Sudden Gains and Critical Sessions in Cognitive–Behavioral Therapy for Depression

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In this study of cognitive–behavioral therapy for depression, many patients experienced large symptom improvements in a single between-sessions interval. These sudden gains' average magnitude was 11 Beck Depression Inventory points, accounting for 50% of these patients' total improvement. Patients who experienced sudden gains were less depressed than the other patients at posttreatment, and they remained so 18 months later. Substantial cognitive changes were observed in the therapy sessions preceding sudden gains, but few cognitive changes were observed in control sessions, suggesting that cognitive change in the pregain sessions triggered the sudden gains. Improved therapeutic alliances were also observed in the therapy sessions immediately after the sudden gains, as were additional cognitive changes, suggesting a three-stage model for these patients' recovery: preparation → critical session/sudden gain → upward spiral.

Since its introduction in the 1970s, cognitive–behavioral therapy (CBT) for depression has become one of the most widely investigated and practiced forms of therapy for depression (DeRubeis & Crits-Christoph, 1998). However, there are still more questions about the mechanisms of change in CBT—that is, how does CBT alleviate depression? Originally, Beck, Rush, Shaw, and Emery (1979) proposed that CBT reduces depressive symptoms by modifying the patients' cognitive processes. Although the cognitive mediation hypothesis has historically dominated this field (see Whisman, 1993), several authors have also proposed that therapeutic alliance and other nonspecific factors play important roles in CBT's mechanism (e.g., Burns & Nolen-Hoeksema, 1992). After reviewing empirical studies investigating these hypotheses, Whisman (1993) concluded that, although most empirical findings are consistent with the cognitive mediation hypothesis, this hypothesis "should be viewed as tentative" (p. 258) because of the limitations of existing studies.

In their influential article, Ilardi and Craighead (1994) introduced the analysis of time course to the field, and their finding appeared to be in direct contradiction to the cognitive mediation hypothesis. They observed that in many CBT efficacy studies, 60% to 70% of total improvement on measures of depression

symptom severity occurred in the first 4 weeks of therapy. The authors assumed that the first 4 weeks in CBT are occupied with activities unrelated to cognitive modification and thus concluded that the 60% to 70% of total symptom improvements cannot be explained by cognitive modification. Their intuitively appealing argument casts serious doubt on the validity of the cognitive mediation hypothesis.

However, as we have argued elsewhere, Ilardi and Craighead's (1994) inferences may be premature. Most relevant to the present article is our observation of substantial heterogeneity of time courses across individual patients, which makes it hazardous to infer mechanism from the group mean time course (see Tang & DeRubeis, 1999a, for a more complete discussion). Despite our disagreements, we believe that Ilardi and Craighead made an important contribution by highlighting the potential of time course analysis for mechanism research.

·In the present project, we aimed to advance their approach by extending time course analysis to data from individual patients. As a general rule, the shapes of individual patients' time courses often differ substantially from that of the group mean time courses and the group mean time courses often do not exhibit orderly patterns shared by many individuals (cf. Bakan, 1954; Estes, 1956; Sidman, 1952; Tang & DeRubeis, 1999a). By investigating the time course of individual patients, this project might detect meaningful patterns that are shared by many individual patients, even if these patterns are not revealed in the group mean time course.

For these reasons, session-by-session depression severity data of individual CBT patients were obtained from the Treatment of Depression Collaborative Research Program (Elkin et al., 1989) and from the Hollon et al. (1992) study, and the time courses of each patient were plotted. In these plots, a remarkable pattern emerged: Many individual patients' depression severity improved suddenly, in some cases dramatically, in one between-sessions interval. The magnitudes of these improvements were much larger than those of typical between-sessions symptom reductions. They also seemed to account for a surprisingly large portion of these

patients' total symptom improvements. We termed these sudden, substantial symptom improvements *sudden gains*.

Given the sudden gains' large magnitude, important insights into CBT's mechanisms might result from an understanding of their causes. The existence of sudden gains raises several questions. Do they represent transient noise, or do they represent a long-lasting decrease in patients' depression severity? Are they "random" mood swings, or are they triggered by important therapeutic breakthroughs?

If sudden gains are triggered by therapeutic breakthroughs in the preceding therapy sessions (the pregain sessions), then these sessions might be the "critical" sessions. Several psychologists have hypothesized that a small number of critical sessions have a much greater impact on outcome than other therapy sessions (Elliott, 1983, 1984; Greenberg, 1977; see also Lambert, DeJulio, & Stein, 1978). However, the existence of critical sessions in a large clinical sample has never been convincingly demonstrated, and psychologists have yet to find a convenient way to identify these critical sessions.[1]

Intrigued by these possibilities, we designed a set of quantitative criteria to identify the sudden gains. We then investigated three aspects of this phenomenon. First, we tried to determine whether sudden gains represent transient noise, as opposed to long-lasting symptom improvement that contributes to ultimate treatment outcome. These results are presented in Part I: Sudden Gains and Outcome. Second, we searched for possible causes of sudden gains in the pregain sessions. These results are presented in Part II: Phenomena Observed in the Critical Session. Third, we examined the sudden gains' impact on later therapy sessions, especially on the therapeutic alliance and additional cognitive changes; these results are presented in Part III: Phenomena Observed After Sudden Gains. In the final discussion, our findings are integrated into a three-stage model of CBT for sudden-gain responders.

Part I: Sudden Gains and Outcome

Method

Data source.   Data were obtained from two efficacy studies: the Elkin et al. (1989) study and the Hollon et al. (1992) study, which were chosen because the audiotapes of all of the sessions, as well as presession Beck Depression Inventory (BDI) scores, were made available to us.

In both studies, the patients were clinically depressed outpatients, and the CBT treatment followed the procedure outlined in Beck et al.'s (1979) treatment manual. The patients received 2 CBT sessions per week for the first 4 to 8 weeks, followed by 1 session per week until the end of treatment. The treatment lasted up to 20 sessions in both studies, over a 12-week period in the Hollon et al. (1992) study and over a 16-week period · in the Elkin et al. (1989) study. Follow-up evaluations were conducted every 6 months, for 18 months in the Elkin et al. study and for 24 months in the Hollon et al. study.

There were 25 patients assigned to CBT in the Hollon et al. (1992) study and 59 in the Elkin et al. (1989) study. From this combined sample, we excluded 23 patients who either had fewer than eight sessions of therapy or had a first-session BDI score of less than 15 points. This was done to exclude patients who received little CBT treatment, as well as those who were already only mildly depressed by the time of the first session.

Measurement.   The BDI (Beck & Steer, 1987) was used as the primary measure of depressive symptomatology because it was administered prior to each session in both the Elkin et al. (1989) study and the Hollon et al. (1992) study. The BDI score at any session *N* reflects the patient's report

of depressive symptom severity between session $N - 1$ and session N. Thus, it is not affected by therapy conducted in session N.

Terminology.   We refer to the therapy session immediately preceding the sudden gain as the *pregain session* and the therapy session immediately after the gain as the *after-gain session*. In addition, the therapy session preceding the pregain session is referred to as the *prepregain session*. Thus, the temporal sequence is as follows: prepregain session $(N - 1) \rightarrow$ pregain session $(N) \rightarrow$ sudden gain $\rightarrow$ after-gain session $(N + 1)$.

Results

Identification and description of sudden gains.   The magnitude of a sudden gain should be large (a) in absolute terms, (b) relative to depressive symptom severity before the gain, and (c) relative to symptom fluctuations preceding and following the gain. .

These considerations were instantiated in three criteria. A sudden gain occurred between session N and session $N + 1$ if (a) the gain was at least 7 BDI points ($BDI_N - BDI_{N+1} \geq 7$); (b) the gain represented at least 25% of the pregain session's BDI score ($BDI_N - BDI_{N+1} \geq 0.25 \times BDI_N$); and (c) the mean BDI score of the three therapy sessions before the gain (sessions $N - 2$, $N - 1$, and N) was significantly higher than the mean BDI score of the three therapy sessions after the gain (sessions $N + 1$, $N + 2$, and $N + 3$) using a two-sample t test, with an alpha of .05.[2]

The cutoff value of 7 was somewhat arbitrary, but there are some empirical justifications for it. In both the Elkin et al. (1989) study and the Hollon et al. (1992) study, the frequency distribution plots of between-sessions BDI score changes appeared to exhibit a secondary peak, which began at 7 BDI points and peaked at 9 BDI points. In addition, we observed a nearly identical secondary peak at 9 BDI points in the data set of Murphy, Simons, Wetzel, and Lustman (1984) and a similar secondary peak at 8 BDI points in the data set of Jacobson et al. (1996; see Tang & DeRubeis, 1996). Thus, between-sessions BDI changes greater than 7 points may be qualitatively different from the smaller ones.[3] Criterion c essentially compares the magnitude of the BDI scores' decrease with the BDI scores' variance before and after the change. Thus, moderate decreases accompanied by large variations will not pass this criterion, nor will extremely short-lived decreases in symptoms. For example, a BDI sequence of 21, 20, and 20 versus 9, 21, and 20 will be screened out by this criterion.

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[1] Elliott (1984) described how he identified a critical session in 1 patient's therapy process. His method appeared to be sophisticated and scientifically well justified, although it is probably too complicated and costly for most researchers to implement, especially in a larger population.

[2] This criterion made it difficult to evaluate changes in depressive symptomatology following the first session or before the last session because it required at least two BDI scores before and after the symptom changes. Thus, all of the symptom changes following the first sessions were excluded from further analysis. First sessions are so different from other sessions that they probably should be excluded in the first place. For between-sessions decreases right before the last session, we obtained the BDI score of the week after the last session and used that score as a substitute for BDI_{N+2} in calculating Criterion c.

[3] We experimented with other cutoffs (e.g., 6 and 8 BDI points). We detected no qualitative changes in our results vis-à-vis outcome, and the effects we detected were limited and predictable (e.g., using a 6-point cutoff yielded a few more sudden gains with slightly smaller magnitudes, whereas using an 8-point cutoff yielded slightly fewer sudden gains with greater magnitudes).

Out of the 927 between-sessions intervals of the 61 patients, our criteria selected 29 sudden gains experienced by 24 different patients. Although sudden gains were observed throughout therapy, the median 50% fell between the 4th and the 10th sessions, with the 5th session being the median and the mode pregain session.

The "average" sudden gain is shown in Figure 1. We averaged all sudden gains' session N (pregain session) BDI scores and repeated this process for sessions $N - 2, N - 1, N + 1, N + 2$, and $N + 3$. To put these means in context, we also display in Figure 1 the average first- and last-session BDI scores of all the patients who experienced sudden gains.

Impact of the sudden gains: Immediate, short term, and long term. The mean magnitude of all identified sudden gains was 11.2 BDI points ($SD = 4.4$). The mean of the total BDI improvement accomplished by the entire CBT treatment was 15.4 points for all of the patients ($SD = 13.0$) and 21.8 points for the patients who experienced sudden gains ($SD = 8.6$).[4] Thus, for patients who experienced sudden gains, the sudden gains accounted for an average of 51% of their total symptom reduction.

One might suspect that the sudden gains represent transient noise, so that the severity of these patients' depressive symptomatology would bounce back to the pregain level in a short period of time. To examine this possibility, we measured how often such reversals occurred before the end of therapy. To be stringent, whenever a patient gave up 50% of the symptom improvement resulting from the sudden gain, we counted it as a reversal. For example, if a patient's pregain session BDI score was 30 and after-gain session BDI score was 20, then whenever his or her BDI

score returned to 25 or higher, it counted as a reversal. Out of the 24 patients who experienced sudden gains, only 4 patients experienced a reversal before the end of therapy.

At posttreatment, patients who had experienced sudden gains enjoyed very good outcomes. Their mean posttreatment BDI score was 5.9 ($SD = 5.6$). When recovery is defined as a posttreatment BDI less than 10 (the criterion used in both the Elkin et al., 1989, study and the Hollon et al., 1992, study), 79% (19/24) of these patients recovered.

These patients' outcomes were also significantly better than the outcomes of patients who did not experience sudden gains (see Figure 2). At the beginning of therapy, the two groups of patients evidenced essentially equal levels of depressive symptomatology: The mean BDI score was 27.7 for patients who later experienced sudden gains ($n = 24, SD = 5.8$) and 27.9 for those who did not ($n = 37, SD = 7.9$). This difference was not significant, $t(59) = 0.11, p = .91$, with a very small effect size (Cohen's $d = 0.03$; Cohen, 1977). At posttreatment, the mean BDI score of the patients who did not experience a sudden gain was 16.8 points ($SD = 13.0$), which was significantly worse than the mean (5.9) for the patients who experienced a sudden gain, $t(59) = 3.86, p < .0003$. The effect size of the difference was substantial ($d = 1.00$). It should also be noted that the magnitude of the difference in posttreatment scores (10.9) is almost identical to the average magnitude of the sudden gains (11.2). Thus, it appears that the relative benefit that accrued to patients with sudden gains was maintained through the end of treatment.

In addition, the recovery rate of the patients who did not experience sudden gains was 41% (15/37), which was significantly lower than the 79% (19/24) recovery rate of the patients who experienced sudden gains, $\chi^2(1, N = 61) = 9.24, p = .02$. Also, 56% (19/34) of the treatment responders had experienced sudden gains. Thus, understanding sudden gains might be important for understanding at least how the majority of responders achieved recovery.

As shown in Figure 2, during the follow-up, the size of the difference between patients with sudden gains and those without sudden gains decreased, but it was still significant at two of the three follow-up assessment points: for 6 months, $Z(25) = 2.41, p = .02$; for 12 months, $Z(25) = 0.31, p = .98$; and for 18 months, $Z(25) = 2.11, p = .03$.[5] This suggests that the benefits of the sudden gains were not temporary and that they might have long-lasting effects on patients' symptom severity.

We also compared the long-term outcomes of the two groups of responders: the sudden-gain responders and the no-sudden-gain responders. The two groups of responders showed equally low levels of depressive symptomatology at posttreatment. If symptom improvements from sudden gains are not as stable as symptom improvements without sudden gains, the sudden-gain responders
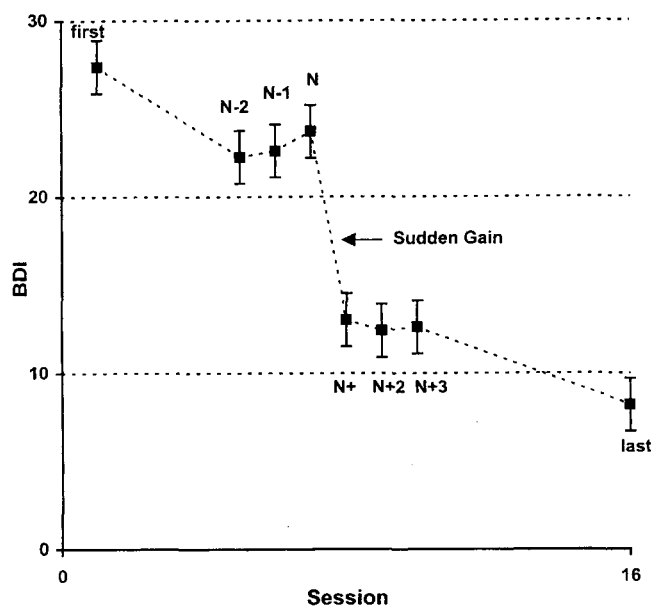
Figure 1. The average sudden gain. The Beck Depression Inventory (BDI) scores shown for sessions $N - 2, N - 1, N, N + 1, N + 2, N + 3$ are the means ($\pm SE$) of the corresponding sessions from the 29 sudden gains in our sample, with session N as the pregain session. To provide the right context, the figure also shows the means ($\pm SE$) of BDI scores from the first and last sessions for all of the patients who experienced sudden gains.

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[4] These numbers are calculated by deducting first-session scores from the last-session scores, which is similar but not identical to deducting pretreatment scores from posttreatment scores. The symptom changes that occurred before the first session and after the last session are therefore not included in these means.

[5] The Wilcoxon rank sums test was used here because the distribution of the BDI scores was very skewed, with many outliers (most patients' BDI scores were close to 0, but several patients' scores were greater than 25).
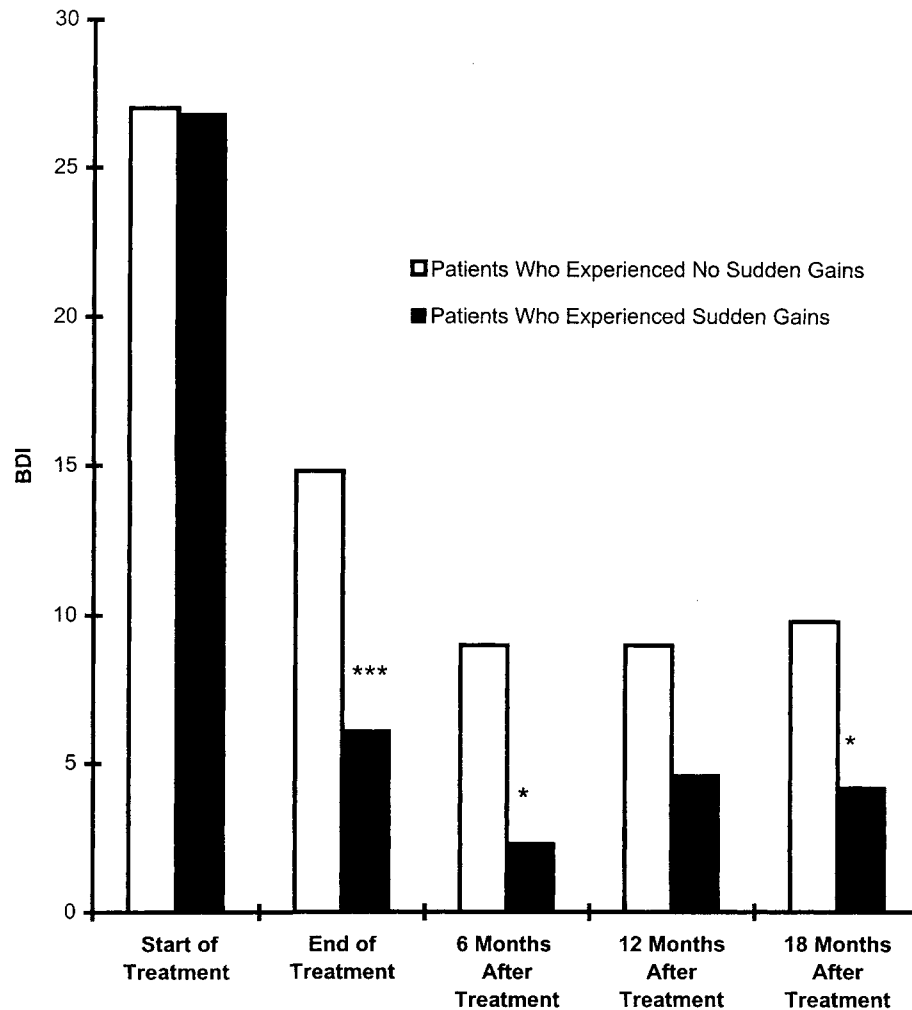
Figure 2. The outcomes of the patients who experienced sudden gains compared with those who did not experience sudden gains. BDI = Beck Depression Inventory. $^*p < .05.$ $^{***}p < .001.$

should have become more depressed than the no-sudden-gain responders during the follow-up period.

As Figure 3 shows, although at 12 months posttreatment the two groups evidenced nearly equal levels of depressive symptomatology, at 6 and 18 months posttreatment the sudden-gain responders were significantly less depressed than the no-sudden-gain responders: for 6 months, $Z(25) = 2.41$, $p = .02$; for 12 months, $Z(25) = 0.31$, $p = .98$; and for 18 months, $Z(25) = 2.11$, $p = .03$. There was no significant difference between the relapse rates of these two groups, $\chi^2(1, N = 27) = 0.11$, $p = .74$.

Discussion

The sudden gains occurred in more than 50% of the CBT responders and accounted for more than 50% of these patients' total symptom improvements. Sudden gains also seem to represent a stable short-term symptom improvement because the depression severity of the patients only infrequently bounced back after the sudden gain. As a result, the patients who experienced sudden gains enjoyed superb treatment outcomes. Their outcomes were

also significantly better than those of patients who did not experience sudden gains at posttreatment, as well as 6 months and 18 months after treatment. Moreover, the long-term outcomes of the sudden-gain responders were at least as good as those of the no-sudden-gain responders.[6] Thus, our results clearly suggest that sudden gains are not transient and inconsequential fluctuations. Instead, they seem to be a part of substantial, long-lasting symptom improvement.

Part II: Phenomena Observed in the Critical Session

In this part of the project, we explored potential determinants of sudden gains. Among the many possibilities, four therapeutic factors in the pregain sessions were investigated: (a) patients' cognitive changes, (b) therapists' application of concrete CBT

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[6] The reason for the instability of the advantage to the patients with sudden gains is not clear. Future studies can help to resolve whether this was due to random variability in this sample.
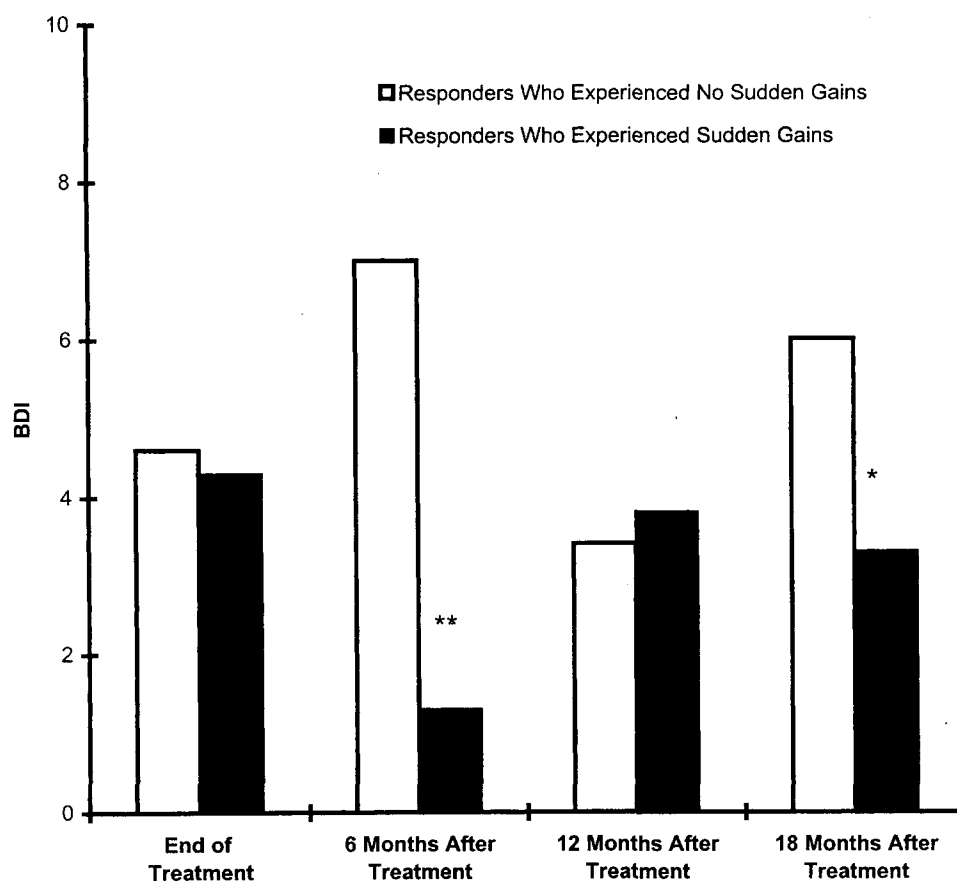
Figure 3. The long-term outcome of sudden-gain responders and no-sudden-gain responders as measured by the Beck Depression Inventory (BDI) 6, 12, and 18 months after the end of treatment. $^*p < .05$. $^{**}p < .01$.

techniques, (c) therapists' application of abstract CBT techniques, and (d) the therapeutic alliance between the therapist and the patient.

Possible cognitive changes include belief changes, schema changes, and the learning of new cognitive techniques. In several studies, patients' cognitive changes during a whole course of CBT have correlated positively with treatment outcomes (e.g., Blackburn & Bishop, 1983; DeRubeis et al., 1990; Seligman et al., 1988; see Whisman, 1993, for a review). DeRubeis et al. also found that cognitive changes in the early half of therapy predicted later changes in symptoms. In the time frame of a single session, there are fewer empirical studies, and results have been less consistent. For example, Persons and Burns (1985, 1986) reported that changes in automatic thoughts within a session correlated with within-session mood changes, but Safran et al. (1987) reported that "cognitive shifts" were unrelated to within-session changes in depressive symptomatology and did not predict the changes in depressive symptomatology following the session.

Therapists' application of concrete CBT techniques concerns the concrete and pragmatic aspects of CBT, in which the therapists use CBT techniques in an attempt to change patients' beliefs (DeRubeis & Feeley, 1990). Therapists' application of abstract CBT techniques refers to the more philosophical and theoretical aspects of CBT, in which the therapist and patient discuss the

cognitive theory of depression and the cognitive mediation hypothesis of CBT and explore the schemas behind beliefs. DeRubeis and Feeley reported that CBT therapists' application of concrete CBT techniques in the second therapy session predicted later symptom improvement but that the application of abstract CBT techniques did not. Feeley, DeRubeis, and Gelfand (1999) have replicated this result.

The above three factors are all closely related to the cognitive mediation hypothesis of CBT, although from different perspectives. The therapists' application of abstract or concrete CBT techniques focuses on how much time and effort therapists spend attempting to produce cognitive change; the patients' cognitive change concerns, in essence, how successful those attempts have been.

Therapeutic alliance refers to the collaborative relationship formed between the therapist and the patient. It has traditionally been considered one of the central ingredients in psychodynamic therapy, and more recently, researchers have examined its role in CBT (e.g., Castonguay, Goldfried, Wiser, Raue, & Hayes, 1996; Gaston, Thompson, Gallagher, Cournoyer, & Gagnon, 1998; Safran & Wallner, 1991). In many studies, the therapeutic alliance has been found to correlate positively with treatment outcome (for a review, see Horvath & Symonds, 1991; see also Feeley et al., 1999), but in two studies, the alliance has not been found to predict

subsequent treatment outcome (DeRubeis & Feeley, 1990; Feeley et al., 1999).

To explore which factors might have triggered the sudden gains, we measured these factors in the pregain session as well as in a within-subject control session for every patient who experienced a sudden gain. If the level of an observed variable in the pregain session was higher than its level in the control session, then that factor might have helped to trigger the sudden gains. The prepregain session was chosen as the within-subject control session for these reasons: (a) the pregain session and the prepregain session were close in time and (b) because both sessions occurred before the sudden gain, the patient's initial state in the prepregain session should, on average, more closely resemble his or her initial state in the pregain session than it would in any other session.

Method

Data sources. Data were collected by rating audiotape recordings of relevant therapy sessions from the Elkin et al. (1989) and Hollon et al. (1992) studies. If a patient had more than one sudden gain, only the first sudden gain was assessed, so that each observation was independent, as it represented 1 unique patient. We did not have access to the therapy session recordings for 8 of the 24 patients with sudden gains, leaving 16 patients available for further analysis.

Procedures. Each audiotape was rated independently by two raters; the average of the two raters' ratings was used as the final score. One primary rater rated all of the sessions, and three secondary raters each rated one third of the sessions. The primary rater was a clinical psychology graduate student with 3 years of experience rating CBT sessions; the three secondary raters were undergraduate students with 6 months of training in rating CBT sessions. To minimize the impact of interrater differences, we always assigned the pregain session and the prepregain session of a given therapist–patient dyad to a single secondary rater. The assignment to secondary raters was also balanced in terms of the data source (the Elkin et al., 1989, study vs. the Hollon et al., 1992, study) and therapist within study. The raters were unaware of the identity of the patients and therapists, session numbers, symptom changes before and after the session, and eventual outcomes.

Measurements. To measure the extent of reported cognitive changes, we designed the Patient Cognitive Change Scale (PCCS).[7] The PCCS includes seven categories of possible cognitive changes: (a) bringing a belief into awareness, (b) identifying an error in cognitive process or belief, (c) arriving at a new belief on a specific issue, (d) bringing a schema into awareness, (e) identifying an error in a schema, (f) arriving at a new schema, and (g) accepting a new cognitive technique.

The ratings were completed while the raters listened to the session audiotape. Whenever the rater heard the patient explicitly acknowledge any of the above events, he or she would classify the event by its content and score its significance according to how the patient reported the event. The event's significance was rated on a 4-point scale (1 = *a possible/potential cognitive change*, 2 = *a definite cognitive change*, 3 = *an important cognitive change*, 4 = *a cognitive change with extraordinary personal significance*). For example, when 1 patient realized that her belief that she was unattractive because her boyfriend had left her was false, she emphatically declared, "I can't believe I actually thought that! How could I let him decide if I am attractive or not?" On hearing this, the rater noted a cognitive change of Category b ("identifying an error in cognitive process") and gave this event a significance score of 3 ("an important cognitive change").

Raters were instructed never to infer cognitive changes but instead to rate only those explicitly reported by the patient. For instance, if the patient in the example above merely reported that she was hopeful about dating other men, the rater would not rate that as a change in her belief about *being unattractive*.

Any of the seven categories of cognitive changes could be used more than once in a given session, if more than one cognitive change of the same category occurred, as long as they differed in content. For example, if the patient in the above example also realized that she had set an unreasonably high standard for her job performance, she would get an additional score in Category b. However, if the same progress was acknowledged more than once, only one rating was given. At the end of the session, all of the scores for all of the categories were summed up as a measure of the magnitude of cognitive changes in the session.

The average interrater correlation coefficient for the scale was .50. Because we used the pooled ratings of the two raters as the final score, the composite reliability of the final score was estimated to be .67 (Allen & Yen, 1979). As it is now, the final composite score is not unacceptable for this type of research, especially in an exploratory phase, but it will require improvement if it is to be used in future studies.

The Collaborative Study Psychotherapy Rating Scale (Hill, O'Grady, & Elkin, 1992; Hollon et al., 1988) was used to assess the therapists' application of CBT techniques. DeRubeis and Feeley (1990) factor analyzed these items into two groups, one that assesses concrete CBT techniques (CT–Concrete) and another that assesses abstract CBT techniques (CT–Abstract). They reported pooled interrater reliability estimates of .63 for CT–Concrete and .86 for CT–Abstract. In our sample, the interrater correlation coefficient was .60 for CT–Concrete and .45 for CT–Abstract. The composite reliability of the two raters' average score was estimated to be .75 for CT–Concrete and .62 for CT–Abstract (Allen & Yen, 1979).

We used three scales to measure therapist–patient alliance: the CBT version of the Vanderbilt Therapeutic Alliance Scale (VTAS; Hartley & Strupp, 1983), the observer version of the Working Alliance Inventory (WAI; Horvath & Greenberg, 1986), and the Penn Helping Alliance Scale (Morgan, Luborsky, Crits-Christoph, Curtis, & Solomon, 1982). In our sample, the interrater correlation coefficient for the three scales was .70, .76, and .60, respectively. The composite reliability of the two raters' average score was estimated to be .82, .86, and .75, respectively (Allen & Yen, 1979).

Data analysis. Because the distribution of cognitive changes in the control sessions was highly skewed, the median was a better index of central tendency in our sample. However, paired t tests were still appropriate for comparing the pregain session against the control session because the difference scores—the amount of cognitive changes in the pregain session minus the amount of cognitive changes in the control session—still showed a normal distribution (Shapiro–Wilk's $w = .94$, $p = .43$; Cohen, 1977; Shapiro & Wilk, 1965).

Results

Cognitive change. Greater cognitive change was accomplished in the pregain sessions (*Mdn* = 5.0) than in the control sessions (*Mdn* = 1.5). A paired t test showed the difference to be statistically significant, $t(15) = 2.51$, $p < .05$, and the effect size was substantial ($d = 0.65$). On average, two "possible/potential" cognitive changes and one "definite" or "important" cognitive change occurred in the pregain sessions. In contrast, only one half of a possible/potential cognitive change and one third of a definite or important cognitive change occurred, on average, in the prepregain sessions.

Other therapeutic factors. Paired t tests showed that the pregain sessions and the control sessions did not differ significantly on any of the other measured factors, all $ts(15) < 1.40$, all $ps \geq .20$, all $ds \leq 0.30$.

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[7] Interested readers may contact Tony Z. Tang or Robert J. DeRubeis for a copy of the scale and more detailed instructions.

Discussion

A substantial amount of cognitive change was observed in the pregain sessions, whereas little cognitive change was noted in the control sessions. The pregain sessions and the control sessions did not differ on any of the other measured factors, such as the application of CBT techniques or the therapeutic alliance. Thus, among the variables investigated in the pregain sessions, cognitive changes were the most likely triggers of the sudden gains. Other variables that were not investigated, such as biochemical changes, life events, patients' readiness for change, and completion of homework, might have contributed to the sudden gains, but they could not be assessed in retrospect.

Together, the results from Parts I and II associate in-session reports of cognitive changes with sudden gains, treatment outcomes, and long-term outcomes, in support of the cognitive mediation hypothesis of CBT. These results also fit well with past findings, such as (a) cognitive changes in the first half of CBT predict change in depressive symptomatology in the second half of CBT (DeRubeis et al., 1990), (b) the extent to which therapists apply CBT techniques is correlated with the treatment outcome (DeRubeis & Feeley, 1990; Feeley et al., 1999), and (c) cognitive changes during a whole course of CBT correlate with treatment outcome (e.g., Blackburn & Bishop, 1983; DeRubeis et al., 1990; Seligman et al., 1988). Together, these results suggest that cognitive change is an important therapeutic factor in CBT.

The present results, like the other results cited above, are correlational in nature, and as such, they carry the usual risks when deriving causal inferences. The design of this project, like that of DeRubeis et al. (1990), rules out the possibility of the reversal of causal relationship because the cognitive changes measured occurred before the sudden gains. Still, it is possible that third variables lead to both cognitive changes in the pregain session and the sudden gains. For example, certain biochemical changes before the pregain session might have led the patient to make cognitive changes in pregain sessions and to show mood improvements afterward. In this case, correlation between cognitive changes and symptom improvements would appear even if there was no direct causal relationship between them.

The findings regarding therapists' applications of CBT techniques and therapeutic alliance appear to contradict past findings on the importance of these variables for therapy outcome. However, there is no real contradiction here because our findings do not mean that these variables did not contribute to the occurrence of cognitive changes and sudden gains. To start with, there is the usual concern about power. Much more importantly, these two factors most likely have contributed to cognitive changes and sudden gains in a longer time frame. In other words, cognitive techniques and therapeutic alliance are likely the long-term causes of the cognitive changes and sudden gains. Our short-term within-subject analysis cannot discern such contributions.

The situation is analogous to a long-time heavy smoker who dies of lung cancer. He or she might have smoked the same number of cigarettes during the day of death as the day before death, yet this obviously does not disprove the contribution of smoking to his or her death. The proper way to discern such long-term effects is through between-subjects comparisons in longer time frames. For the smoker, it means comparing the life expectancy of smokers versus nonsmokers. For sudden gains, it

means measuring these variables in an early therapy session and seeing if their levels in an early therapy session predict later occurrence of sudden gain in a between-subjects design. We have conducted such a study, and the preliminary results suggest that both therapist application of CBT techniques and the therapeutic alliance predict the later occurrences of sudden gains (Tang & DeRubeis, 1999b).

The most disappointing aspect of this project was the rather low reliability in rating cognitive changes in session. Two characteristics of cognitive changes might have contributed to this problem. First, the acknowledgment of cognitive changes happens quickly, typically taking less than a minute. If a rater's attention slips in that minute, he or she would miss that cognitive change. Second, cognitive changes happen rarely, and in our sample, an average hour-long session yielded only two cognitive changes. As a result, if one rater missed one cognitive change, it would generate a large between-raters discrepancy. Thus, in contrast to rating qualities of the whole session, like therapist warmth, or rating frequently occurring events, like therapists applying CBT techniques, rating cognitive changes acknowledged in session poses some unique challenges that current methods do not handle well. More rigorous training, and the use of session transcripts, might overcome this problem, albeit at a significant increase in cost.

Part III: Phenomena Observed After Sudden Gains

The sudden gains and the cognitive changes in the pregain sessions might alter the course of the therapy that follows them. For example, they might lead to better therapeutic alliance. After a rapid and sizable symptom decrease, the patient might feel grateful toward the therapist or the therapy, and both the patient and the therapist might feel more confident about working together. In addition, many patients might have observed that sudden gains occurred right after they made important changes in their beliefs or thinking processes. This might lead them to conclude for themselves that the stated rationale of CBT is indeed valid; this should enhance their belief in CBT's efficacy and encourage them to form stronger alliances with their therapists.

Second, the sudden gains and the cognitive changes in the pregain sessions might increase the likelihood of additional cognitive changes, given that mastered cognitive techniques can be applied to other beliefs; modification of one particular belief can be generalized to similar beliefs; and changes in less central beliefs can set the stage for the change of core beliefs and schemas. In addition, as the patients experience lower levels of depressive symptomatology after the sudden gains, they might become less biased in their thinking. If, in addition, the therapeutic alliance has improved, this should facilitate further cognitive changes as well. All of these factors might work together to produce extensive cognitive changes in the therapy sessions after the sudden gains.

To test these possibilities, we compared the levels of therapeutic alliance and cognitive change in the after-gain sessions with those in the prepregain and pregain sessions. Therapists' application of abstract and concrete CBT techniques were also measured in the after-gain sessions because we were interested in whether the sudden gains affected these aspects of therapist behaviors as well.

Method

Part III was conducted concurrently with Part II. The after-gain sessions were mixed with the prepregain and pregain sessions, and the raters rated them all together according to the Part II procedures.

Results

The therapeutic alliance in the after-gain session was higher than that in the pregain session. The difference was statistically significant for the Penn Helping Alliance Scale, $t(15) = 2.90, p = .03, d = 0.75$, and at the level of a nonsignificant trend for both the VTAS, $t(15) = 2.10, p = .06, d = 0.52$, and the WAI, $t(15) = 2.00, p = .07, d = 0.54$.

The top of Figure 4 shows the average amount of cognitive changes reported in the prepregain, pregain, and after-gain sessions. The amount of cognitive change reported in the after-gain sessions was comparable with that in the pregain sessions, $t(15)= 1.20, p = .34, d = 0.29$, and it was significantly higher than that in the prepregain sessions, $t(15) = 3.00, p = .02, d = 0.77$.

The levels of therapists' application of concrete and abstract CBT techniques in the after-gain sessions were not significantly different from their levels in either the prepregain or pregain sessions, all ts ≤ 0.72, all ps $\geq .48$, all ds $\leq .18$.

Results Integrated: The Synchronized Time Courses of Depressive Symptomatology, Cognitive Change, and Therapeutic Alliance

Results from all three parts of the project can be juxtaposed by synchronizing their time courses. Figure 4 presents the synchronized time courses of patients' cognitive changes, their level of depressive symptomatology, and the therapeutic alliance. Note that assessments of cognitive change and the therapeutic alliance were made from tapes of each respective session, whereas assessments of depressive symptomatology refer to the time in between sessions. As Figure 4 shows, increases in the patients' cognitive changes occurred first, followed immediately by the sudden gains. The sudden gains were, in turn, followed immediately by increases in therapeutic alliance. Thus, the data suggest the following chain of events: substantial cognitive changes → sudden gain → improved alliance + further cognitive changes.

Discussion

Interaction between therapeutic alliance and symptom improvement. The after-gain-session results suggest that the therapeutic alliance can show significant improvements after important cognitive changes and symptom improvements have taken place. Crits-Christoph, Barber, and Kurcias (1993) also reported that the accuracy of therapists' interpretations in psychodynamic therapy predicts later changes in therapeutic alliance. These findings suggest that the therapeutic alliance can improve in response to positive therapeutic changes. Thus, when a positive correlation between therapeutic alliance and outcome is observed, it does not automatically warrant the conclusion that therapeutic alliance contributed to outcome. To establish such a causal relationship, researchers need to control for the contribution of symptom improvement to therapeutic alliance (cf. DeRubeis & Feeley, 1990; Feeley et al., 1999). This might be a difficult task in some situa-
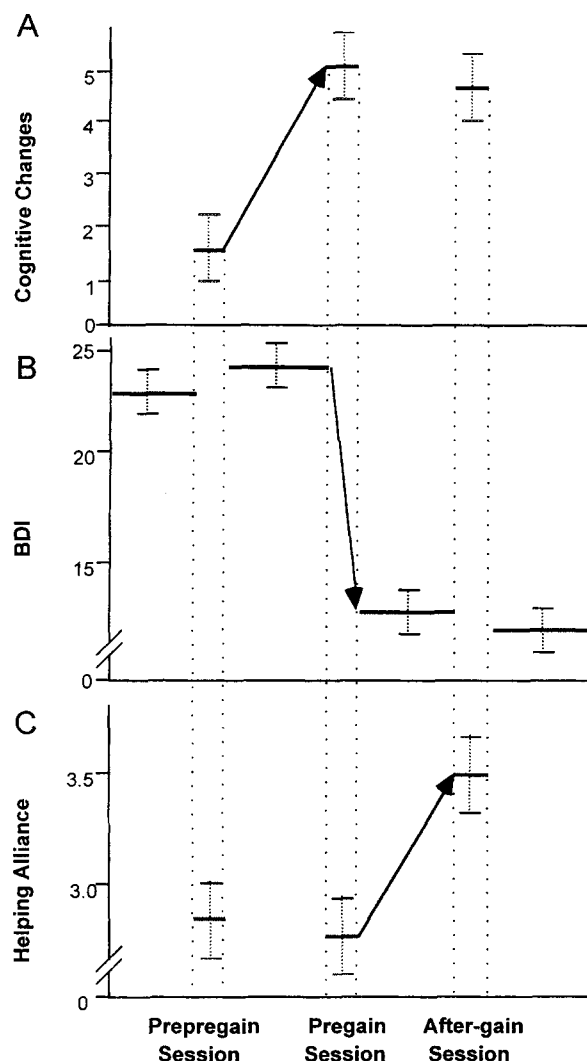

Figure 4. Synchronized time courses of (A) cognitive changes, (B) severity of depressive symptomatology, and (C) therapeutic alliance. The figure demonstrates that an increase in the amount of cognitive changes preceded a decrease in depressive symptomatology, which in turn preceded an increase in therapeutic alliance. (Cognitive changes and therapeutic alliance concern the time in therapy session, but changes in depressive symptomatology apply to the time between sessions, and they are plotted as such in this figure. Means and standard errors are shown. $N = 16$.) BDI = Beck Depression Inventory.

tions, but it can be accomplished by assessing the alliance before important symptom improvements.

The after-gain sessions and the upward spiral. These results suggest that after the pregain sessions, a positive feedback loop was triggered, as cognitive changes in the pregain sessions eventually led to additional cognitive changes in the after-gain sessions. We speculate that this positive feedback loop might extend beyond the after-gain sessions: Cognitive changes and alliance improvements in the after-gain session might be followed by additional cognitive changes, alliance improvements, and symptom improvements in the later therapy sessions. We named this extended

positive feedback loop the *upward spiral*. It may be such an upward spiral that eventually brings these patients to sustained recovery. Future analyses of therapy sessions beyond the after-gain session are needed to test this hypothesis.

General Discussion

Beginning with the observation of sudden gains, our project focused and capitalized on the temporal discontinuities of three variables: severity of depressive symptomatology (sudden gains in Part I), the amount of cognitive changes occurred in session (Part II), and therapeutic alliance (Part III). Moreover, the temporal sequence of these variables' discontinuities (see Figure 4) led us to propose a set of possible causal relationships among these variables.

The present results suggest that sudden gains are not transient mood fluctuations and that they are triggered by a substantial amount of cognitive modification in the pregain sessions. Their occurrence was followed by more cognitive changes and better therapeutic alliance in the after-gain sessions. At the end of therapy, patients who experienced sudden gains evidenced significantly lower levels of depressive symptomatology than the patients who did not. This remained true through most of the follow-up period. Considering this evidence, the sudden gain appears to be a meaningful phenomenon with long-lasting impact on therapy process and outcome.

Combined with past findings, these results provide direct support for the cognitive mediation hypothesis of CBT. They revealed that in about 50% of the treatment responders, superior treatment outcome was preceded by sudden gains, which were in turn immediately preceded by substantial cognitive changes in the pregain sessions.

By revealing the significant differences among the prepregain, pregain, and after-gain sessions (see Figure 4), the results also demonstrate that CBT is not a uniform and homogenous process. In particular, the pregain sessions appear to be one type of critical sessions (Elliott, 1983, 1984; Greenberg, 1977; see also Lambert et al., 1978). The pregain sessions seem "critical" because important therapeutic events occurred in these sessions, the nature of the therapy seems to have changed significantly after these sessions, and these sessions strongly influenced the therapy outcome. Incorporating our observations into Beck et al.'s (1979) original cognitive mediation hypothesis, we propose the following three-stage model of CBT for sudden-gain responders.

1. *Preparation stage.* At the beginning of the therapy, the therapist begins to teach the patient the cognitive model and basic cognitive techniques, and they begin to establish a therapeutic alliance. At the time, these activities produce relatively few cognitive changes, and only minor symptom improvement, but they lay the foundation for future improvements (see Tang & DeRubeis, 1999). The duration and success of the preparation stage likely depends on the therapists' efforts, the patient's readiness for change, and the quality of therapist–patient interaction.

2. *Critical session/sudden gain.* In the pregain session, the patient experiences critical belief changes and schema changes. This leads to a large decrease in the level of depressive symptomatology—the sudden gain.

3. *Upward spiral.* The sudden gain leads to better therapeutic alliance in later therapy sessions. The improved alliance and mood

facilitate cognitive modification, and the cognitive changes that occurred in the pregain sessions also set the stage for additional cognitive changes. The resulting cognitive changes, in turn, sustain symptom relief and eventually lead to recovery.

The above model applies primarily to sudden-gain responders, that is, patients who respond to CBT after experiencing sudden gains. It cannot yet address the mechanism of responders who did not experience sudden gains. Indeed, the results from another of our projects suggest that those patients who responded to CBT without a sudden gain did so through fundamentally different mechanisms (Tang & DeRubeis, 1999b). For nonresponders, this model would suggest that failure in any one of the three stages might lead to failure to respond to therapy. The fact that few nonresponders had sudden gains also suggests that most of the patients failed to respond to CBT because they were never able to advance from the preparation stage to the sudden gain/critical session stage. Finally, although our model focuses on positive progress, the actual therapy experience must involve setbacks. These setbacks might occur in all stages of therapy; sometimes they will be overcome, and sometimes they might lead to therapeutic failure. The exploration of these setbacks is an interesting line of inquiry in and of itself (e.g., Safran & Muran, 1996).

This model is our first attempt to incorporate the concepts of "nonuniformity" and "critical sessions" into CBT's mechanism. These concepts have been discussed in theoretical work for decades, yet they have been ignored by most empirical investigators. A search of PsycINFO revealed only two empirical studies of critical sessions: Rand's (1979) unpublished doctoral dissertation and Elliott's (1984) intensive single-case study. This is unfortunate because these concepts have important implications for empirical mechanism research. For example, the concept of critical session implies that important therapeutic changes might be concentrated in certain sessions rather than spread evenly across all sessions. As a consequence, the common approach of analyzing the correlations among variables in a random sample of therapy sessions might overlook some of the most important events in therapy because such correlations can only reveal what happens in typical sessions, not what happens in the few sessions that lead to therapeutic breakthroughs (Lambert et al., 1978).

Two factors have probably contributed to this lack of research interest. First, the presence of critical sessions has not been demonstrated in a large sample, and there have been no convenient objective methods to identify potential critical sessions. Our results partially resolve both problems, suggesting that one subtype of critical sessions, the pregain session, not only exists but occurs in about half the CBT responders. Our method for identifying the sudden gains also might provide a convenient way to identify these critical sessions.

The present results leave one crucial question unanswered: If cognitive changes lead to sudden gains, which then lead to good outcome, what leads to the cognitive changes? Future research is needed to address these questions. Preliminary findings from our own research suggest that cognitive changes may result from a strong therapeutic alliance and consistent application of CBT techniques in the early stages of therapy (Tang & DeRubeis, 1999b).

The findings we have reported were obtained in an initial study of these phenomena. There are many ways in which the work described here can and should be refined. For example, our rating

method is disappointing in its reliability; substantial revision is needed, perhaps through the use of transcripts or better rater training methods. In addition, key links in our model, such as the causal relationship between cognitive changes and sudden gains, are supported solely by correlational data. Many variables that might have contributed to sudden gains and cognitive changes— such as patient readiness, life events, and homework—need to be examined. The generalizability of our findings also needs to be established in future empirical investigations. In any effort to replicate or extend these findings to other populations (e.g., more mildly depressed patients and patients with anxiety disorders), the specific criteria we used to identify sudden gains will need to be modified.

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