Sudden gains in two psychotherapies for posttraumatic stress disorder

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Abstract

We examined sudden, large, and stable shifts in symptoms from one therapy session to the next in two treatments for posttraumatic stress disorder (PTSD). Shifts in a positive direction (sudden gains) have so far been more frequently analyzed than those in a negative direction (sudden losses). We analyzed data from 102 outpatients suffering from PTSD who received either a cognitive-behavioral or a Gestalt-based intervention. Sudden gains, at 22.5%, were more frequent than sudden losses (3.9% of patients). Participants who had experienced sudden gains had lower PTSD scores at posttreatment, but not at the 6-month follow-up. As sudden losses were so rare, they were not analyzed statistically. Sudden gains accounted for 52% of overall treatment gains or 26% of overall change in a positive direction. Among very successful patients, those with sudden gains were overrepresented, but in absolute terms, there were as many patients without sudden gains in this group. There was no connection between sudden gains and type of intervention or depressive symptoms. Sudden gains and sudden losses occurred in our sample of PTSD patients, but in the light of current results, their clinical importance seems to be limited.

In psychotherapy research, symptomatic change over the course of therapy has so far most frequently been measured on a group level. However, in the last decade the individual course of symptom change has garnered more attention (Laurenceau, Hayes, & Feldman, 2007). In this context, the phenomenon of substantial symptomatic improvement occurring between one therapy session and the next, termed “sudden gain”, has been studied, most frequently in depression treatment. Tang and DeRubeis (1999) suggested statistical criteria for sudden gains which require changes in symptoms to be large in absolute terms, large compared to the level before the gain, and temporally stable.

To our knowledge, sudden gains have been examined in four studies on PTSD so far. Kelly, Rizvi, Monson, and Resick (2009) examined sudden gains in 72 female PTSD patients who were treated either once or twice per week. Symptom scores were collected weekly, that is, part of the sample completed the measure at every other session, which is a deviation from the usual procedure. Patients who had experienced a sudden gain (“gainers”) made up 39% of the sample. Gains were connected with better treatment outcome for PTSD at posttreatment, but not at the 6-month follow-up. Gainers showed significantly lower depression scores both at posttreatment and follow-up, but not at pretreatment.

Doane, Feeny, and Zoellner (2010) examined sudden gains in a sample of 23 women receiving prolonged exposure therapy (PE; Foa, Hembree, & Rothbaum, 2007). Gainers (52%) achieved better PTSD outcome at posttreatment and reported less depressive symptoms both before and after therapy. (Follow-up data is not reported.)

Aderka, Appelbaum-Namdar, Shafar, and Gilboa-Schechtman (2011) report on a mixed-gender sample of 63 children and adolescents treated according to a developmentally adjusted PE protocol. They found sudden gains in 49% of their sample. Gainers had better PTSD outcome at posttreatment as well as at follow-ups after 3 and 12 months. The same results emerged for depressive symptoms for all time points except the second follow-up.

The most recent study was the largest (N = 200) and the only one including a pharmacological treatment condition. Jun, Zoellner, and Feeny (2013) found comparable rates of sudden gains in both the PE (42%) and the sertraline (31%) conditions, there were, however, differences in the timing, magnitude, and reversal rates. Again, gainers experienced more pre- to posttreatment symptom change.

In contrast to the PTSD studies, which have so far focused only on sudden gains and outcome, more factors have been studied in treatment for other disorders. In the original sudden gain study,
Tang and DeRubeis (1999), working with a sample of patients with depression, compared the sessions before sudden gains (pregain sessions) with control sessions and with postgain sessions. They found that patients expressed more cognitive changes in pregain sessions than in control sessions, and that both therapeutic alliance and symptoms improved between the pregain and postgain sessions. These findings support the authors’ hypothesis that substantial cognitive changes can cause sudden reductions in symptoms which in turn lead to improved alliance and further cognitive change in an upward spiral. Support for more cognitive change in pregain sessions also comes from a replication study (Tang, DeRubeis, Beberman, & Pham, 2005) and a study on group therapy for anxiety disorders (Norton, Klenck, & Barrera, 2010). In contrast, Andrusyna, Luborsky, Pham, and Tang (2006) did not find higher levels of cognitive change in pregain sessions in their study of a psychodynamic treatment for depression. However, both therapeutic alliance and accuracy of therapists’ interpretations were rated higher in pregain sessions. Taken together these findings suggest different precursors of sudden gains in different forms of therapy. The findings that sudden gains did not correlate with general factors such as early therapeutic alliance (Hardy et al., 2005) or changes in self-esteem (Kelly, Roberts, & Ciesla, 2005) also fit with the notion of sudden gains being connected to the content of the therapy specifically. On the other hand, two studies (Jun et al., 2013; Vittengl, Clark, & Jarrett, 2005) found sudden gains in purely pharmacological interventions.

In their recent meta-analysis on sudden gains, Aderka, Nickerson, Bae, and Hofmann (2012) were able to include 20 treatment conditions from 16 studies, mostly on depression or anxiety disorders. Gainers had significantly better outcome in the primary outcome measure both at posttreatment and follow-up, with medium effect sizes (posttreatment: Hedges’ g = 0.62, follow-up: g = 0.56). Moderator analyses indicated that the effect of sudden gains in cognitive behavioral therapies (CBTs) was significantly greater than in non-CBT interventions, even though gains occurred at similar rates in both treatment types. Sudden gain effect sizes did not differ between treatments for anxiety disorders and depression and were not affected by modifications in the sudden gain criteria or inclusion vs. exclusion of first session gains.

The process research and meta-analysis described above suggest that sudden gains are connected to therapy content and that their connection with improved outcome is stable across different treatments and disorders. Aderka et al. (2012) conclude that their “review indicates that sudden gains have a lasting, long-term effect on psychiatric symptoms” (p. 100). Still, the question of whether sudden gains are important for treatment outcome remains doubtful. The usual procedure is to compare therapy outcome between gainers and non-gainers. This may lead to classing patients who did not show much improvement at all or even deteriorated as non-gainers, which lowers that group’s average outcome, even if other non-gainers were very successful. Vittengl et al. (2005) addressed this issue by analyzing the impact of sudden gains only among treatment responders. They found no differences in short term outcome between responders who had experienced sudden gains and those who had not. In the long term, patients without sudden gains even did better in two areas of functioning. The authors conclude that: “Consequently, we speculate that sudden gains represent only one pathway to acute phase treatment response [...] and confer no advantages among treatment responders.” (p. 180).

Another possible explanation for the sudden gain phenomenon is that they are just especially large fluctuations in symptoms over the course of therapy. This explanation has been advanced by Hofmann, Schulz, Meuret, Moscovitch, and Suvak (2006) who examined sudden gains in a sample of patients suffering from social phobia. They found significantly higher pretreatment symptom levels in gainers, but no significant difference either at posttreatment or follow-up. The authors suggest that the sudden gain methodology might select those participants who have started out with more severe symptoms and then regressed toward the mean. A similar argument can be applied to samples where the groups with and without gains do not differ before treatment. It is a universal finding that some patients profit more from a given form of (psycho)therapy than others. It is possible that if more change happens overall, there is a greater likelihood of large changes between consecutive sessions.

Only few studies have considered the opposite of sudden gains, that is, large increases in symptoms that stay stable for a few sessions (“sudden losses”). Lutz, Bachmann, Tschaftsaz, Smart, and Lambert (2007) studied sudden gains and sudden losses in a sample of 1640 clients of a university clinic using a general measure of current functioning. They found that gainers (9.3%) achieved better outcome than other patients. The groups who had experienced both gains and losses (1.6%), and neither (82.7%), did not differ from each other but achieved better outcome than those with losses only (6.2%). The low rates of sudden gains in this sample may be explained by the fact that the sample did not meet criteria for any DSM-IV diagnosis, possibly limiting those clients’ range for improvement as well as the generalizability of results. The authors note that while patients with gains were more likely than others to achieve good outcome, the great majority of reliably improved patients had not experienced a sudden gain.

Even though empirical evidence about sudden gains is accumulating, the most important question, that is, the question of their significance in individual patients, cannot be answered yet. On the one hand, sudden gains are connected to good outcome when comparing patients with and without gains, and seem to occur after sessions in which core concepts of the therapy are realized to a great extent. Also, the stronger connection between sudden gains and therapy success in CBT treatments compared to other treatments (Aderka et al., 2012) seems to indicate that sudden gains are connected to some aspect of therapy content. The fact that Aderka et al. (2012) calculated effect sizes seems to imply causation. On the other hand, it is impossible to statistically disentangle overall symptomatic improvement from sudden gains because sudden gains are part of overall improvement. Therefore it is equally possible that sudden gains are only an expression of good therapy response, which is a more parsimonious explanation (Hofmann et al., 2006). In addition, there is some evidence that early sudden gains may be more important, suggesting that these early gains may be an expression of the phenomenon of “rapid response” to therapy (e.g. Lutz et al., 2007).

As becomes apparent from the above, research on sudden gains in PTSD treatment is not as advanced as for depression. In this context, our study has several aims. The first is to add to the literature on sudden gains in PTSD treatment. We expect to find a rate and magnitude of sudden gains similar to those found in other PTSD treatment studies and we expect gainers to achieve better average outcome for PTSD and depressive symptoms than non-gainers. In addition, we plan to determine whether a sudden gain was necessary for large overall improvement. Our second aim is to see whether the two therapies used in our study, one CBT and one gestalt-based intervention, differ with respect to frequency or timing of sudden gains or their connection to outcome. In accordance with Aderka et al. (2012) we expect sudden gains to occur at comparable rates in both conditions, but to be stronger related to outcome in the CBT condition. Our third aim is to examine sudden losses and their connection to outcome. We expect to find fewer losses than gains because symptom levels decrease on average. As
there has been some evidence that early sudden gains may be more strongly connected to outcome, our fourth aim is to compare the outcome of patients with early vs. late gains. In the fifth place, to examine whether patients with sudden gains generally show more fluctuation in their symptoms, we compare the overall fluctuations (both increases and decreases from session to session) between groups.

Method

Sudden gain and sudden loss criteria

We used the sudden gain criteria suggested by Tang and DeRubeis (1999). To meet the first criterion, absolute magnitude, the decrease in symptoms had to exceed what could be accounted for by the instrument’s unreliability. Therefore, we calculated the reliable change index (RCI; Jacobson & Truax, 1991) using the standard deviation and Cronbach’s alpha from our sample (pre-treatment measurement, complete study sample). As suggested by Martinovich, Saunders, and Howard (1996), we substituted the Cronbach’s alpha statistic for the retest reliability in the RCI formula because this statistic could be obtained directly from our sample. This resulted in an absolute magnitude criterion for a sudden gain of at least 21 points on the Impact of Event Scale — Revised (IES-R; Weiss & Marmar, 1997). This translates to 18% of total range of the IES-R, which lies between the percentages used in adult PTSD studies so far (Doane et al., 2010; 14%; Kelly et al., 2009; 24%) and is considerably stricter than the cut-off used by Aderka et al. (2011; 8%). The second criterion of relative magnitude is defined as a decrease of at least 25% of the pregain score (Tang & DeRubeis, 1999). The third criterion requires conducting two-sample t-tests with an alpha level of .05 comparing the symptom scores of the three sessions preceding the gain with those of the three sessions following it. This poses the difficulty that only gains occurring after session three can be analyzed. Therefore, we decided to include pre- and posttreatment scores as well, which enabled us to analyze sudden gains occurring as early as after the second and as late as before the twenty-third session.

For sudden losses, we used equivalent criteria, that is, to be considered as a sudden loss, an increase had to be at least 21 IES-R points. We fixed the criterion for relative magnitude at 33% of the preloss score. This is not in accordance with the criteria used in research so far, as Lutz et al. (2007) used a criterion of 25% for losses as well. However, by requiring an increase of 33%, one arrives at the same absolute numbers for gains and losses which seems to make more sense than having a less strict requirement for losses. For example, a decrease of 25 points from 100 to 75 points (25% of 100) would be a gain and an increase of 25 points from 75 to 100 (33% of 75) a loss. The stability of sudden losses was established with t-tests, as described above for gains. This means that a sudden loss is different from a symptom spike as described by Hayes, Laurenceau, Feldman, Strauss, and Cardaciotto (2007) because even though a spike also represents a sudden increase in symptoms, there is no requirement for temporal stability.

To further assess the stability of sudden gains, Tang and DeRubeis suggested looking for a reversal. A reversal is defined as an increase in symptoms at any point after the gain such that the symptom score is at least as high as the score of the postgain session plus 50% of the gain. Accordingly losses were considered reversed when the symptom score was lower than the postloss score minus 50% of the loss.

For distinguishing between early and late gains, we followed Lutz et al. (2007) and considered gains occurring before the fifth session as early. Patients were classed according to their earliest sudden gain.

Procedure

We report on data from a randomized treatment outcome study comparing two active treatments (Butollo, Karl, König, & Rosner, 2014). Patients suffering from PTSD after diverse, mostly civilian, traumas were treated with up to 24 sessions of either dialogical exposure therapy (DET, Butollo & Karl, 2012), a treatment based on Gestalt therapeutic principles, or with a slightly adapted form of cognitive processing therapy (CPT; Resick, Monson, & Chard, 2007), a cognitive-behavioral treatment with a well-established efficacy. For ethical as well as practical reasons, there was no untreated control group. Participants provided written informed consent prior to their inclusion in the study. The study was approved by the institution’s ethics committee.

We used a flexible dose of therapy, meaning that when patient and therapist agreed that the patient had profited sufficiently, therapy was terminated and the patient was considered a treatment completer. While the great majority of patients attended the maximum of 24 sessions (short term therapy in the framework of compulsory health insurance), the range was 10–24 sessions (M = 22.75, SD = 3.02). Since ten sessions are still comparable to the regular lengths of therapies examined in other sudden gain studies, we decided not to exclude patients with few sessions because these were all treatment completers, and were not considered to be less likely to have experienced sudden gains. To control for the influence of number of sessions, we used this variable as a covariate in the analyses.

Participants

Of the 141 study participants, 122 completed treatment. For 102 (83.6%) of these, data from pre- and posttreatment and during treatment were available. The analyses presented here are based on this subsample of N = 102. Participants’ ages ranged from 19 to 78 years (M = 35.8, SD = 11.5). The majority (70.6%) were female. The most frequent traumatic events in this group were interpersonal violence (34.3%) and accidents (31.4%), followed by job-related trauma (15.7%). For 70 patients, follow-up data collected 6 months after the end of therapy were available as well.

All participants fulfilled the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; American Psychiatric Association [APA], 1994) criteria for PTSD as established by one of two experienced clinicians according to the International Diagnostic Checklists for DSM-IV and ICD (IDCL; Hiller, Zaudig, & Mombour, 1996). They were randomized to receive either DET (46.1%) or CPT (53.9%; the difference between the two subsample sizes reflects exclusion of incomplete data sets).

Measures

We measured sudden gains with the German version of the Impact of Event Scale — Revised (IES-R; Maercker & Schützwohl, 1998), a 22-item self-report measure of PTSD symptoms. Respondents rate how frequently they have experienced each symptom in the last seven days. The instrument is not appropriate for diagnosing PTSD because the items do not correspond to DSM criteria, but it is a widely used instrument for measuring PTSD symptom severity with good psychometric properties. Maercker and Schützwohl (1998) report subscale reliabilities between α = .71 and α = .90. Therapists administered the IES-R before each treatment session, and it was also part of the assessment battery at pre- and posttreatment and follow-up.

The Posttraumatic Diagnostic Scale (PDS; Foa, Cashman, Jaycox, & Perry, 1997) was administered at pre- and posttreatment and follow-up. The PDS is a self-report measure that can be used for
determining diagnostic status for PTSD according to DSM-IV criteria, as well as symptom severity. The severity score of the German version has good psychometric properties (internal consistency of the total score \( \alpha = .94 \), concurrent validity \( r = .76 \); Griesel, Wessa, & Flor, 2006).

A master-level clinician established comorbid diagnoses with a computer-aided interview, the diagnostic expert system for mental disorders (DIA-X; Wittchen & Pfister, 1997), a computerized version of the German translation of the Structured Clinical Interview for DSM-IV (SKID; Wittchen, Zaudig, & Fydrich, 1997). The Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983) was included as a measure of overall psychological distress. The BSI is a shortened version of the well-established Symptom Checklist 90—Revised (Derogatis, 1977) with very good psychometric properties (Geisheim et al., 2002). It contains a depression subscale consisting of six items which we used as a depression measure, in lieu of a specific measure of depressive symptoms which was unfortunately lacking.

Interventions

Cognitive processing therapy (CPT; Resick et al., 2007) is a PTSD treatment from a cognitive therapy tradition. While the focus is on cognitive work, it includes an exposure component in the form of (usually two) written accounts of the traumatic experience, which patients write at home and then read aloud in session. The cognitive work is supported by worksheets which build upon one another and enable patients to work with their dysfunctional thoughts independently of the therapist. Towards the end of the therapy, the therapist introduces five issues that are frequently difficult for trauma survivors: safety, trust, power/control, esteem, and intimacy. The therapy was originally developed for rape victims and subsequently adapted for use in the military context. We used a German adaptation of the manual (König, Resick, Karl, & Rosner, 2012) which includes 15 sessions instead of the original 12. Also, therapists can include behavioral exercises, and some worksheets were slightly modified. We allowed more sessions if patients had difficulties with a particular issue or worksheet or if the therapist felt that spending more sessions on the trauma account would be helpful. Many patients also wished to discuss the five issues in more detail.

Dialogical exposure therapy (DET) is an integrative treatment for PTSD which includes CBT components, but relies on a Gestalt therapeutic framework. DET is structured in four phases: safety, stability, confrontation, and integration. Phases do not have prescribed lengths but patient processes determine progression from one phase to the next. Therapists used a treatment manual (Butollo & Karl, 2012), but the therapy is more process-oriented and less structured than CPT. The core intervention, dialogical exposure, is a modified form of empty-chair work in which the patient is supported in entering into a dialogue with the imagined presence of the perpetrator. A more in-depth description of DET has been published by Butollo, Karl, König, & Hagl (2014). A feasibility study has yielded encouraging results (Butollo, König, Karl, Henkel, & Rosner, 2014).

Data analysis

We used chi-square analyses to compare rates of gains between genders and between those with and without comorbid depression. To test for differences in treatment outcome, we conducted repeated measures analyses with IES-R total score and BSI depression as dependent variables, gain status and intervention group as independent variables, and number of sessions as covariate.

To determine whether sudden gains were necessary for very good outcome, we considered those participants whose IES-R pre-post differences were more than one standard deviation above the mean, and calculated the percentage of gainers in this group.

To examine how large sudden gains were in relation to overall symptom change when fluctuations in symptoms in both directions were considered, we calculated the overall amount of change in both directions by adding the absolute values of session-to-session differences to receive “fluctuation scores”. Two patients might have the same pre-post difference in IES-R, but their fluctuation scores might differ widely because positive and negative changes during therapy canceled each other out. We compared average fluctuation scores between patients with and without sudden gains.

Results

Based on the criteria detailed above, 23 patients (22.5%) experienced 28 sudden gains and 4 patients (3.9%) experienced 5 sudden losses. No patients experienced both types of shift. Five gains (18%), and all of the losses were reversed. The occurrence of sudden gains did not differ with gender, \( \chi^2(1) = 0.015, p = .903 \), or depression status, \( \chi^2(1) = 0.018, p = .776 \). Sudden gains occurred as early as after the second (first session gains were not examined) and as late as after the 18th session.

Sudden gains and interventions

Sudden gains occurred at nearly identical rates in the two treatment conditions: 15 gains (27.3%) in CPT and 13 (27.7%) in DET. The distributions are depicted in Fig. 1.

Sudden gains tended to occur earlier in CPT (pregain session \( M = 6.6, SD = 4.6, Mdn = 5 \)) than in DET (pregain session \( M = 9.7, SD = 6.5, Mdn = 8 \)). This difference, however, was not statistically significant (Mann–Whitney U test, \( p = .201 \)). Neither the average magnitudes of the gains (CPT: \( M = 29.7, SD = 7.3 \); DET: \( M = 29.3, SD = 10.7 \)) nor the average magnitudes as a percentage of overall improvement (CPT 53.0%, DET 51.0%) differed between the two treatments.

Sudden gains and outcome

We conducted a 2 (time) \( \times \) 2 (intervention) \( \times \) 2 (gain status) MANOVA with IES-R total score and BSI depression score as dependent variables and number of sessions as a covariate. Results indicated a significant interaction of time and gain status, \( F(2, \)
96) = 6.60, \ p = .002, partial \ \eta^2 = .12 as well as a significant main effect of time, \ F(2,96) = 9.14, \ p < .001, partial \ \eta^2 = .16. These results led us to perform univariate analyses which yielded a significant main effect of time for IES-R, \ F(1) = 12.35, \ p = .001, partial \ \eta^2 = .11, but not for BSI depression, \ F(1) = 0.062, \ p < .001, partial \ \eta^2 < .01. The interaction effect between time and gain status, again, was significant for IES-R, \ F(1) = 12.89, \ p = .001, partial \ \eta^2 = .12, but not for BSI depression, \ F(1) = 1.76, \ p = .188, partial \ \eta^2 = .02. No other main or interaction effects approached significance. Post-hoc \ t\text{-}tests revealed that gainers did not significantly differ from non-gainers at pretreatment, \ t(100) = -.98, \ p = .328, but had significantly lower posttreatment scores, \ t(61.67) = 3.13, \ p = .003, indicating that this group improved more with treatment than non-gainers. The same analyses were conducted with PDS severity scores and yielded the same pattern of effects (data not shown).

There were 14 patients whose pre- to posttreatment change on the IES-R was more than one standard deviation above the sample mean. Seven (50\%) of them had experienced a sudden gain, and the other half had not.

### Sudden gains and follow-up

We conducted follow-up analyses with the subsample of 70 patients for whom data were available. The (3 \times 2) MANOVA, again with IES-R total score and BSI depression as dependent variables and number of sessions as covariate, indicated a significant main effect of time, \ F(4, \ 62) = 3.83, \ p = .008, partial \ \eta^2 = .20, and a significant interaction effect of time and gain status, \ F(4, \ 62) = 2.66, \ p = .041, partial \ \eta^2 = .15. Univariate analyses (using Greenhouse-Geisser to adjust for nonsphericity) again showed a significant main effect of time for IES-R, \ F(1, \ 51) = 7.32, \ p = .001, partial \ \eta^2 = .10 but not BSI depression, \ F(1, \ 51) = 0.26, \ p = .775, partial \ \eta^2 < .01. As in the pre-post analyses, the interaction effect of time and gain status proved significant for IES-R, \ F(1, \ 51) = 7.07, \ p = .003, partial \ \eta^2 = .10, but not for BSI depression, \ F(1, \ 51) = 0.89, \ p = .368, partial \ \eta^2 = .01.

Again, post-hoc \ t\text{-}tests revealed that patients with sudden gains had lower symptom scores than those without at posttreatment, however this was only at the level of a trend, \ t(39.802) = 2.000, \ p = .052, possibly due to the reduced sample size. There were no significant differences between the two groups at either pretreatment, \ t(68) = -1.46, \ p = .150, or follow-up, \ t(68) = 0.91, \ p = .365. Therefore, patients with sudden gains tended to improve more with therapy than patients without sudden gains, but the advantage was lost at follow-up. Again, the analyses were repeated for PDS data and were very similar.

### Sudden losses

As sudden losses were very rare, we do not report statistical tests but summarize descriptive data. As can be seen in Table 1, sudden losses occurred in both therapy conditions. The one patient who experienced two losses still had a high IES-R score posttreatment, despite an overall reduction of 23 points. The other three loss patients were quite successful. Unfortunately, no follow-up data are available for any sudden loss patients.

### Fluctuations in symptoms

Gainers achieved an average reduction in IES-R scores of 56.8 points. During the average sudden gain, scores decreased by 29.5 points. Thus sudden gains could be said to account for 52\% of overall treatment gains. Many authors (e.g. Hofmann et al., 2006; Tang & DeRubeis, 1999) have illustrated this by averaging the three sessions preceding (\ N - 2, \ N - 1, and \ N) and following (\ N + 1, \ N + 2, and \ N + 3) the gain (with \ N being the pregain session) as well as pre- and posttreatment data. For our sample the equivalent illustration is given in the upper part of Fig. 2. The figure suggests that little change happens before and after the gain. The question is, however, whether such a figure really captures therapeutic change on an individual level. For comparison, we have added two patients' individual symptom courses in the lower part of the figure. One patient experienced a sudden gain (after session 11), the other did not. (Patients were selected because of the similarity of their pre- and posttreatment scores to each other and to the group mean.) It becomes very apparent that symptoms fluctuate quite a lot in both cases.

The average fluctuation score for sudden gainers was 165.8 (SD = 59.4), which can be broken down into an overall decrease of

<table>
<thead>
<tr>
<th>Patient</th>
<th>Therapy condition</th>
<th>Preloss session</th>
<th>Loss size</th>
<th>Reversal</th>
<th>IES-R score pre</th>
<th>IES-R score post</th>
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<td>23</td>
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<td>13</td>
<td>24</td>
<td>yes</td>
<td>75</td>
<td>52</td>
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<tr>
<td>3</td>
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<td>30</td>
<td>yes</td>
<td>58</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>CPT</td>
<td>10</td>
<td>34</td>
<td>yes</td>
<td>85</td>
<td>21</td>
</tr>
</tbody>
</table>

Note. IES-R = Impact of Event Scale – Revised, pre = pretreatment, post = posttreatment, preloss session = session before sudden loss.
symptoms of 56.8 points plus fluctuations in positive and negative direction of 54.5 each. Thus, it could be said that sudden gains accounted for about one quarter (26%) of total change in a positive direction. This is still substantial, but it calls into question whether graphs such as the one in Fig. 2, centered around the sudden gain and averaging the other sessions, may suggest a greater impact of the sudden gain than is reasonable.

However, gainers’ fluctuation scores did not differ significantly from those of non-gainers (M = 147.9, SD = 69.8), t(100) = 0.031, p = .975, indicating that sudden gains are not simply a product of greater variability of scores in this group.

**Discussion**

We found sudden gains in 22.5% of our PTSD treatment sample. This number is somewhat lower than those reported in other PTSD studies (Aderka et al., 2011; Doane et al., 2010; Jun et al., 2013; Kelly et al., 2009). Possible explanations include that our cut-off for absolute magnitude was larger than in two of the previous studies and that we measured symptoms at each session, in contrast to Kelly et al. (2009). Also our therapies were substantially longer. While it might be argued that this affords patients more time to experience sudden gains, our overall effect sizes were not larger than those found in other studies (Butollo, Karl, König, and Hagl, 2014; Butollo, Karl, König, and Rosner, 2014; Butollo, König, et al., 2014), which means the average session-to-session change was smaller. It is possible that knowing they had more time made therapists deliver the therapies in a less stringent manner. Therefore the study setting may have been more similar to routine clinical practice than some of the other research studies, and it seems that in routine settings, rates of sudden gains are generally lower (Greenfield, Gunthert, & Haaga, 2011; Lutz et al., 2007; Stiles et al., 2003). However, our result is still well within the range of those reported by Aderka et al. (2012) in their meta-analysis (14.6–52.2%). Sudden gains were related to better PTSD outcome at posttreatment, but not at 6-month follow-up. This is in accordance with Kelly et al. (2009), but at odds with Aderka et al. (2012). This may have to do with the greater similarity of our study with that published by Kelly et al.: We used CPT in half of our sample, and the sample consisted of adults.

The two treatment conditions did not differ with respect to the magnitude or frequency of sudden gains or the strength of the gains’ connection to outcome. However, in the meta-analysis (Aderka et al., 2012), sudden gains in non-CBT treatments showed weaker average relationships to outcome than those in CBT treatments. The authors speculate that the reason for this may be that in CBT treatments, sudden gains are more likely to cause “upward spirals” in symptoms. They offer some possible explanations for this, such as “specific cognitive techniques, behavioral techniques, the directive role of the CBT therapist, or skills acquisition” (Aderka et al., 2012, p. 100). One explanation for the lack of difference in our sample might be that DET includes CBT components and therefore may not differ from “pure” CBT as much as other treatments. Also, therapists’ adherence to the treatment manual was not formally evaluated and the two treatments may have been more similar than intended. On the other hand, the meta-analysis included only four non-CBT conditions. It included only one study directly comparing CBT and non-CBT treatments and in that study, the effect sizes for sudden gains were actually higher in the two non-CBT conditions. Therefore the result of an overall smaller effect size in the meta-analysis might be an effect of other differences between studies.

While the two treatments did not differ with respect to sudden gain frequency or the connection of sudden gains and outcome, DET gains occurred later in treatment than CPT gains, even though this difference was not statistically significant. This may be connected with the temporal structure of the two therapies: In the German CPT adaptation, patients are asked to write the first account of their trauma in session 5, which was the median pregain session in this sample. This should not be over-interpreted, however. As we used a flexible approach, there is no guarantee that gains coincided with trauma accounts in all cases. In DET, on the other hand, focus on the traumatic event occurs in phase 3, that is, more toward the end of treatment (Butollo & Karl, 2012). The results can be understood in the light of evidence that a focus on the traumatic event may be an important feature of effective therapies for PTSD (Bisson et al., 2007). However, as in the sample examined by Doane et al. (2010), many gains occurred either before the beginning of exposure or much later in treatment. The idea that exposure may be related to sudden gains is also not supported by the fact that the rates found in PTSD samples (all receiving trauma-focused interventions), between 23% (this study) and 52% (Doane et al., 2010), are not higher than those found in the depression studies (in which exposure to feared stimuli is typically not a central part of treatment) reported by Aderka et al. (2012), which ranged from 26% to 51%.

Depressive symptoms as measured with the BSI depression subscale were not connected with sudden gains. This is surprising because so far, all PTSD studies have found some such connection (Aderka et al., 2011; Doane et al., 2010; Kelly et al., 2009). A possible reason for our null finding is the lack of an established depression measure. The depression subscale of the BSI only contains six items and while the subscale has a good concurrent validity, the expected factor structure of the BSI with its nine subscales has not been proven empirically (Geisheim et al., 2002), making it possible that we measured general distress rather than depression.

Contrary to our expectations and to some existing studies (Kelly et al., 2005; Lutz et al., 2007), early gains did not show a stronger connection to outcome than later gains. We can only speculate on reasons for this. The initial worsening experienced by some patients in PTSD treatment (Nishith, Resick, & Griffin, 2002), which does not necessarily predict worse outcome, may be responsible.

In addition to sudden gains, we analyzed “sudden losses”. We found five such losses which did not appear to be consistently related to worse outcome. This does not fit with the results by Lutz et al. (2007) who found fewer sudden gains and more sudden losses, as well as worse outcome in sudden loss patients. Because sudden losses were so rare in our sample, no conclusions about them can be drawn with any confidence. As symptoms decreased on average, the low number of losses might be expected. It would be interesting to consider sudden losses as well as sudden gains in more in-depth psychotherapy process research. Also sudden losses might be compared to symptom spikes, short-lived increases of symptoms (Hayes et al., 2007). As this is a PTSD sample, one might argue that sudden losses were an expression of the transient worsening of symptoms which is sometimes observed with the beginning of exposure (Nishith et al., 2002). As the temporal structure of the therapies was flexible, this cannot be wholly ruled out, but the timing of the sudden losses seems to correspond with the probable beginning of exposure (early in CPT, later in DET) only in one of the four patients (i.e., the DET patient with the two losses).

This study has several limitations. Arguably the most serious one for this set of analyses is the considerable attrition in the follow-up period. The reason for this is probably that patients were not reimbursed for participation in diagnostics, but nonetheless, the loss of almost one third of participants is a serious limitation. The lack of a well-established measure for depressive symptoms has been mentioned already. Also, we did not measure treatment adherence. Therapists were required to regularly attend supervision and to complete documentation on the content of each session.
Furthermore, each therapist conducted only one type of treatment, and therapists identified with their own approach, but, while CPT therapists had no knowledge of DET at all, DET therapists also had CBT training (though not CPT specifically). This makes the lack of external evaluation of treatment adherence and therapist competence problematic because it is possible that our findings of no differences between therapies with respect to sudden gains were due to the therapies being more similar than they should have been.

On the other hand, the study has several strengths. We were able to include a total of 102 patients in the analyses which is a large sample size compared to earlier PTSD sudden gain studies. Also, the length of treatments and a more flexible approach to the structuring of treatment possibly make the study more typical of routine clinical practice than studies done in a specialized research context.

All in all, our results indicate that patients with sudden gains are more likely to achieve good outcome at posttreatment, but not at a six-month follow-up, and that sudden gains are not caused by more variability in scores. We found very few sudden losses which seemed to be unconnected to outcome but could not be analyzed statistically. Also our sample exhibited a lot of symptomatic fluctuation in both positive and negative directions. If this was taken into account, the average contribution of a gain to therapy outcome decreased by half. Gains tended to occur earlier in the treatment in CPT than in DET, but this difference was not statistically significant. However, it might be worthwhile to examine whether the presence of sudden gains indicate especially effective phases of treatment. This would tie in with findings that pregain sessions differ in meaningful ways from other sessions (Andrusyna et al., 2006; Norton et al., 2010; Tang & DeRubeis, 1999; Tang et al., 2005). It would be interesting to examine the differences between pregain sessions in DET and CPT as this could indicate mechanisms by which the two therapies work. Therefore, the sudden gains methodology seems particularly suited for selecting sessions for in-depth study in therapy process research.

Notably, there were as many non-gainers as gainers among the very successful patients, indicating that sudden gains are not necessary in order to profit from therapy. Therefore sudden gains seem unsuited to make predictions of therapy outcome in individuals. As clinicians, we find this reassuring because it means that it is not as important how suddenly our patients improve as how much they improve. It means that we do not have to go striving to create “magic moments” or “perfect sessions” but we can do our best in every session, rejoicing in sudden gains but knowing that those patients who improve more gradually (as long as they do improve) have as good a chance of doing well in the long run.

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