of Jacobson and Truax, 1991) and were considered recovered. Eleven more participants (8 female) showed a reliable RCI (pre- to posttreatment change) in SPIN scores and were considered significantly improved (see Moscovitch et al., 2012). SPIN scores for the remaining eight participants (4 female) were either unchanged or deteriorated (nonresponders). Gender was evenly represented among responders and nonresponders ($\chi^2 = .023, \text{ns}$).

2.7. Data analyses

As an initial step, change in the self-report and autonomic measures was examined in 2 (group) by 4 (visit) mixed model ANOVAs, followed by Bonferroni-corrected pair-wise tests.

Then, exploratory correlational analyses of anxiety scores with reappraisal, suppression, or RSA were carried out to examine simple relations between predictors and anxiety. Separate correlations were carried out for responders versus nonresponders, and medicated versus unmedicated participants. Parsing the data further, separate correlations were carried out for medicated versus unmedicated responders, and medicated versus unmedicated nonresponders.

Finally, using SPSS (IBM version 20), we ran a series of multilevel models to test for relations between self-reported social anxiety and its predictors over the course of the study. Analysis of social anxiety on four separate occasions provided estimates of within-participant variability in anxiety (Level 1). The average of the scores from all four visits quantified the variability that existed between individual participants (Level 2). The proportion of within-participant variance to total variance quantified the extent to which self-reported anxiety changed across visits. The proportion of between-participant variance to total variance quantified the extent to which self-reported anxiety differed among participants.

Scores from self-reports collected on multiple occasions are presumed to derive from differences among the individual participants, differences among measurement occasions, and random measurement error. Multilevel models allow one to evaluate variance in a dependent variable across different, hierarchically-arranged levels in a nested data structure (Singer and Willett, 2003). These models assess the effects of predictors from different levels and also test for interactions within and across levels. Variance in the dependent variable is partitioned according to a) within-participant differences, i.e., differences related to the measurement occasion (Level 1) and b) between-individual differences, i.e., differences among individual participants (Level 2). We evaluated self-reported social anxiety (SPIN scores) as the dependent variable in comparative models that progressed from an unconditional model with no predictors to models with the following independent predictors: visit (Model 1); treatment response status (Model 2); medication status (Model 3); cognitive reappraisal (Model 4); expressive suppression (Model 5); resting RSA (Model 6); RSA and reappraisal as co-predictors (Model 7); and RSA and suppression as co-predictors (Model 8). We also ran a final exploratory model in which reappraisal and suppression were entered as co-predictors (Model 9). In the first four models, predictors were added in a cumulative fashion, such that each subsequent model contained a new predictor in addition to the predictors that came before. In Models 4–9, visit, response group, and medication status were all included as covariates.

3. Results

3.1. Self-report indices

3.1.1. Social anxiety

Mean SPIN scores across visits are depicted in Fig. 1. Responder status was determined by the RCI, which defined a cut-off for the change in SPIN scores required for membership in the responder group. Mean total social anxiety symptoms declined over the course of the study ($p < .001$), an effect that, naturally, interacted with our defined groups ($F(3, 60) = 21.13, p < .001, \eta^2_p = .51$) (see Moscovitch et al., 2012). At therapy’s end, anxiety scores were lower in treatment responders than nonresponders ($t(21) = -4.40, \ p < .001$). Prior to treatment, the groups did not differ ($t(21) = 0.10, p > .90; \text{Visit 2}$).

3.1.2. ERQ reappraisal

Self-reported use of reappraisal increased during the study ($p < .02$), but this effect interacted with responder status ($F(3, 63) = 3.96, p < .02, \eta^2_p = .16$). Responders reported greater use of reappraisal than nonresponders ($t(21) = 3.43, p < .01$), a difference that was maintained at the end of treatment (Visit 4; $t(21) = 2.10, p < .05$). Reappraisal did not differ by group before treatment ($t(21) = 0.57, p > .55; \text{Visit 2}$).

3.1.3. ERQ suppression

There were no main effects of visit or group for suppression ($ps > .30$), and any change in suppression across visits interacted very marginally with responder status ($F(2, 42) = 2.51, p < .10, \eta^2_p = .11$).

3.2. Physiological index: RSA

Mean resting RSA levels declined over the course of the study ($F(3, 54) = 3.83, p < .03, \eta^2_p = .18$). Pairwise tests indicated RSA was higher at Visit 1 than at Visit 4 ($p < .04$). There was no difference between responders and non-responders ($p > .35$), or interaction with group ($p > .75$). A separate mixed model ANOVA found no overall gender differences in resting RSA ($p > .65$). However, the decline across visits was gradual in women, whereas men showed a steep drop between Visits 1 and 3, followed by some recovery (interaction: $p < .04$).

3.3. Correlational analyses

As we were ultimately interested in modeling change in anxiety over time, the dataset was converted to a stacked format for multilevel analysis. The stacked dataset (scores from 23 participants × 4 visits) was incomplete as there were 4 missing RSA values and one missing

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1 Treatment response was operationalized as a continuous variable reflecting individual differences in anxiety reduction during the study. Responder group status was determined by the Reliable Change Index (RCI), which defined a cut-off for the degree of change in SPIN scores necessary for membership in the responder group. Thus, a decline in anxiety levels that failed to meet the criteria for responder group membership would still contribute to change in anxiety in both the ANOVA and multilevel growth models. Responder group status and SPIN scores are different, though related, metrics.
SPIN value. To ensure comparable models, the data points were filtered to account for these missing values, leaving 87/92 possible data points for analysis.

Zero-order correlations of anxiety with reappraisal, suppression, and RSA were calculated in order to examine simple relations between these predictors and anxiety. In these analyses, scores from all visits constituted the units to be analyzed, rather than individual participants ($N = 87$ scores). Across the entire sample, reappraisal was inversely associated with both anxiety ($r(87) = -0.24, p = .025$) and resting RSA ($r(87) = -0.22, p = .043$). There were no other significant relations ($ps > .16$).

Separate correlations were then carried out for scores from all responders versus all nonresponders. Among responders ($n = 56$ scores), anxiety was inversely correlated with reappraisal ($r(56) = -0.34, p = .01$) and positively correlated with resting RSA ($r(56) = 0.27, p = .048$). Together, these findings suggested that in responders, reappraisal and resting RSA acted in opposition with respect to anxiety. Among responders, anxiety scores were positively related to suppression scores ($r(56) = .26, p = .05$). There were no other relations ($ps > .08$). Among nonresponders, reappraisal and suppression scores were positively correlated ($r(31) = .40, p = .026$) with no other relations ($ps > .13$).

Separate correlations were then carried out for scores from all medicated versus all unmedicated participants. Among medicated participants, anxiety was inversely related with reappraisal ($r(55) = -0.27, p = 0.047$) with no other relations ($ps > .09$). There were no significant correlations among these variables for unmedicated participants ($n = 32; ps > .25$).

Parsing the data further, separate correlations were carried out for medicated versus unmedicated responders, and medicated versus unmedicated nonresponders. In medicated responders, anxiety and reappraisal scores were inversely related ($r(29) = -0.39, p = .036$) but anxiety and suppression showed a positive correlation ($r(29) = .43, p = .022$). Moreover, in this subgroup, reappraisal and suppression scores were inversely correlated ($r(29) = -0.53, p = .003$). There were no relations for unmedicated responders ($ps > .12$). In medicated nonresponders, reappraisal and suppression scores were positively correlated ($r(26) = .47, p < .015$) with no other relations ($ps > .28$). In unmedicated nonresponders, the correlation between anxiety and reappraisal scores was strongly positive ($r(5) = .94, p = .016$) with no other relations ($ps > .19$), but it must be noted that this subgroup of scores was very small.

### 3.4. Predictions of anxiety in multilevel analyses

A series of growth curve models of increasing complexity were constructed to test our hypotheses concerning predictors of change in anxiety during the study. In these models, treatment responder status and medication status were each represented as yes/no (1,0) dichotomous variables. Continuous variables (RSA, reappraisal, and suppression scores) were centered within participant prior to analysis, to estimate the intercept (starting point) and the slope (growth) of individual anxiety trajectories. The intercept is interpreted as a participant’s average anxiety level at the start of therapy; the slope represents the average rate of change in anxiety during the study.

Regression coefficients for the fixed and random effects of variables predicting anxiety are presented in Table 1. We began by fitting an unconditional model (one with no predictors) to the observed data, providing a comparison model against which the fit of other models could be judged. The unconditional means model represented the grand mean of SPIN scores (41.27) for all participants across all visits, which was significant ($t = 19.11, p < .001$). However, substantial differences in starting levels of anxiety across participants (Level 2; Wald = 2.55, $p = .011$), and across visits (Level 1; Wald = 5.67, $p < .001$) remained unexplained by the unconditional model.

Model 1 confirmed that anxiety scores declined linearly across consecutive visits during the study ($t = -5.55, p < .001$), in accord with our definition of treatment response. The starting points for participants’ anxiety levels varied significantly (Wald = 3.02, $p = .003$), in Model 1 and all of the remaining models ($all ps < .01$). Anxiety scores also differed in their rates of change across visits (Wald = 2.57, $p = .01$), but there was no evidence for covariation between the starting levels and the rates of change (Wald = 0.54, $p = .59$).

Responder status was added as a predictor in Model 2 to account for differential declines in anxiety between responders and nonresponders during therapy (reported by Moscovitch et al., 2012). A significant interaction between responder status and visit confirmed that anxiety declined more steeply in those who were ultimately deemed to have shown significant improvement by the end of therapy ($t = 6.95, p < .001$). In this model, estimates for the covariance parameters now indicated no difference in rates of change across visits (Wald = 0.73, $p = .466$), a finding that emerged in most of the remaining analyses as well ($ps > .10$ except Model 9). There was no significant covariation between initial anxiety levels and their rates of change (Wald = 0.59, $p = .553$) in this or any of the subsequent analyses ($all ps > .11$).

Medication status was added in Model 3 to account for the effects of psychotropic medication use on anxiety. Surprisingly, taking medications was associated with higher anxiety scores across the sample ($t = 2.40, p = .023$), suggesting a selection effect whereby those with higher levels of anxiety were more likely to be prescribed medications. In this model, a significant three-way interaction among visit, responder status, and medication status, indicated that the observed decline in anxiety was qualified by both responder status and medication use ($t = 2.49, p = .02$).

Cognitive reappraisal was added in Model 4 to assess the relation between anxiety and self-reported use of reappraisal during the study. In this model, a significant three-way interaction among reappraisal, responder status, and medication status ($t = 2.62, p = .013$) indicated that the relation between reappraisal and anxiety also depended on responder status and medication use.

The relation between anxiety and self-reported use of suppression was assessed in Model 5. This model indicated that the decline in anxiety was qualified by medication status and responder status, in a three-way interaction among medication status, responder status, and visit ($t = 2.59, p = .015$). Another three-way interaction among suppression, responder status, and medication status ($t = -2.41, p = .02$) indicated that the relation between suppression and anxiety also depended on responder status and medication use.

Based on previous reports that higher resting RSA may be associated with more efficient emotion regulation (e.g., Brosschot et al., 2007; Geisler et al., 2010), we introduced RSA as a hypothesized predictor of anxiety in Model 6. Here, a significant three-way interaction among RSA, responder status, and medication status ($t = -2.70, p = .01$) suggested that the relation between RSA and anxiety also depended on responder status and medication use.

Cognitive reappraisal and RSA were then entered together as co-predictors in Model 7. Initially, this model would not converge because the test statistic for variance in the rate of change in anxiety across visits was near zero and could not be calculated. Therefore, Model 7 was tested again, with visit modeled as a fixed effect only, and not as a random effect. Removing the random effect of visit meant that the revised Model 7 did not model any variation in individual slopes (rates of change in anxiety across visits). This model successfully converged, and indicated that the decline in anxiety over time was influenced by all four predictors, as demonstrated in two significant four-way interactions involving visit, medication status, reappraisal and responder status ($t = -2.86, p < .005$) and visit, medication status, reappraisal, and RSA ($t = -4.09, p < .001$). Suppression and RSA were examined as co-predictors of anxiety in Model 8. In this model, suppression did not predict anxiety, either as a
Table 1
Multilevel models predicting social anxiety.

<table>
<thead>
<tr>
<th>Fixed effects</th>
<th>Unconditional</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
<th>Model 5</th>
<th>Model 6</th>
<th>Model 7</th>
<th>Model 8</th>
<th>Model 9</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
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<td>14.23 (8.30)</td>
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<td>18.62 (8.21)</td>
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Random parameters

Within-participant (Level 1)

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Between-participant (Level 2)

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Notes: In Model 7, Visit was modeled as a fixed effect, but removed as a random effect, because variance in anxiety related to visit was close to zero.

Visit = testing occasion; Group = responder group; Med = medication status; CR = cognitive reappraisal; ES = expressive suppression; RSA = respiratory sinus arrhythmia.

**Bold** = p < .05, † = p < .06.
main effect or in interaction with other variables (all ps > .17). In contrast, RSA still predicted change in anxiety over time, depending on medication status, in a three-way interaction involving RSA, visit and medication status (t = −3.12, p = .003).

Finally, reappraisal and suppression were modeled together to assess their interactive effects on anxiety (Model 9). Here, a four-way interaction among visit, medication status, reappraisal, and suppression indicated that the decline in anxiety was qualified by medication status and both of the emotion regulation strategies (t = −5.44, p < .001). In this final model, individual slopes (rates of change in anxiety across visits) differed significantly (Wald = 2.41, p = .016).

3.5. Model fit

Goodness of fit tests for the above models (i.e., statistical tests of the correspondence between the observed data and their predicted values) are presented in Table 1. Evaluation of model fit with respect to the observed data was estimated by −2 log likelihood tests. Lower estimates indicate better fit (Hox, 2002). Overall, our models explained increasing amounts of variance in anxiety up to and including Model 7 (−2 log likelihood; unconditional model: 677.82, Model 7: 517.41). To examine the contribution of each new variable or combination of variables to predictions of anxiety, we compared each model to the model that preceded it, by turn, with three exceptions. Models 4, 5, and 6 were each compared to the model that preceded predictions of anxiety, we compared each model to the model that preceded it, by turn, with three exceptions. Models 4, 5, and 6 were each compared to the model that preceded.
additional variance related to individual differences in starting levels of anxiety, but did not explain more variance related to anxiety reduction over time. The finding regarding starting levels was interpreted as a possible selection effect, whereby participants with higher levels of anxiety were more likely to be taking medications. Although medications may promote anxiety reduction, the present analysis suggests that the use of medications did not actually account for very much change in anxiety across visits, possibly because medication status (medication type and dosage) was held constant for the entire period of the study. As medication use did not determine the response to CBT, the reduction in self-reported anxiety over time likely occurred as a result of the group therapy intervention.

Adding reappraisal to Model 4 improved predictions of anxiety reduction across visits. Participants who acquired reappraisal skills and began to apply them to their daily lives (mainly responders) showed greater declines in anxiety than those who did not (mainly nonresponders), suggesting that lower levels of anxiety may be achieved partly through cognitive means. Interestingly, substituting suppression (Model 5) for reappraisal produced as good a fit to the data as did the model with reappraisal, intimating that increased use of reappraisal may have been mirrored by decreased use of suppression. As expected, correlational analyses indicated that while reappraisal was inversely associated with anxiety, suppression use predicted higher levels of anxiety in some participants. We note that responders appeared to fit this pattern, i.e., anxiety was lower in those who were more successful at acquiring reappraisal skills and were also better able to relinquish the habitual use of suppression to cope with anxiety. This pattern came into even sharper focus in the subgroup of medicated responders, who exhibited a significant inverse correlation between reappraisal and suppression as well as the inverse relation between anxiety and reappraisal and the positive relation between anxiety and suppression. Together, these findings are consistent with what is known about antecedent- and response-focused strategies of emotion regulation (e.g., Gross and John, 2003). In contrast to responders, the use of reappraisal in nonresponders was positively related to suppression, suggesting that adults who ultimately did not do well in group CBT may have tried in vain to apply both strategies simultaneously in their efforts to regulate anxiety.

Findings with respect to RSA were surprising. Higher levels of resting RSA, reflecting an individual’s capacity for physiological self-regulation, were expected to be inversely related to anxiety. However, resting RSA levels were positively related to anxiety in responders, and also declined significantly across testing sessions. This unforeseen reduction in RSA may have been attributable to the demands of the testing sessions. At each visit, participants were asked to prepare and deliver a short, videotaped speech following a baseline cardiac recording. During the baseline recording in Visit 1, participants were naı́ve to these demands. After the initial visit, they appeared to have anticipated the anxiety-provoking speech task during baseline recordings, rather than simply resting (see Schmidt et al., 2012). Therefore, the unforeseen reduction in RSA across testing sessions (visits) may have reflected parasympathetic withdrawal in anticipation of an expected stressor. Moderate RSA withdrawal prior to an anxiety-provoking public-speaking task is an appropriate autonomic response to an anticipated increase in environmental demands. Like high levels of resting RSA, moderate RSA withdrawal has been associated with optimal preparedness to respond, increased physiological flexibility and adaptability (Beauchaine, 2001). Conversely, failure of parasympathetic withdrawal during particular cognitive tasks (e.g., timed tasks) has sometimes been associated with poor performance (Duschek et al., 2009).

Our models indicated that RSA explained substantial variance in anxiety reduction related to visit, rather than to individual differences in participants’ starting levels of anxiety. Thus, it was not the case that individuals with higher starting levels of resting RSA exhibited lower overall levels of anxiety. Indeed, higher levels of resting RSA across the study were positively, rather than negatively associated with anxiety scores. Therefore, change in RSA levels – (in this case, RSA reduction in response to challenge) – over the course of therapy appeared to be more important for predicting change in anxiety symptoms than did baseline levels of RSA. The degree to which RSA is reduced in response to challenge may constitute a marker for competent physiological regulation in adults with SAD, as it is in non-clinical populations (Beauchaine, 2001). Furthermore, reappraisal is known to influence cardiovascular and cognitive responses to stress (Jamieson et al., 2012). Individuals’ capacity to downregulate the physiological arousal associated with emotional reactivity may be an important aspect of reappraisal skills training for social anxiety.

Ultimately, anxiety levels were best explained when medication status was accounted for, and reappraisal and RSA were modeled simultaneously. Medication status predicted significant variance related to individual differences in participant anxiety when it was introduced in Model 3. The combination of reappraisal and RSA in Model 7 resulted in major reductions in the proportional prediction error for variance in anxiety that was associated with visit over other models that did not include RSA (compared to Model 4: 48%; Model 5: 43%; Model 6: 41%), or included suppression (Model 8: 19%). Only Model 9 (reappraisal and suppression) accounted for variance in anxiety reduction across visits better than Model 7 (36%), but this model explained variance in individuals’ starting levels of anxiety less well (–18%) and represented a worse overall fit to the observed data. In sum, predictions of within-and between-participant variance in social anxiety were greatest when reappraisal, resting RSA, and medication status were modeled together with responder status.

These results suggest the possibility of engaging the parasympathetic system in therapeutic interventions for SAD. Studies have shown that RSA biofeedback training may reduce physiological arousal in adults with high levels of stress (e.g., Sherlin et al., 2010) or anxiety (e.g., Zucker et al., 2009), with carryover effects that extend to repeated stressors. Increasing parasympathetic regulatory capacity may represent an additional target for therapeutic intervention with socially anxious adults.

4.1. Limitations

Although our findings suggest a plausible relation between parasympathetic regulation and anxiety reduction in patients with SAD, several limitations of the study bear mentioning. First, we acknowledge the constraints inherent in self-report measures. Our measures have been well-validated and are in common use, but participant reports of reappraisal and suppression may not correspond perfectly to actual rates of utilization, and responses could reflect either a degree of social desirability or desire for progress. It will be important to replicate these preliminary results, especially the findings for suppression, given the modest reliability of this measure.

Second, while our findings are consonant with reports involving other anxious populations, inclusion of a waitlist group would have allowed comparison of the effects of group CBT versus the mere passage of time on the parameters of interest. We note, however, that CBT is an efficacious form of therapy that has been shown in numerous studies to induce significant changes over 12 weeks in comparison to inert control conditions (see Smits and Hofmann, 2009).

Third, collecting data across multiple visits depended on consistent attendance of participants both in the CBT program and at the testing sessions, which restricted the final sample to a smaller group. The limited sample may have affected the ability of Model 7 to assess variance in the rates of change in this sample. Small sample sizes are quite common in clinical research, especially when repeated visits are required. Yet even with this restricted sample, Models 4 (reappraisal) and 6 (RSA) indicated that reappraisal skills and RSA


