

Sudden Gains During Therapy of Social Phobia

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The present study investigated the phenomenon of sudden gains in 107 participants with social phobia (social anxiety disorder) who received either cognitive–behavioral group therapy or exposure group therapy without explicit cognitive interventions, which primarily used public speaking situations as exposure tasks. Twenty-two out of 967 session-to-session intervals met criteria for sudden gains, which most frequently occurred in Session 5. Individuals with sudden gains showed similar improvements in the 2 treatment groups. Although cognitive–behavioral therapy was associated with more cognitive changes than exposure therapy, cognitive changes did not precede sudden gains. In general, the results of this study question the clinical significance of sudden gains in social phobia treatment.

Keywords: sudden gains, social anxiety disorder, social phobia, cognitive–behavioral therapy, exposure therapy

In recent years, psychotherapy researchers have investigated the phenomenon of large, rapid, and stable decreases in symptomatology during treatment, which has been referred to as *sudden gains* (Tang & DeRubeis, 1999b). Sudden gains are typically defined by a set of three quantitative criteria: (a) the sudden gain must be large in absolute terms, (b) the sudden gain must represent at least a 25% reduction from the level of symptomatology before the gain occurred, and (c) the mean level of symptomatology in the three therapy sessions preceding the gain must be significantly higher than the mean level of symptomatology in the three postgain sessions. Most studies of sudden gains have examined treatment changes in depression with the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961; but see also Stiles et al., 2003; Vittengl, Clark, & Jarrett, 2005).

When applying these criteria, Tang and DeRubeis (1999b) found that sudden gains occurred in more than 50% of individuals who responded to cognitive–behavioral therapy for depression and that these gains accounted for more than 50% of these individuals' total improvement in depression. Compared with participants who did not experience sudden gains at posttreatment, individuals with sudden gains showed better outcome at posttreatment and at 6-month and 18-month follow-ups.

The initial study by Tang and DeRubeis (1999b) was followed by a number of other studies examining sudden gains (Gaynor et al., 2003; Hardy et al., 2005; Kelly, Roberts, & Ciesla, 2005; Tang, DeRubeis, Beberman, & Pham, 2005; Tang, Luborsky, & Andrusyna, 2002; Vittengl, Clark, & Jarrett, 2005). Interventions in

these studies ranged from traditional cognitive therapy to supportive expressive therapy or pharmacotherapy. Although sudden gains were primarily assessed in efficacy trials of time-limited individual treatments (e.g., Tang & DeRubeis, 1999b; Vittengl et al., 2005), their incidence was also assessed in group therapy for depression (Kelly et al., 2005) and individuals with mixed psychiatric diagnoses who were given routine treatments in outpatient clinic settings (Stiles et al., 2003). Overall, results corroborated the existence of sudden gains during the acute phase of treatment, as defined by the criteria of Tang and DeRubeis (1999b), with the median pregain session occurring at Session 5. The mean magnitude score reduction on the BDI varied from 10 to 13 points, with 17%–50% of clients experiencing sudden gains.

All studies showed larger overall posttreatment symptom reductions in patients who experienced sudden gains during acute treatment compared with patients who did not. Predictions for long-term maintenance of gains, however, were mixed. Some studies showed better outcomes at follow-up for patients with sudden gains (Gaynor et al., 2003; Hardy et al., 2005; Stiles et al., 2003; Tang & DeRubeis, 1999b), whereas other studies found no differences at follow-up for patients with and without sudden gains (Tang et al., 2002; Vittengl et al., 2005). Vittengl et al. (2005) reported that patients with sudden gains during acute treatment actually showed more depressive symptoms and negative failure attributions at follow-up than those who had not experienced sudden gains. The authors argued that the sudden gain treatment response pathway might be more likely to occur for patients who show higher pretreatment scores on self-report measures of depression.

The studies examining sudden gains to date have exclusively focused on depression. The present study is the first to investigate the occurrence of sudden gains during psychological treatment of social phobia (social anxiety disorder), the most common form of anxiety disorder in the population (Kessler et al., 1994). The most efficacious psychological interventions for social phobia are exposure therapy and cognitive–behavioral treatments (Hofmann & Barlow, 2002). Contemporary theories of social anxiety and social phobia emphasize the role of cognitive processes, especially self-

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perception, in the maintenance of the disorder (e.g., Clark & Wells, 1995; Leary & Kowalski, 1995; Rapee & Heimberg, 1997). It has therefore been suggested that effective psychological treatment changes the person's representation of the self in a more positive direction (e.g., Clark & Wells, 1995; Rapee & Heimberg, 1997). Supporting evidence for this hypothesis comes from a number of treatment studies suggesting that effective treatment of social phobia specifically reduces negative self-perception (Hofmann, Moscovitch, Kim, & Taylor, 2004; Woody, Chambless, and Glass, 1997).

The objective of the present study was to examine changes in social anxiety and self-perception during the course of treatment for social phobia. Treatment consisted of either cognitive-behavioral group therapy (CBGT) or exposure group therapy (EGT; without explicit cognitive interventions). On the basis of the existing sudden gain literature, we predicted that we would be able to identify sudden gains during both treatment modalities when adopting the criteria to define sudden gains to the social phobia treatments. We expected that individuals receiving CBGT would show more frequent sudden gains and that these gains would be preceded more often by changes in cognitions (i.e., a reduction in negative self-statements) than individuals receiving EGT. Finally, we predicted that individuals with sudden gains would show better treatment response at the end of treatment and at follow-up than individuals without such gains.

Method

Participants

Data were compiled from 107 patients who received a principal *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994) diagnosis of social phobia on the basis of an assessment with the Anxiety Disorders Interview Schedule for

DSM-IV: Lifetime Version (DiNardo, Brown, & Barlow, 1994). The study was approved by the Institutional Review Board of the university. All participants gave their consent by signing a consent form that was reviewed with participants. Informed consent was obtained by a member of the research staff at the initial meeting prior to the pretreatment assessment.

Exclusion criteria for this study included (a) prior nonresponse to adequately delivered study treatment, (b) current diagnosis of psychoactive substance abuse or dependence, (c) current active suicidal potential, (d) current diagnosis of bipolar disorder, and (e) current diagnoses of schizophrenia or other psychotic disorders. In addition, participants had to report at least moderate public speaking anxiety, which was defined by a self-report rating of 4 or greater on an 8-point Likert scale. This inclusion criterion was chosen because repeated exposure to speech situations was an important component of one of the treatment protocols (EGT). Although this intervention used only public speaking situations as in-session exposure practices, we found in previous studies that the treatment effects easily generalized to other social situations (Hofmann, 2004). Most individuals with social phobia who presented at our clinic for treatment (88.9%) met all study criteria and were invited to participate in the study.

Participants were predominantly men ($n = 63$; women, $n = 44$) and Caucasian ($n = 94$). Non-Caucasian participants identified themselves as African American ($n = 4$), Asian ($n = 4$), or Hispanic ($n = 2$). The average age at intake was 32.54 years ($SD = 9.86$), with a mean of 16.01 ($SD = 2.46$) years of education. They had a mean annual income of \$45,762 ($SD = 47,968$; Mode = 30,000; $Mdn = 32,500$; range = 0–310,000). Seventy-three percent of the patients ($n = 78$) were diagnosed with the generalized subtype of social phobia, including 1 patient with a principal diagnosis of avoidant personality disorder. Forty-eight percent of the participants ($n = 52$) received one, and 18% ($n = 19$) more than one, additional secondary clinical diagnosis. The most common comorbid Axis I diagnoses were major depressive disorder ($n = 33$), generalized anxiety disorder ($n = 12$), and dysthymia ($n = 7$). A comparison between the two treatment conditions in the demographic and clinical characteristics is presented in Table 1.

Table 1
Comparison Between Treatment Conditions in Demographic and Clinical Characteristics

Variable	EGT ($n = 59$)			CBGT ($n = 48$)			χ^2	t	df	p
	N	M	SD	N	M	SD				
Subject characteristics										
Gender							0.02		1	.89
Men	35			28						
Women	24			20						
Age (years)		32.08	9.78		33.10	10.04		0.53	105	.60
Ethnicity							0.00		1	.99
White	52			42						
African American	3			1						
Asian	2			2						
Hispanic	1			1						
Other	1			2						
Family and social background										
Income (US \$)		44,726	42,922		47,118	54,420		0.23	88	.82
Married	14			15			1.23		1	.27
Generalized subtype	44			33			0.89		1	.34
Additional Axis I diagnoses	42			30			1.45		1	.23
Self-report measures at pretest										
LSAS		40.75	18.10		44.58	15.79		1.11	95	.27
SPAI		102.35	27.81		108.01	24.74		1.10	102	.28

Note. EGT = exposure group therapy; CBGT = cognitive-behavioral group therapy; LSAS = Liebowitz Social Anxiety Scale; SPAI = Social Phobia and Anxiety Inventory.

Treatments

Participants attended at least 8 of 12 weekly sessions of CBGT ($n = 48$) or EGT ($n = 59$). CBGT is a comprehensive cognitive-behavior treatment protocol (Heimberg, 1991), whereas EGT is an exposure treatment without explicit cognitive interventions (Hofmann, 1999). Some of the participants were part of a previous study (Hofmann, 2004). As part of this study, participants were randomly assigned to one of two treatment modalities (CBGT vs. EGT).¹ Participants in the two groups did not differ in marital status, gender, or education status ($ps > .2$). However, participants in the EGT group were slightly older (in years, $M = 34.39$, $SD = 10.29$) than participants in the CBGT group ($M = 30.27$; $SD = 8.89$), $t(105) = 2.19$, $p = .03$.

The treatment attrition rates were 16% (9 of 57) from CBGT and 22% (17 of 76) from EGT. The two study treatments did not differ in their attrition rates, $\chi^2(1) = 0.66$, $p > .40$. Furthermore, attrition from the study was not associated with any demographic variables or Axis I symptomatology ($ps > .20$, $ds < 0.17$). More detailed results on these attrition data will be reported elsewhere (Hofmann & Suvak, in press).

The two study treatments were conducted in a group format with two therapists and 5–7 participants per group and delivered according to structured treatment protocols. Nine therapists with comparable experience had been trained to implement the two treatments. The training consisted of (a) reading the detailed treatment protocol, (b) listening to an audiotaped first treatment session (in which the treatment rationale was presented), (c) attending weekly supervision meetings, and (d) coleading at least one complete treatment group. All therapists were advanced doctoral students in clinical psychology. Harlan Juster, a senior therapist and former collaborator of Richard G. Heimberg, the author of the CBGT protocol, provided weekly telephone supervision for the CBGT groups. Stefan G. Hofmann, who developed the EGT manual, supervised the EGT therapists on a weekly basis. The EGT protocol included repeated in-session in-vivo exposures to social performance situations, video feedback, didactic training, and weekly homework assignments. Although participants feared numerous social situations, this intervention focused primarily on the patients' public speaking anxiety. We limited the exposure situations of the EGT group to repeated public speaking situations because public speaking is the most commonly feared social situation that can easily be created in a group setting. Previous studies have shown that treatments that primarily target public speaking anxiety generalize to other social fears in that they have similar acute treatment effects on generalized social anxiety symptoms as more comprehensive treatments (e.g., Newman, Hofmann, Trabert, Roth, & Taylor, 1994). Moreover, we modified the topic or setting of the situation to elicit at least moderate anxiety even in participants who did not rate public speaking as their main social fear. For example, a participant who reported dating as his or her main fear might have been asked to give a speech on "dating rules" or "my most embarrassing moment during dating," and a person who was concerned with assertiveness situations and disagreeing with others might have been asked to give a speech on a controversial topic, in which he or she argued against the majority opinion of the group.

In contrast, patients in the CBGT groups were taught skills to identify negative cognitions, observe the covariation between anxious mood and cognitions, examine thinking errors—including overestimation of social cost—and formulate rational alternatives to these errors. Exposure exercises were conducted in session and assigned for homework. Exposures were explained as a means by which patients could scientifically test the validity of anxious predictions. In addition to the difference in the emphasis on cognitive strategies, the two protocols also differed in the treatment rationale, which was based on the cognitive model of anxiety in the case of CBGT and on a basic habituation rationale in the case of EGT. The results from previous meta-analyses (Feske & Chambless, 1995; Gould, Buckminster, Pollack, Otto, & Yap, 1997; Taylor, 1996) suggest that both treatment protocols produce comparable short-term treatment effects.

Treatment Integrity

A random sample of 41 treatment sessions were audiotaped (19 different CBGT sessions and 22 different EGT sessions) and selected to be evaluated blindly for protocol adherence and therapist competence by a 5th-year doctoral student in clinical psychology. This person had been trained in various empirically supported interventions and closely supervised in her clinical work by experienced and licensed clinicians. She was further familiar with both treatment protocols involved in the study. To examine the reliability of the rating system, a master's student also blindly evaluated a random subsample of 22 taped sessions (11 CBGT tapes and 11 EGT tapes).

Therapist adherence to the two protocols was determined by rating the following items on a 5-point Likert scale (1 = *not met*, 3 = *partly met*, 5 = *fully met*): "The therapists used a 'purely' behavioral model of social phobia (i.e., without specific cognitive interventions)" and "The therapists used cognitive restructuring techniques." We estimated interrater agreement by calculating intraclass correlation coefficients (ICC; Shrout and Fleiss, 1979). Specifically, we used Shrout and Fleiss's ICC (2, 1) equation, which assumes that the two raters are random effects. The results suggest that the two raters showed high agreement in the degree to which therapists used a "purely" behavioral model (ICC = 0.82, $p < .0001$). Moreover, the two raters agreed in their assessment of the extent to which therapists used cognitive restructuring techniques (ICC = 0.995, $p < .0001$). The EGT therapists used a more behavioral model ($M = 4.77$, $SD = 0.87$) relative to CBGT therapists ($M = 1.00$, $SD = 0.00$), $t(21) = 20.36$, $p < .0001$, $d = 5.97$, whereas the CBGT therapists used relatively more cognitive restructuring techniques ($M = 4.79$, $SD = 0.92$) relative to EGT therapists ($M = 1.27$, $SD = 0.55$), $t(39) = 15.12$, $p < .0001$, $d = 4.49$.

The senior rater further evaluated the 41 tapes for therapist competency using an adaptation of the Vanderbilt Psychotherapy Process Scale (Suh, Strupp, & O'Malley, 1986). Specifically, the rater evaluated each tape using a 5-point Likert scale (1 = *not at all*, 2 = *some*, 3 = *fair amount*, 4 = *pretty much*, and 5 = *great deal*) to characterize the therapeutic relationship and the therapist's demeanor. The results showed that both treatment groups were rated high on the general quality of the relationship between the group members and the therapists (CBGT: $M = 4.53$, $SD = 0.51$; EGT: $M = 4.59$, $SD = 0.59$), on their productivity (CBGT: $M = 4.57$, $SD = 0.50$; EGT: $M = 4.41$, $SD = 0.50$), and on how well the therapists and the group members worked together (CBGT: $M = 4.74$, $SD = 0.45$; EGT: $M = 4.82$, $SD = 0.39$).

¹ The randomized clinical trial was based on 90 participants. To maximize our sample size, we included additional individuals who were part of the regular client flow. Those individuals were assigned, after a short waiting period, to one of the two treatment groups (CBGT vs. EGT), depending on therapist availability, which explains the unequal number of participants in the two conditions. In addition, we included participants who were part of the waiting list in the randomized, controlled trial. Therefore, the present study deviates from a randomized clinical trial, and we have insufficient data available to present a flow diagram of the progress. A comparison between the 90 participants who were part of the randomized trial and the 17 participants who were later added to the analyses showed no differences in any demographic characteristics, including gender, age, ethnicity, education, income, marital status, and comorbidity ($ps > .5$). Furthermore, there was no difference in the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987) at pretest ($p > .5$). The only difference was a slightly higher Social Phobia and Anxiety Inventory (SPAI; Turner, Beidel, Dancu, & Stanley, 1989) difference score in participants from the randomized trial at pretreatment, $t(102) = 2.10$, $p = .04$. However, the results of the subsequent analyses on sudden gains did not change when we excluded these 17 participants from the analyses.

Moreover, the therapists' demeanor during the sessions was rated as being involved (CBGT: $M = 5.00$, $SD = 0.00$; EGT: $M = 4.91$, $SD = 0.29$), optimistic (CBGT: $M = 4.53$, $SD = 0.70$; EGT: $M = 4.5$, $SD = 0.51$), and respectful (CBGT: $M = 4.95$, $SD = 0.23$; EGT: $M = 4.91$, $SD = 0.29$). Therapists from neither group appeared to be annoyed (CBGT: $M = 1.05$, $SD = 0.23$; EGT: $M = 1.00$, $SD = 0.00$), authoritative (CBGT: $M = 1.26$, $SD = 0.45$; EGT: $M = 1.18$, $SD = 0.39$), defensive (CBGT: $M = 1.00$, $SD = 0.00$; EGT: $M = 1.00$, $SD = 0.00$), or judgmental (CBGT: $M = 1.11$, $SD = 0.32$; EGT: $M = 1.00$, $SD = 0.00$; all $ps > .1$, all $ds < 0.44$). Because only the senior rater gave competency ratings, no reliability data were available for these data.

Assessments

Before treatment, all participants received the lifetime version of the Anxiety Disorders Interview Schedule for *DSM-IV*. This interview lasted between 4 and 6 hr per participant and was conducted by advanced clinical psychology doctoral students, who were unaware of the objective of this study. The kappa coefficient between two independent raters at our Center was .77 for social phobia as a principal diagnosis (Brown, DiNardo, Lehman, & Campbell, 2001). The reliability coefficients (kappa) for the other anxiety diagnoses ranged between .67 (generalized anxiety disorders) and .86 (specific phobias) and between .22 (dysthymia) and .72 (major depressive disorder) for mood disorders (Brown et al., 2001).

Additional assessments occurred immediately after treatment (posttest) and at 6-month follow-up (without receiving any additional treatments between the posttest and the follow-up assessment). All participants entered into the study with the understanding that they were not permitted to initiate any additional therapy for their social phobia for the duration of the study. This information was included in the consent form. None of the participants, including those who received a 6-month follow-up, initiated any psychological or pharmacological treatments during the course of the study.

At all assessment points, participants were asked to fill out the Social Phobia and Anxiety Inventory (SPAI; Turner, Beidel, Dancu, & Stanley, 1989). The SPAI is a 109-item self-report instrument that has been widely used to assess the cognitive, somatic, and behavioral dimensions of social phobia. This measure is capable of discriminating socially phobic persons from those with other anxiety disorders (Turner et al., 1989) and from normal controls (Beidel, Borden, Turner, & Jacob, 1989). Internal consistency estimates of the SPAI are high, ranging from .85 to .96 (Osman et al., 1996; Turner et al., 1989). The internal consistency of the SPAI difference score in the current sample was .99 at pretest, posttest, and follow-up. Stability of the SPAI has also been found to be high, with Turner et al. (1989) reporting a 2-week test-retest reliability of .86 on the SPAI difference score in a sample of college students. Compared with other popular measures of social anxiety, the SPAI was among the most reliable instruments and the most sensitive to treatment change and for distinguishing social phobia subtypes (Ries et al., 1998). Therefore, the SPAI was chosen as the primary outcome variable and measure of treatment change.

Prior to each session, participants' level of social anxiety was measured with a self-report version of the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987). This is a 24-item scale to measure fear and avoidance in 24 different social situations. The clinician-administered scale is widely used in studies of pharmacological treatment of social phobia. A recent study using a large sample of individuals with social phobia examined the psychometric characteristics of the self-report version of the LSAS (Baker, Heinrichs, Kim, & Hofmann, 2002). The self-report version of the LSAS was highly correlated with the clinician version, with correlations between these versions ranging from .78 to .85, depending on the subscales. Baker and colleagues (2002) also reported good 12-week test-retest reliability for the total score ($r = .83$). The internal consistency for the total score (Cronbach's $\alpha = .95$) was high. The self-report version of the instrument was also sensitive to treatment change. For the purpose of this study, we

averaged the ratings of all fear and avoidance items, which were scaled from 0 to 100. The LSAS was chosen as a session-by-session measure of treatment change.

In addition to these general measures of social anxiety, we also examined changes in cognitions related to social anxiety. For this purpose, we administered the Self-Statement During Public Speaking Test (SSPS, Hofmann & DiBartolo, 2000) prior to each session. The SSPS is a 10-item instrument that has two 5-item subscales to measure positive (SSPS-P) and negative self-statements (SSPS-N) related to a public speaking situation. The 2-factor structure of the measure was replicated in student and clinical samples. The internal consistency in two large undergraduate student samples ranged from .75 to .84 for the SSPS-P and from .83 to .86 for the SSPS-N. Similar results were found in clinical samples (Hofmann & DiBartolo, 2000). Clinical data showed that the SSPS-N (but not the SSPS-P) was sensitive to treatment changes. Participants who scored high on the SSPS-N had lower expectations for their success in a public speaking task, reported more anxiety during the task, and were less satisfied with their performance than participants who showed low SSPS-N scores (Hofmann & DiBartolo, 2000). We specifically examined the negative subscale scores of this scale as a measure of negative self-statements, which appear to be a mediator of treatment change in social phobia (as opposed to positive self-statements; Hofmann et al., 2004).

The internal consistencies (Cronbach's α) of the measures that were administered to the current sample at each session were high for the LSAS (.90-.96), SSPS-P (.86-.91), and SSPS-N (.83-.91).

Definition of Sudden Gains

Consistent with the terminology introduced by Tang and DeRubeis (1999b), the session immediately preceding the sudden gain is referred to as pregain session ($N - 1$), and the session preceding the pregain session is referred to as prepregain session ($N - 2$). The session immediately after the sudden gain is referred to as the aftergain session ($N + 1$). The criteria for sudden gains were as follows:

Criterion 1. The gain between session N and session $N + 1$ should be large in absolute terms. Some studies (e.g., Hardy et al., 2005; Stiles et al., 2003) defined the magnitude of the gain on the basis of the reliable change index (Jacobson & Truax, 1991). Consistent with this methodology, we derived the LSAS cutoff score for a sudden gain by dividing the LSAS change score from Sessions 1 to 12 by the standard error of the LSAS difference scores. This resulted in a critical LSAS difference score of 10.30. Tang and DeRubeis (1999b) reported that "for patients who experienced sudden gains, the sudden gains accounted for an average of 51% of their total symptom reduction" (p. 896). When we used an LSAS cutoff score of 10, the sudden gains accounted for an average of 50.27%. When we used the next higher cutoff score (11), the sudden gains accounted for 56.79% of the total symptom reduction. Therefore, to approximate Tang and DeRubeis' (1999b) methodology and to be consistent with the previous sudden gain literature, we chose an LSAS difference score of 10 as the cutoff to define a sudden gain.²

² The choice of the cutoff criterion for a sudden gain is arbitrary, and other strategies are possible. For example, one of the reviewers suggested that we define the cutoff as the score that is one standard deviation from the clinical sample mean. In fact, Tang and DeRubeis (1999b) chose 7 BDI points as the cutoff score, which happens to be approximately one standard deviation from the mean of depressed individuals. This strategy would translate to an LSAS cutoff score of 16. When we used this cutoff score, only 6 out of a total of 967 between-session intervals featured sudden gains, too few to conduct any meaningful statistical analyses. Although this is a plausible method, there is no precedence in the sudden gains literature for choosing one standard deviation as the cutoff rule. More important, it

Criterion 2. The improvement should be large relative to symptom severity before a sudden gain. Hence, the gain of the LSAS score at session $N + 1$ was required to be at least 25% of the score at session N .

Criterion 3. Finally, an improvement should be large in relation to symptom fluctuations before and after the gain. Tang and DeRubeis (1999b) used an independent t test, comparing the mean score of three therapy sessions before the gain (sessions $N - 2$, $N - 1$, and N) with the mean score of the three therapy sessions after the gain ($N + 1$, $N + 2$, $N + 3$). This criterion was subsequently criticized because of autocorrelation of the data (e.g., Hardy et al., 2005). Therefore, many studies have used the critical value, $t(4) = 2.78$, as the cutoff criterion to identify a sudden gain (e.g., Hardy et al., 2005; Vittengl et al., 2005).

We used both criteria and obtained identical results. Following the recommendations by Tang and DeRubeis (1999b), changes in the first session or before the last session were excluded from the analyses.

Handling of Missing Data

To establish Criteria 1 and 2, we compared two adjacent sessions. Of a total of 1,284 adjacent sessions, 139 sessions had missing data, with statistically equal numbers of missing data in each group (63 of 708 sessions in EGT and 76 of 576 in CBGT), $\chi^2(1) = 0.05$, $p = .82$. Therefore, 210 of 1,177 session to session comparisons could not be performed (93 of 210 in EGT and 117 of 210 in CBGT), $\chi^2(1) = 1.66$, $p = .20$.

To establish Criterion 3, we conducted independent-samples t tests to compare the mean of the three consecutive LSAS session scores before a possible sudden gain with the mean of the three consecutive LSAS scores after a possible sudden gain. Five comparisons in Criterion 3 had missing data (EGT = 2; CBGT = 3). One comparison in the EGT group was excluded because of significant missing data. The remaining 4 tests were performed with at least one missing data point (the one test in the EGT group had two missing data points, whereas each of the comparisons in the CBGT group had only one missing data point). The number of comparisons with missing data points did not differ between the two groups (CBGT: 3 of 112 vs. EGT: 1 of 27), $\chi^2(1) = 0.14$, $p = .71$. Results remained the same regardless of whether these tests were performed with fixed degrees of freedom (4) or whether degrees of freedom were adjusted to account for missing data.

To examine whether missing data might have influenced our results, we followed up each repeated measures analysis of variance (ANOVA) with an analogous analysis in a multilevel regression framework using the software program Hierarchical Linear Modeling-5 (HLM-5; Raudenbush, Bryk, Cheong, & Congdon, 2001). Multilevel, or mixed-effects, regression models have been increasingly used as an alternative technique to an ANOVA approach for analyzing repeated measure data (e.g., Bryk & Raudenbush, 1992; Raudenbush & Bryk, 2002). One advantage of this approach is that it is more efficient in analyzing unbalanced designs (i.e., when the number of data points and/or the time between data points varies from person to person). The results did not differ between the two methods. Therefore, we reported the repeated measures ANOVA analyses to facilitate comparisons to existing studies.

is questionable whether changes of one standard deviation in LSAS scores are clinically comparable to changes of one standard deviation in BDI scores in a different clinical sample, because of differences in the measurement constructs (depression vs. social anxiety), the measurement instruments, and the sample characteristics. In fact, when we used an LSAS cutoff score of 16, the sudden gains accounted for 63.57% ($M = 23.59$, $SD = 10.45$) of the total symptom reduction ($M = 37.11$, $SD = 17.52$), which is substantially higher than what has been reported in the sudden gains literature on depression. Therefore, we adopted the strategies by Hardy et al. (2005) and Stiles et al. (2003) for defining the cutoff score.

Results

Differences in Treatment Groups

To examine the short-term treatment effects, we conducted a 2 (group: EGT vs. CBGT) \times 2 (time: pretreatment vs. posttreatment) repeated measures ANOVA with the SPAI difference score as the dependent variable. The results showed a significant time effect, $F(1, 81) = 77.40$, $p < .001$, partial $\eta^2 = .49$. The group effect, $F(1, 81) = 0.03$, $p = .86$, partial $\eta^2 < .01$, and Time \times Group interaction effect, $F(1, 81) = 2.73$, $p = .10$, partial $\eta^2 = .03$, were not significant.

To examine the long-term treatment effects, we used a 2 (group: EGT vs. CBGT) \times 2 (time: post vs. follow-up) repeated measures ANOVA. The findings revealed no significant time effect, $F(1, 49) = 0.47$, $p = .50$, partial $\eta^2 = .01$, group effect, $F(1, 49) = 1.18$, $p = .28$, partial $\eta^2 = .02$, or Time \times Group interaction, $F(1, 49) = 2.22$, $p = .14$, partial $\eta^2 = .04$.³ The statistical assumptions were met for these and all other tests.

Occurrence of Sudden Gains

A total of 20 of 107 patients (18.69%) experienced at least one sudden gain. Two patients had two sudden gains. Therefore, only 22 of a total of 967 between-sessions intervals featured sudden gains. Sudden gains were distributed throughout the whole course of treatment. There were 7 individuals with sudden gains (41 without sudden gains) in the CBGT group and 13 individuals with sudden gains (46 without) in the EGT group. This difference was not statistically significant, $\chi^2(1) = 0.33$, $p = .57$. The sudden gains occurred most commonly in Session 5 (4 sudden gains) in the CBGT group and in Sessions 4 (2 sudden gains) and 11 (2 sudden gains) in the EGT group. When combining the two treatment groups, the gains occurred most commonly in Session 5 (5 sudden gains).⁴

The mean magnitude of all sudden gains (Figure 1) was 15.78 LSAS points ($SD = 7.20$). Individuals who experienced sudden

³ Because the two treatment groups differed in age, we also conducted 2 (group) \times 2 (time) repeated measures analyses of covariance with age as a covariate and SPAI difference score as the dependent variable. For the pre-post comparison, the results showed again a significant time effect, $F(1, 80) = 5.91$, $p = .02$, partial $\eta^2 = .07$, but no Time \times Group interaction effect, $F(1, 80) = 2.68$, $p = .11$, partial $\eta^2 = .03$, or group effect, $F(1, 80) = 0.02$, $p = .90$, partial $\eta^2 < .001$. The age effect and Time \times Age interaction effect were not statistically significant ($ps > .24$). For the post versus follow-up comparison, the results again showed no significant time effect, $F(1, 48) = 0.01$, $p = .97$, partial $\eta^2 < .001$, group effect, $F(1, 48) = 1.27$, $p = .27$, partial $\eta^2 = .03$, Time \times Group interaction effect, $F(1, 48) = 2.16$, $p = .15$, partial $\eta^2 = .04$, age effect, or Time \times Age interaction effects ($ps > .14$). Similar results were obtained when choosing the LSAS as the dependent variable.

⁴ To examine whether the group format of the treatment systematically influenced the results (e.g., whether participants in the same treatment group experienced sudden gains at the same time), we used HLM, a Bernoulli regression model, by means of the software program HLM-5. HLM has been developed for the analyses of nested data structures in which 1 participant contributes multiple data points at various time points. This procedure allows for unequal numbers of time series data points per person and variable spacing of time points across individuals (e.g., Bryk & Raudenbush, 1992; Raudenbush & Bryk, 2002). Level 1 of the multilevel

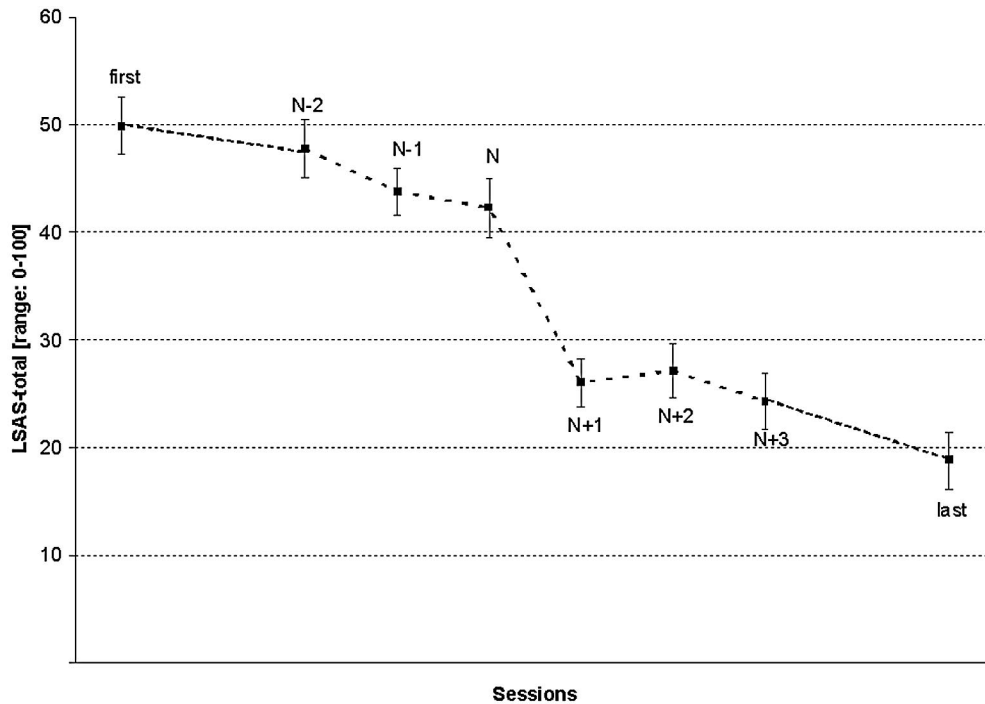


Figure 1. Mean sudden gains in the total sample. The total scores of the Liebowitz Social Anxiety Scale (LSAS) shown for Sessions $N - 2$, $N - 1$, N , $N + 1$, $N + 2$, and $N + 3$ are the means (with error bars representing standard errors) of the corresponding sessions from the 20 sudden gains in our sample, with session N as the pregain session. The figure also shows the first and last sessions of all patients who experienced sudden gains.

gains improved by 31.39 points ($SD = 13.31$) from pre- to post-treatment. Therefore, sudden gains accounted for 50.27% of participants' overall improvement. In contrast, individuals who did not experience a sudden gain only improved by 14.96 LSAS points ($SD = 14.32$). This difference was statistically significant, $t(23) = 4.03$, $p < .001$, $d = 1.19$. Patients experiencing sudden gains had significantly higher LSAS scores at pretreatment ($M = 51.07$, $SD = 11.00$) than patients without sudden gains ($M = 40.39$, $SD = 17.53$), $t(73) = 2.25$, $p = .03$, $d = 0.73$. Table 2 shows the comparison of patients with sudden gains in the two treatment groups.

Reversal of Sudden Gains

Consistent with the sudden gains literature, we examined how often participants gave up 50% or more of the gain achieved between Sessions N and $N + 1$ (i.e., a score greater than $LSAS_{N+1}$

+ .5 [$LSAS_N - LSAS_{N+1}$] for one or more sessions following the sudden gain session). When this criterion was used, 2 patients showed a reversal of their sudden gains.

Sudden Gains and Treatment Groups

To examine whether sudden gainers showed the same degree of change in social anxiety (as measured with the SPAI) during treatment as individuals without sudden gains, we used a 2 (group: individuals with vs. without sudden gains) \times 2 (time: pre vs. post) repeated measures ANOVA with the SPAI difference score as the dependent variable. The results revealed a significant time effect, $F(1, 81) = 70.50$, $p < .001$, partial $\eta^2 = .47$, and a significant Time \times Group interaction effect, $F(1, 81) = 4.91$, $p = .03$, partial $\eta^2 = .06$. Sudden gainers improved more during the course of treatment ($M = 34.18$, $SD = 25.65$) than patients without sudden gains ($M = 19.90$, $SD = 23.17$), $t(81) = 2.22$, $p = .03$, $d = 0.58$ (Figure 2). The group effect was not statistically significant, $F(1, 81) = 1.80$, $p = .18$, partial $\eta^2 = .02$.

To examine longer-term changes, we used a 2 (group: individuals with sudden gains vs. those without) \times 2 (time: post vs. 6-month follow-up) repeated measures ANOVA with the SPAI difference score as the dependent variable. The results showed no significant time effect, $F(1, 49) = 0.14$, $p = .71$, partial $\eta^2 = .003$, group effect, $F(1, 49) = 1.15$, $p = .29$, partial $\eta^2 = .02$, or Time \times Group interaction effect, $F(1, 49) = 0.003$, $p = .96$, partial $\eta^2 < .001$.

model consisted of individual participants who were nested within the treatment groups, the Level 2 component of the model. For the current analyses, 107 participants were nested within 24 groups. Examining the variance component of the unconditional model revealed that the probability of experiencing a sudden gain did not significantly vary across groups, $\chi^2(23) = 26.17$, $p = .29$. Because there was no effect of the nested structure of the data (i.e., individuals nested within groups) on the probability of experiencing a sudden gain, all subsequent analyses did not model treatment group-level variance.

Table 2
Comparison of Patients With Sudden Gains in EGT and CBGT

Variable	EGT (<i>n</i> = 59)	CBGT (<i>n</i> = 48)	χ^2	<i>t</i>	<i>df</i>	<i>p</i>
Individuals with sudden gains (<i>N</i>)	13	7	0.54		1	.46
Mode session of sudden gain	5	4 and 11				
Median session of sudden gain	7	8				
Average magnitude (% total improvement)	49.28	60.00				
Magnitude of first sudden gains				0.98	18	.34
<i>M</i>	17.44	14.03				
<i>SD</i>	8.94	2.18				
Range	10.46–44.38	11.25–17.08				
Total improvement of patients experiencing sudden gains				1.77	13	.10
<i>M</i>	35.39	23.38				
<i>SD</i>	14.20	6.83				
Range	16.66–63.32	15.84–29.40				

Note. EGT = exposure group therapy; CBGT = cognitive-behavioral group therapy.

To further examine whether individuals with sudden gains in the two treatment groups differed in the magnitude of the LSAS treatment change, we conducted a 2 (time: pre vs. post) \times 2 (group: EGT vs. CBGT) \times 2 (sudden gains: individuals with sudden gains vs. those without) repeated measure ANOVA with the LSAS as the dependent variable. Results showed the expected time effect, $F(1, 71) = 113.34, p < .001$, partial $\eta^2 = .62$, Time \times Group effect, $F(1, 71) = 4.68, p = .03$, partial $\eta^2 = .06$, and Time \times Sudden Gain effect, $F(1, 71) = 11.56, p = .001$, partial $\eta^2 = .14$. The group effect, $F(1, 71) = 1.74, p = .19$, partial $\eta^2 = .02$, sudden gain effect, $F(1, 71) = 0.64, p = .43$, partial $\eta^2 = .01$,

Group \times Sudden Gain interaction, $F(1, 71) = 0.04, p = .84$, partial $\eta^2 = .001$, and Time \times Group \times Sudden Gain interaction, $F(1, 71) = 0.50, p = .48$, partial $\eta^2 = .01$, were not statistically significant. The latter result suggests that individuals with sudden gains showed similar improvements in the two treatment groups.

Because the sudden gain phenomenon has been studied primarily in the depression literature, we further examined whether the sudden gains were specifically associated with major depressive disorder (MDD), which was the most common comorbid condition in the present sample. Of the 33 participants who had a comorbid diagnosis of MDD, 9 (27%) experienced a sudden gain, whereas of

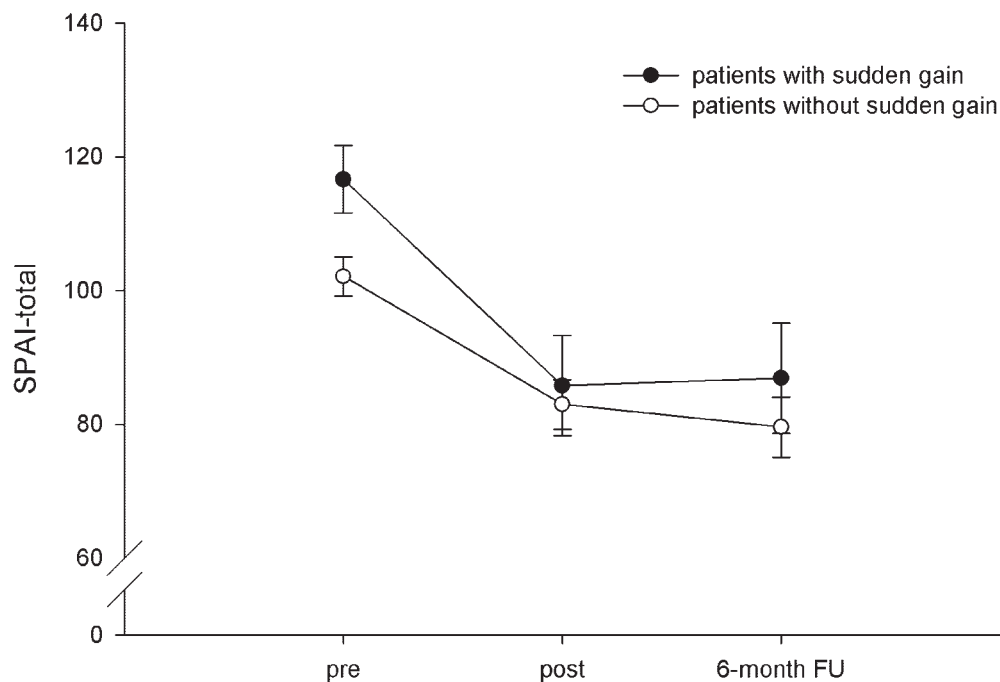


Figure 2. Mean change scores (with error bars representing standard errors) in Social Phobia and Anxiety Inventory (SPAI) scores from pretreatment (pre), to posttreatment (post), and to 6-month follow-up (FU) among individuals who showed sudden gains (black circles) and those who did not (white circles).

the 74 participants who did not have a MDD diagnosis, 11 (15%) experienced sudden gains. This difference was not statistically significant, $\chi^2(1) = 2.31, p = .13$. Of the 59 participants assigned to the EGT group, 20 (34%) had a comorbid MDD diagnosis, whereas of 48 participants assigned to the CBGT group, 13 (27%) had a comorbid MDD. This difference was not statistically significant, $\chi^2(1) = 0.58, p = .45$.

Cognitive Changes Preceding Sudden Gains

Changes in cognitions were measured with the SSPS-N (Hofmann & DiBartolo, 2000), which was administered together with the LSAS at the beginning of each session.⁵ We selected the SSPS-N because studies have shown that self-focused attention (Woody & Rodriguez, 2000; Woody et al., 1997) and in particular negative self-focused attention (Hofmann, 2000; Hofmann et al., 2004) enhances social anxiety and decreases with successful treatment.

To examine cognitive change prior to the sudden gains, we computed change scores of the cognitive measure from the beginning of the prepregain session ($N - 1$) to the beginning of the pregain session (N). We refer to this as *changes before pregain sessions*. We used the cognitive change from the session before the prepregain session ($N - 2$) to the beginning of the pregain session ($N - 1$) as a control interval (*prepregain change*).

A repeated measures ANOVA with group (EGT vs. CBGT) as the between factor and time (changes before pregain vs. changes before prepregain change) as the within-subjects factor revealed a significant main effect of group, $F(1, 16) = 6.25, p = .024$, partial $\eta^2 = .28$. CBGT was associated with greater cognitive change overall. However, the main effect of time, $F(1, 16) = 0.02, p = .88$, partial $\eta^2 = .00$, and the Time \times Group interaction were not significant, $F(1, 16) = 0.50, p = .49$, partial $\eta^2 = .03$.

To explore whether sudden gains were accompanied by cognitive changes in the pregain session and the interval between the pregain session and the aftergain session, we computed a repeated measures ANOVA with group (EGT vs. CBGT) as the between factor, and time (beginning of pregain session vs. beginning of aftergain session) as the within-subjects factor for the SSPS-N. This analysis showed no significant main effect of group, $F(1, 17) = 1.54, p = .23$, partial $\eta^2 = .08$, time, $F(1, 17) = 2.73, p = .117$, partial $\eta^2 = .14$, or Time \times Group interaction, $F(1, 17) = 0.62, p = .44$, partial $\eta^2 = .04$.

In addition to the SSPS-N, we also examined the ratio SSPS-P/(SSPS-P + SSPS-N). This ratio is derived from the states of mind model (Schwartz & Garamoni, 1989) and measures the balance between positive and negative thoughts. The results showed, again, a significant main group effect, $F(1, 16) = 5.86, p = .028$, partial $\eta^2 = .27$, but no time effect, $F(1, 16) = 1.88, p = .19$, partial $\eta^2 = .11$, or Time \times Group interaction, $F(1, 16) = 0.38, p = .549$, partial $\eta^2 = .02$. As expected, participants in the CBGT group showed greater treatment changes ($M = 0.43, SD = 0.13$) than participants in the EGT group ($M = 0.34, SD = 0.29$). Analyses of the SSPS-P revealed no significant main effect of group, $F(1, 16) = 3.23, p = .091$, partial $\eta^2 = .17$, time, $F(1, 16) = 2.41, p = .140$, partial $\eta^2 = .13$, or Time \times Group interaction, $F(1, 16) = 1.13, p = .304$, partial $\eta^2 = .07$. Similar results were found when examining changes in the SSPS-P and the ratio SSPS-P/(SSPS-P + SSPS-N) from the beginning of the pregain to the beginning of the aftergain session.

Discussion

Sudden gains are enduring reductions in symptom intensity from one session to the next, which have, thus far, exclusively been studied in the treatment of depression (Gaynor et al., 2003; Hardy et al., 2005; Tang & DeRubeis, 1999b; Tang et al., 2002; Tang et al., 2005; Vittengl et al., 2005). It remains uncertain whether the sudden gain phenomenon can also be observed in disorders other than depression. Moreover, the factors that determine these gains remain largely unexplored. Some authors assume that the therapeutic strategies in cognitive-behavioral therapy that encourage cognitive change are responsible for sudden gains in treatment (e.g., Hollon, 1999; Tang & DeRubeis, 1999a). This is consistent with the cognitive model, which assumes that changes in cognitions precede symptom improvements in therapy. In contrast, other authors assume that common factors, rather than specific cognitive factors, are responsible for sudden gains (Ilardi & Craighead, 1994, 1999; Lambert, 2005). For example, Ilardi and Craighead (1994) argued that most of the symptom improvement in cognitive-behavioral therapy for depression (60%–70%) appears to occur during the first 4 weeks of treatment and cannot, therefore, be explained by cognitive modification. The objective of this study was to examine the phenomenon of sudden gains during cognitive-behavioral therapy and exposure therapy for social phobia. We expected that individuals receiving cognitive-behavioral therapy would show more frequent sudden gains. We further predicted that these gains would be preceded more often by changes in cognition in individuals receiving cognitive-behavioral therapy compared with those receiving exposure therapy without explicit cognitive interventions.

Consistent with the depression literature, our results showed that some treatment sessions are characterized by relatively large improvements in social anxiety from one session to the next in a subgroup of individuals. However, sudden gains were not more commonly associated with cognitive-behavioral intervention and changes in cognitions. It should be noted, however, that these results cannot rule out the possibility that important cognitive changes did occur during the pregain sessions. The measurement interval for cognitive changes in our study was from the beginning of the prepregain session to the beginning of the pregain session. In contrast, Tang and DeRubeis (1999b) emphasized the importance of examining in-session cognitive changes within the pregain session. Moreover, the degree of cognitive intervention was not the only distinguishing characteristic between the two study groups. The treatments also differed in the treatment rationale participants received and the type of exposure situations participants underwent (EGT exposures consisted of public speaking tasks, whereas CBGT exposures consisted of a number of different situations). Therefore, future studies should examine cognitive changes within the pregain session, perhaps by designing a questionnaire that asks patients about cognitive changes within that session and uses an additive design to examine the specific effects of cognitive interventions on sudden gains.

To define, identify, and investigate sudden gains, we closely followed the methodology by Tang and DeRubeis (1999b), who

⁵ This procedure to measure cognitive change differed from the method used by Tang and DeRubeis (1999b), who measured cognitive change by rating audiotaped recordings of the pregain sessions.

first described this phenomenon. For this reason, we primarily limit the comparison between our results and the findings from the depression literature to the original Tang and DeRubeis study. The cutoff score to define a sudden gain in Tang and DeRubeis' study accounted for an average of 51% of the total symptom reduction in LSAS scores. Similarly, the cutoff score we chose to define a sudden gain accounted for 50.27% overall improvement among those participants who experienced sudden gains. We observed 22 sudden gains out of 967 between-session intervals (2.23%). This is consistent with Tang and DeRubeis' study, which found 29 sudden gains out of 927 between-session intervals (3.13%). Tang and DeRubeis further reported that the median pregain session of a 16–20 session course of individual cognitive–behavioral therapy for depression occurred in Session 5. Similarly, we observed that most sudden gains of a 12-session group treatment occurred in Session 5. In case this finding is reliable, future research should explore whether the fifth session contains any unique characteristics. Finally, consistent with the study by Tang and DeRubeis, sudden gains were not associated with better outcomes at follow-up.

In addition to these similarities, we also observed a number of important differences in our results compared with those reported by Tang and DeRubeis (1999b). First, the percentage of patients experiencing a sudden gain in our sample was lower than what was found in the Tang and DeRubeis study (18.67% vs. 39.34%). However, both percentages are within the range of what would be expected on the basis of the sudden gains literature, which showed that between 17% and 50% of clients experience sudden gains (Gaynor et al., 2003; Hardy et al., 2005; Stiles et al., 2003; Tang et al., 2002, 2005; Vittengl et al., 2005). Second, we only identified 2 patients (1.9%) with a reversal of sudden gains. In contrast, Tang and DeRubeis found that 17% of their sample experienced a reversal in sudden gains. Other studies identified between 9% (Gaynor et al., 2003) and 47% (Tang et al., 2002) of individuals with reversed sudden gains. This may suggest that individuals with social phobia show a more stable rate of improvement as compared with individuals with depression. It is further possible that the group format in which the social phobia treatments were conducted had a moderating and stabilizing effect on the treatment gains. It should be noted, however, that the probability of experiencing a sudden gain did not significantly vary across the treatment groups. Third, Tang and DeRubeis (1999a) reported that participants with sudden gains had better outcome at posttreatment, 6-month, and 18-month follow-up assessments. In contrast, we observed no differences in outcome between individuals with and without sudden gains at the posttreatment or 6-month follow-up assessments. This finding is in line with some studies (Tang et al., 2002; Vittengl et al., 2005) but at odds with other reports (Gaynor et al., 2003; Hardy et al., 2005).

It is interesting that we found that participants with sudden gains had higher levels of pretreatment social anxiety. Similarly, previous studies on sudden gains during the treatment for depression also observed higher levels of pretreatment symptomatology in individuals who later experienced sudden gains (e.g., Vittengl et al., 2005). This may provide an alternative explanation for the nature of the sudden gain phenomenon. Specifically, it is possible that our (or any other) methodology to identify participants with sudden gains simply selects individuals who are more severe at pretreatment. Because of regression to the mean, those individuals

are expected to show greater gains before the end of treatment than the less severe participants.

In sum, the results of this study revealed a number of similarities to the depression literature with regards to the magnitude and frequency of sudden gains. However, the sudden gains in this study did not predict better short-term or long-term treatment outcome, were not correlated with a particular treatment modality, and were not associated with measurable changes in cognitions. These findings question the external validity of the sudden gain phenomenon. In fact, the most parsimonious explanation for the phenomenon in the present study is that participants simply show clinically nonsignificant fluctuations in their symptomatology during therapy.

Limitations of the study include the sole reliance on self-report measures to assess cognitive changes and social anxiety. Furthermore, we only examined cognitions pertaining to negative self-perception. Although there is some justification in the social phobia literature to primarily concentrate on self-perception, other cognitions, especially those related to estimated social cost (Foa, Franklin, Perry, & Herbert, 1996; Hofmann, 2004; Wilson & Rapee, 2005) and emotional control (Hofmann, 2005), may prove to be more important than changes in self-perception. Finally, the study was not a randomized, controlled trial and participants were primarily Caucasians with relatively high socioeconomic status, which limits the generalizability of the study findings. Finally, one of the most significant problems in comparing our study with previous work in sudden gains involves the cutoff score used to define a sudden gain (Criterion 1 of the sudden gain criteria). This is an arbitrary score that is dependent on the measuring construct, the measuring instrument, and the sample characteristics. However, it is not unusual in the clinical literature to choose different cutoff scores of a construct for different patient groups. For example, the expressed emotions literature accepts different cutoffs for high and low expressed emotions in individuals with depression and schizophrenia.

Despite these limitations, this study points to an important area of future inquiry: the empirical analysis of the psychotherapy process. Consistent with the depression literature, sudden gains in individuals who underwent group therapy for social phobia were identified. Although CBGT was associated with more cognitive changes and lower levels of social anxiety at follow-up than EGT, the session-by-session data provided little support for the notion that cognitive changes produce sudden gains. Therefore, the cognitive model is either inadequate to explain sudden gains during treatment for social phobia and/or sudden gains are not associated with cognitive changes. Alternatively, it is possible that changes in cognitions do, in fact, precede sudden gains. However, this effect might be delayed, and cognitions immediately preceding the sudden gain only poorly predict those gains. In fact, Tang and DeRubeis (1999b) suggested that the pregain session is the critical session in which the therapeutic breakthrough occurs. Furthermore, it is possible that changes in cognitions did precede the sudden gains in the present study but that the instruments to assess these changes were inadequate. In either case, the validity of the sudden gain phenomenon remains questionable unless it can be demonstrated that this rare occurrence has any clear clinical significance.

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Call for Papers: Special Section on Suicide and Self-Harm Behaviors

The *Journal of Consulting and Clinical Psychology* is requesting submissions of empirical papers that focus on suicide and self-harm behaviors, including non-suicidal self-injury. In particular, submissions are requested that may address one of the following topics: (1) beyond the identification of broad biopsychosocial risk factors, what are possible specific mechanisms that promote self-harm behavior, and might be addressed in prevention/intervention efforts? (2) how might cross-disciplinary theoretical perspectives (e.g., biological, interpersonal) be integrated to understand or treat self-harm behavior? (3) what are some innovative methodological paradigms for investigating self-harm behaviors? (4) randomized clinical trial data on preventions/ interventions designed to reduce self-harm behavior. The papers must present original empirical findings. The goal of this special section is to have a set of papers that represent the lifespan.

The deadline for submissions of manuscripts is February 1, 2007. Final editorial decisions will be made by late 2007, with an anticipated publication date of early 2008. All submissions should be entered through the main submission portal for the journal (www.apa.org/journals/ccp.html). Authors should indicate in their accompanying cover letter that the paper is to be considered for the special section on “suicide and self-harm.” All submitted papers must be in APA format and conform to the all the guidelines for submission for JCCP (see www.apa.org/journals/ccp).

Questions or inquiries regarding the special section should be directed to the section editor, Mitch Prinstein (mitch.prinstein@unc.edu).