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Sudden gains in cognitive behavioral therapy among children and adolescents with obsessive compulsive disorder

Eric A. Storch[a,*], Joseph F. McGuire[b], Sophie C. Schneider[a], Brent J. Small[c], Tanya K. Murphy[d], Sabine Wilhelm[e], Daniel A. Geller[e]

[a] Menninger Department of Psychiatry and Behavioral Sciences, Baylor College of Medicine, USA
[b] Division of Child and Adolescent Psychiatry, Johns Hopkins University School of Medicine, USA
[c] School of Aging Studies, University of South Florida, USA
[d] Department of Pediatrics, University of South Florida, USA
[e] Massachusetts General Hospital and Harvard Medical School, USA

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ABSTRACT

Background and objectives: This study examined the occurrence of sudden gains (or reversal of gains) among children with obsessive-compulsive disorder (OCD) during the course of cognitive-behavioral therapy (CBT), as well as the association of sudden gains with treatment response, treatment group, and pre-treatment clinical characteristics.

Methods: The sample consisted of 136 youth (ages 7–17) with a primary diagnosis of OCD who were randomized in a double-blinded fashion to 10 sessions of CBT with augmentation of either D-cycloserine or placebo. Sudden gain status was determined based on clinician-rated obsessive-compulsive symptom severity, which was collected on 9 occasions across the study period.

Results: 42.6% of youth experienced at least one sudden gain, which tended to occur either after starting exposure and response prevention or towards the end of treatment. After applying the Benjamini-Hochberg procedure for multiple comparisons, there were no significant pre-treatment predictors of sudden gains and only reduced insight predicted the reversal of gains. Individuals with at least one sudden gain had improved overall treatment outcomes, measured both by reduction in OCD symptom severity, and by global illness severity.

Limitations: Several clinical constructs were not examined. Symptomatology was not assessed at every treatment session. Differences in those who achieved sudden gains and those who did not may be obscured. There is the possibility that a sudden gain reflected a scoring error generated by an optimistic or inaccurate report. Finally, a relatively homogenous sample may limit the generalizability of results.

Conclusions: The course of CBT for pediatric OCD is variable with many children experiencing sudden gains, but a sizable percentage experience a reversal of gains which was related to reduced insight. Sudden gains tended to occur after starting exposure and response prevention and towards the end of treatment.

Trialsregistration: ClinicaltrialsgovRegistry:NCT00864123. https://www.clinicaltrials.gov/ct2/show/NCT00864123.

1. Introduction

Cognitive behavioral therapy (CBT) for pediatric obsessive-compulsive disorder (OCD) has demonstrated considerable efficacy with average response rates extending upwards to 70% and 50% of youth experiencing clinical remission (McGuire et al., 2015). However, few investigations have been conducted examining the course of improvement associated with CBT for pediatric OCD and, among children and adolescents, there has been limited attention into the construct of sudden treatment gains over a short period of time (i.e., significant improvements relative to symptom severity prior to the gain, and symptom fluctuations before/after gain; Aderka, Nickerson, Boe, & Hofmann, 2012a, b; Tang & DeRubeis, 1999). The current report aims to address these gaps in the literature.

Investigating sudden gains could have significant clinical relevance. Sudden gains may indicate that the child/family has acquired a meaningful understanding of the treatment model and ability to utilize core therapeutic components (Tang & DeRubeis, 1999). Additionally, the presence of sudden gains may correspond with more robust symptom improvement (Bohn, Aderka, Schreiber, Stangier, & Hofmann,

* Corresponding author. 1977 Butler Blvd, Suite 4-400, Houston, TX, 77030, USA.
E-mail address: eric.storch@bcm.edu (E.A. Storch).

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Table 1
Pre-treatment characteristics and comparisons between youth with and without sudden gains.

Demographic Characteristics	Entire Sample (N = 136)	Sudden Gains (n = 58)	No Sudden Gains (n = 78)		
	N (%)	**N (%)**	**N (%)**	**χ^2**	***V***
Female	72 (53%)	33 (57%)	39 (50%)	.64	.07
Caucasian	122 (90%)	56 (97%)	66 (85%)	5.13*	.19
Site A recruitment site	71 (52%)	34 (59%)	37 (47%)	1.67	.11
Baseline Comorbidity					
Co-occurring anxiety disorder	62 (46%)	24 (41%)	38 (49%)	.72	.07
Co-occurring ADHD	37 (27%)	11 (19%)	26 (33%)	3.47	.16
Co-occurring chronic tic disorders[b]	11 (8%)	6 (10%)	5 (6%)	.41	.07
Co-occurring major depressive disorder	19 (14%)	7 (12%)	12 (15%)	.30	.05
Medication Status at Baseline					
Selective serotonin reuptake inhibitor	38 (28%)	14 (24%)	24 (31%)	.73	.07
Stimulant/non-stimulant ADHD medication	7 (5%)	1 (2%)	6 (8%)	2.43	.13
Antipsychotic medications	3 (2%)	1 (2%)	2 (3%)	.11	.03
Treatment Group					
Randomized to DCS augmentation	66 (49%)	28 (48%)	38 (49%)	< .01	< .01
	Mean (SD)	**Mean (SD)**	**Mean (SD)**	***t***	***d***
Age	12.46 (2.91)	12.33 (3.25)	12.56 (2.34)	-.47	.08
Intake CY-BOCS Total Score	25.94 (5.26)	26.26 (4.99)	25.71 (5.47)	.61	.11
Intake CY-BOCS Insight Score (Item #11)	1.19 (1.08)	1.19 (1.08)	1.19 (1.08)	-.01	.00
Intake CGI-Severity Score	3.71 (0.77)	3.66 (0.72)	3.74 (0.81)	-.66	.10
Baseline Clinician CDRS Total Score[d]	26.36 (8.71)	24.66 (7.63)	27.60 (9.29)	−1.94	.34

Note: ADHD = Attention Deficit/Hyperactivity Disorder, CY-BOCS = Children's Yale-Brown Obsessive Compulsive Scale, CDRS = Child Depression Rating Scale. *p < .05, **p < .01, ***p < .001.
[a] Anxiety disorders included separation anxiety, social phobia, and generalized anxiety.
[c] Antidepressants medication included selective serotonin reuptake inhibitors, clomipramine, serotonin norepinephrine reuptake inhibitors, and any other antidepressants.
[b] Chronic tic disorders included Tourette Disorder and chronic motor/vocal tic disorder.
[d] Four clinician-administered CDRS Total Scores were missing at baseline.

2013; Clerkin, Teachman, & Smith-Janik, 2008; Doane, Feeny, & Zoellner, 2010; Hedman et al., 2014; Present et al., 2008), as well as maintenance of gains over time (Aderka, Nickerson, et al., 2012a, b; Bohn et al., 2013; Hedman et al., 2014; Tang & DeRubeis, 1999). Furthermore, sudden gains may also highlight time-points that one might expect response informing optimal duration of treatment or intervention foci.

Although a number of studies have investigated sudden gains in non-OCD anxiety disorders (Aderka, Nickerson, et al., 2012a, b) and depressive disorders (Lemmens, DeRubeis, Arntz, Peeters, & Huibers, 2016; Tang & DeRubeis, 1999), to date, there have been only two investigations of sudden gains among adults with OCD (Aderka, Nickerson, et al., 2012a, b; Collins & Coles, 2017) and no reports among youth. In these two studies, between 34 and 52% of adults experienced a sudden gain which accounted for 61–66% of total obsessive-compulsive symptom reduction (Aderka, Nickerson, et al., 2012a, b; Collins & Coles, 2017). Presence of a sudden gain was associated with reduced obsessive-compulsive severity after CBT, but did not exhibit an association with treatment response (Collins & Coles, 2017) or reduction in depressive symptoms (Aderka, Nickerson, et al., 2012a, b). Baseline clinical variables of gender, age, marital status, medication status, or severity of OCD and depression symptoms did not predict presence of a sudden gain (Aderka, Nickerson, et al., 2012a, b; Collins & Coles, 2017). Sudden gains also did not differ according to treatment condition (cognitive therapy, exposure therapy, or fluvoxamine with either cognitive or exposure therapy; Aderka, Nickerson, et al., 2012a, b).

The present report extends the literature on sudden gains to children and adolescents with OCD. The first aim was to examine the proportion of youth who experienced a sudden gain (or reversal of gains) during CBT, as well as when these sudden gains occurred in the course of CBT. Consistent with the adult findings, we predicted that over 40% of children and adolescents would experience a sudden gain during the treatment course, but that only 15% would experience a sudden reversal of gains. We also predicted that the majority of sudden gains would occur at two time points, namely upon starting exposure and response prevention sessions and at the end of treatment after receiving maximal treatment dose together with an impending treatment termination. As a second aim, we sought to examine the extent to which sudden gains were associated with therapeutic improvement. Based the adult literature, we expected that sudden gains would account for approximately 65% of overall treatment gains. Finally, given the limited research on predictors of sudden gains in OCD, we conducted exploratory analyses to determine whether pre-treatment demographic or clinical variables were associated with the presence of sudden gains. We also examined the potential impact of treatment condition (augmentation of CBT with D-cycloserine [DCS] or placebo) on sudden gains.

2. Materials and methods

2.1. Participants

Participants were 136 children and adolescents between 7 and 17 years of age (M = 12.46, SD = 2.91) who completed all clinician administered OCD assessments and exposure-based CBT visits that were part of a multi-site clinical trial evaluating the augmentative benefit of DCS or placebo with exposure-based CBT (Storch et al., 2016). Youth who participated in this clinical trial were enrolled between June 1, 2011, and January 30, 2015, at two sites (Site A and Site B) and met the

following inclusion criteria: current and primary diagnosis of OCD on a structured clinical interview (Kaufman et al., 1997); a CY-BOCS Total Score of at least 16 (Scahill et al., 1997); and a full scale IQ of at least 85 (Wechsler, 1999). Youth were excluded based on the following criteria: contraindications for DCS (e.g., epilepsy, renal insufficiency, DCS allergy); unable to swallow study medication; active suicidality or suicide attempt in the past year; co-occurring psychosis, bipolar disorder, autistic disorder, anorexia nervosa, or non-OCD primary hoarding symptoms. Children were also excluded if they initiated an antidepressant or antipsychotic medication within 12 weeks or 6 weeks, respectively, before enrollment, or had an increase in medication dosage before enrollment (8 weeks for antidepressants, 6 weeks for antipsychotics). Any current medication was required to be stable throughout treatment. Further details on study design and criteria can be found elsewhere (Storch et al., 2016).

Table 1 provides demographic and clinical characteristics for the sample. Youth were predominately Caucasian (90%), had moderate pre-treatment OCD symptom severity (range: 16–38), and few were taking a selective serotonin reuptake inhibitor (28%), stimulants/non-stimulant ADHD medication (5%), or antipsychotic medications (2%). Youth commonly experienced co-occurring anxiety disorders (46%), attention-deficit/hyperactivity disorder (27%), chronic tic disorders (8%), and major depressive disorder (14%).

2.2. Measures

Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS-PL; Kaufman et al., 1997). The K-SADS is a clinician-administered structured diagnostic interview of DSM-IV childhood disorders, which was administered at the screening visit to ascertain primary and comorbid psychiatric conditions. The K-SADS-PL has demonstrated good reliability and validity (Kaufman et al., 1997).

Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS; Scahill et al., 1997*).* The CY-BOCS is a semi-structured clinician-administered interview that assesses the presence and severity of OCD symptoms over the past week. The CY-BOCS was administered at the screening visit, during most treatment weeks, and at the post-treatment assessment (see Table 2 for a timeline of assessments). The 10-item CY-BOCS total score has demonstrated reliability, validity, and treatment sensitivity (Scahill et al., 1997; Storch et al., 2004; Storch, Lewin, De Nadai, & Murphy, 2010). The CY-BOCS item 11 is a clinician rating of the participant's insight, and ranges from *excellent insight* (0) to *lacks insight* (4).

Table 2
Timeline of study visits and measures.

Visit	Nature of Session	Study Week	Study Measures
0	Screening assessment	0	CY-BOCS, CGI-S, KSADS, demographics, medication
1	Baseline assessment	1	CY-BOCS, CDRS
2	Treatment session 1	2	
3	Treatment session 2	2	CY-BOCS
4	Treatment session 3	3	
5	Treatment session 4	3	CY-BOCS
6	Treatment session 5	4	
7	Treatment session 6	5	CY-BOCS
8	Treatment session 7	6	CY-BOCS
9	Treatment session 8	7	CY-BOCS
10	Treatment session 9	8	
11	Treatment session 10	9	CY-BOCS
12	Post treatment assessment	10	CY-BOCS, CGI-S, CGI-I

Note: CY-BOCS = Children's Yale-Brown Obsessive Compulsive Scale, CGI-S = Clinical Global Impression of Severity, KSADS = Schedule for Affective Disorders and Schizophrenia for School-Age Children, CGI-I = Clinical Global Impression of Improvement, CDRS = Child Depression Rating Scale.

Child Depression Rating Scale (CDRS; Poznanski & Mokros, 1996*).* The CDRS is a semi-structured clinician-administered scale administered at the baseline visit, which assesses the presence and severity of depressive symptoms. The CDRS Total Score has demonstrated good psychometric properties (Poznanski & Mokros, 1996).

Clinical Global Impression of Severity and Improvement (CGI-Severity and CGI-Improvement; Guy, 1976). The CGI-Severity scale is a clinician-rated instrument that provides a global rating of OCD severity on a 7-point scale ranging from *not at all ill* (0) to *extremely ill* (6). The CGI-Severity was completed at the screening and post-treatment visits. Meanwhile, the CGI-Improvement is a clinician rating of improvement relative to baseline on a 7-point scale that ranges from *very much improved* (1) to *very much worse* (7). The CGI-Improvement was completed at the post-treatment visit, and is well-validated in treatment studies of youth with OCD (Skarphedinsson, De Nadai, Storch, Lewin, & Ivarsson, 2017; Storch et al., 2010). Ratings of *much improved (2)* and *very much improved (1)* correspond with a positive treatment response.

2.3. Procedures

All study procedures were approved by the institutional review boards at the two recruitment sites (Site A and Site B). After explaining study procedures to interested youth and parents, written parental consent and youth assent were obtained. See Table 2 for a summary of the timeline of assessments, treatment sessions, and measure administration. Briefly, at the screening assessment, youth and parents were administered the K-SADS and CY-BOCS to ascertain study eligibility. Afterward, clinicians administered the CY-BOCS and CDRS at the baseline visit to characterize pre-treatment obsessive compulsive and depressive symptom severity. Treatment consisted of 10 sessions over 8 weeks; the first 4 sessions occurred twice weekly, with the remaining 6 sessions occurring weekly. The CY-BOCS was collected at 6 of these treatment sessions. Treatment sessions 1–3 consisted of psychoeducation, hierarchy development and cognitive therapy. Prior to the 4th session in which exposure therapy started, youth who continued to meet inclusion/exclusion criteria were randomly assigned by a computer-generated randomization program to receive DCS or placebo augmentation of the remaining CBT sessions (1:1 ratio, see Storch et al., 2016 for further detail). The week following the final CBT session, youth and parents completed a post-treatment assessment. All clinical evaluators and therapists were blind to treatment condition.

2.4. Analytic plan

Consistent with prior evaluations (Aderka, Nickerson, et al., 2012a, b; Collins & Coles, 2017), a sudden gain was characterized by the following three criteria based on the guidelines outlined in Tang and DeRubeis (1999). First, *the gain had to be large in absolute terms*. Consistent with prior OCD evaluations (Aderka, Nickerson, et al., 2012a, b; Collins & Coles, 2017), a reliable change index was calculated by dividing the average change score (pre-treatment to post-treatment) by the standard error of measurement (Jacobson & Truax, 1991). This produced a cutoff CY-BOCS total score of 5.96, which was rounded up to 6.00 (as the CY-BOCS uses whole integers) and constituted 15% of the CY-BOCS total score range. Second, *the gain had to be large relative to symptom severity prior to the gain*. Consistent with prior studies (Aderka, Nickerson, et al., 2012a, b; Collins & Coles, 2017), the gain should exceed 25% of the pre-gain score. Third, *the gain had to be large relative to symptom fluctuations before and after the gain.* Similar to prior studies (Collins & Coles, 2017; Hardy et al., 2005), t-tests were conducted between the assessments before and after the gain to determine if the gain

was significant. Gains fulfilled this criterion when $t \geq 2.50$ when comparing the mean of the three pre-gain scores with the mean of the three post-gain scores, or $t \geq 3.00$ if only two pre-gain or two post-gain scores were available (Collins & Coles, 2017; Hardy et al., 2005). Finally, consistent with previous studies, we also examined the presence of *reversal of sudden gains*. A reversal of sudden gains was characterized by a loss of 50% or greater of the sudden gain at any point in treatment following the gain. When multiple gains were present, the first sudden gain was used as a benchmark.

First, the dataset was coded to classify *sudden gains* and *reversals of sudden gains*. Descriptive statistics characterized the occurrence of sudden gains and reversal of sudden gains. Second, chi-square and independent t-tests compared pre-treatment differences between youth who exhibited sudden gains and those who did not across demographic, treatment condition (DCS or placebo augmentation), and clinical characteristics. Third, in order to examine whether sudden gains were associated with greater therapeutic improvement, two repeated measures analysis of variance (ANOVA) were completed. In the first ANOVA, the independent variables of time (pretreatment CY-BOCS Total score versus post-treatment CY-BOCS total score), treatment condition (DCS versus placebo) and presence of a sudden gain (absent versus present) were entered into the model. For the second ANOVA, the independent variables of time (pretreatment CGI-Severity score versus post-treatment CGI-Severity score), treatment condition (DCS versus placebo) and presence of a sudden gain (absent versus present) were entered into the model. Significance levels reflect the Greenhouse-Geisser correction for sphericity. T-tests were used to follow-up on significant main effects and interactions, and a chi-square evaluated whether sudden gains were associated with treatment response. Finally, independent logistic regression examined pre-treatment predictors of sudden gains and reversal of sudden gains for demographic and clinical constructs. We applied the Benjamini-Hochberg correction for family-wise error, with a false discovery rate set at 25%.

3. Results

The occurrence of sudden gains and reversal of sudden gains. Based on the outlined criteria, 58 youth (42.6%) experienced at least one sudden gain, with five of these experiencing two sudden gains and one youth experiencing three sudden gains. The average magnitude of all sudden gains was 9.62 points ($SD = 3.02$) decrease on the CY-BOCS total score and constituted 79.7% of the average reduction in CY-BOCS total scores during treatment ($M = 12.07$, $SD = 7.25$). Sudden gains mostly occurred between Visits 5 and 7 ($n = 23$, 16.9%) and Visits 9 and 11 ($n = 20$, 14.7%), with sudden gains observed less often between other assessments (Visits 3 and 5, $n = 1$, 0.7%; Visits 7 and 8, $n = 11$, 8.1%; Visits 8 and 9, $n = 10$, 7.4%). Meanwhile, 18 youth who experienced at least one sudden gain, also exhibited a reversal of the sudden gain before the completion of treatment (31%) that consisted of an average increase of 6.28 points on the CY-BOCS total score ($SD = 3.27$). Interestingly, youth who experienced both a sudden gain and reversal ($n = 18$) exhibited both greater improvement ($M = 8.50$, $SD = 7.01$) and reversal of improvement ($M = 6.28$, $SD = 3.27$) following the sudden gain, in comparison to youth who only exhibited sudden gains (Improvement: $M = 14.58$, $SD = 4.73$, $t_{56} = 2.49$, $p < .02$; Reversal: $M = 1.30$, $SD = 1.70$, $t_{56} = 7.65$, $p < .001$). Therefore, even experiencing some loss of therapeutic improvement, these youth continued to exhibit an overall improvement in OCD symptom severity.

Comparison of youth who did or did not exhibit sudden gains. Table 1 compares differences in demographic and pre-treatment clinical characteristics between youth with OCD who did or did not exhibit a sudden gain during CBT. While those experiencing sudden gains were more often Caucasian (97% versus 85%, $p < .05$), there were no other significant differences in pre-treatment characteristics between groups. There were no differences in the occurrence of sudden gains between those who received DCS-augmented CBT or placebo-augmented CBT (Table 1).

Table 3
Repeated measures ANOVA results examining the relationship between sudden gains and therapeutic improvement.

OCD Symptom Severity (CY-BOCS)	*F*	*p*	η_p^2
Sudden Gain	7.60	.007	.05
Treatment Group	.13	.72	< .01
Time	474.22	< .001	.78
Sudden Gain x Treatment Group	1.00	.32	.01
Time x Sudden Gain	26.33	< .001	.17
Time x Treatment Group	2.87	.09	.02
Time x Treatment Group x Sudden Gain	.68	.41	.01
Overall OCD Severity (CGI-Severity)	***F***	***p***	$\boldsymbol{\eta_p^2}$
Sudden Gain	8.89	.003	.06
Treatment Group	.03	.88	< .01
Time	311.15	< .001	.70
Sudden Gain x Treatment Group	.09	.77	< .01
Time x Sudden Gain	11.42	< .001	.08
Time x Treatment Group	2.32	.13	.02
Time x Treatment Group x Sudden Gain	.17	.68	< .01

Note: Sudden gain refers to the absence/presence of a sudden gain. Treatment group refers to whether the participant was receiving CBT augmented with either DCS or placebo. CY-BOCS = Children's Yale-Brown Obsessive Compulsive Scale. CGI = Clinical Global Impression.

The relationship between sudden gains and therapeutic improvement. Table 3 presents the repeated measure ANOVAs. For the CY-BOCS total score, there was a main effect for time, with post-treatment OCD symptom severity ($M = 13.88$, $SD = 7.00$) significantly lower than pre-treatment symptom severity ($M = 25.94$, $SD = 5.26$, $d = 1.66$). There was also a main effect for sudden gains and the interaction between sudden gains and time. While there was no significant difference in CYBOCS scores between youth who did and did not exhibit sudden gains at pre-treatment (see Table 1, $d = 0.11$), there was a significant difference between groups at post treatment (Sudden Gains Group: $M = 10.73$, $SD = 5.59$; No Sudden Gains Group: $M = 16.14$, $SD = 7.13$; $t_{134} = -4.70$, $p < .001$, $d = 0.82$). The main effect and interactions with treatment group (DCS vs. placebo augmentation) were not significant (Table 3).

For the CGI-Severity, there was a main effect for time, with post-treatment overall OCD severity ($M = 2.24$, $SD = 1.02$) significantly lower than pre-treatment symptom severity ($M = 3.71$, $SD = 0.77$, $d = 1.44$). There was also a main effect for sudden gains and the interaction between sudden gains and time. While there was no significant difference in CGI-Severity between youth who did and did not exhibit sudden gains at pre-treatment (see Table 1, $d = 0.10$), there was a significant difference between groups at post treatment on the CGI-Severity (Sudden Gains Group: $M = 1.86$, $SD = 0.87$; No Sudden Gains Group: $M = 2.53$, $SD = 1.03$; $t_{134} = -3.97$, $p < .001$, $d = 0.70$). Similar to the CY-BOCS ANOVA, the main effect and interaction with treatment group were not significant on the CGI-Severity (see Table 3). Finally, youth with sudden gains were also more likely to be classified as treatment responders (n = 55, 95%) relative to youth without sudden gains on the CGI-Improvement (n = 52, 67%, $\chi^2 = 15.72$, $V = 0.34$, $p < .001$).

Predictors of sudden gains and reversal of sudden gains during treatment. Independent logistic regressions examined whether demographic and pre-treatment clinical characteristics included in Table 1 predicted the occurrence of sudden gains. While race (χ^2 (1) = 5.79, $p < .02$)

and baseline CDRS Total Score (χ^2 (1) = 3.92, $p < .05$) significantly predicted the occurrence of sudden gains in treatment, these predictors were no longer significant after applying the Benjamini-Hochberg correction. Additionally, gender (χ^2 (1) = 0.64, $p = .43$), treatment group (χ^2 (1) = < 0.01, $p = .96$), site (χ^2 (1) = 1.67, $p = .20$), age (χ^2 (1) = 0.22, $p = .64$), anxiety disorder (χ^2 (1) = 0.72, $p = .40$), ADHD (χ^2 (1) = 3.56, $p = .06$), chronic tic disorder (χ^2 (1) = 0.68, $p = .41$), major depressive disorder (χ^2 (1) = 0.31, $p = .58$), SSRI medication (χ^2 (1) = 0.73, $p = .39$), ADHD medication (χ^2 (1) = 2.76, $p = .10$), antipsychotic medication (χ^2 (1) = 0.11, $p = .74$), CY-BOCS Total Score (χ^2 (1) = 0.37, $p = .54$), insight (χ^2 (1) = 0.00, $p = .99$), and CGI-Severity at intake (χ^2 (1) = 0.44, $p = .51$) did not predict the occurrence of a sudden gain.

Additionally, independent logistic regressions examined whether demographic and pre-treatment clinical characteristics in Table 1 predicted the occurrence of the reversal of sudden gains for those participants who experienced a sudden gain ($n = 58$). Major depressive disorder (χ^2 (1) = 5.62, $p < .02$), SSRI medication (χ^2 (1) = 5.94, $p < .02$), and insight (χ^2 (1) = 6.52, $p < .01$) predicted the reversal of a sudden gain during CBT. However, only insight remained a significant pre-treatment predictor of the reversal of sudden gains after applying the Benjamini-Hochberg correction. Meanwhile, race (χ^2 (1) = 1.52, $p = .22$), gender (χ^2 (1) = 0.19, $p = .66$), treatment group (χ^2 (1) = 03, $p = .86$), site (χ^2 (1) = 0.71, $p = .40$), age (χ^2 (1) = 0.48, $p = .49$), anxiety disorder (χ^2 (1) = 0.10, $p = .75$), ADHD (χ^2 (1) = 0.18, $p = .68$), chronic tic disorder (χ^2 (1) = 0.72, $p = .40$), ADHD medication (χ^2 (1) = 2.38, $p = .12$), antipsychotic medication (χ^2 (1) = 0.75, $p = .39$), CDRS Total Score (χ^2 (1) = 0.71, $p = .40$), CY-BOCS Total Score (χ^2 (1) = 0.45, $p = .50$), and CGI-Severity at intake did not (χ^2 (1) = 0.53, $p = .47$).

4. Discussion

These data suggest that the course of CBT can be variable for some youth with OCD. Indeed, nearly half of the youth experienced a sudden gain during CBT, with a subset also experiencing a reversal of such gains. Consistent with other studies in OCD (Aderka, Nickerson, et al., 2012a, b; Collins & Coles, 2017) and non-OCD anxiety disorders (Aderka, Nickerson, et al., 2012a, b), presence of sudden gains was associated with improved treatment response. Presence of sudden gains accounted for approximately 80% of the reduction in obsessive-compulsive severity over treatment. This may reflect children and parents successfully comprehending treatment components and integrating them into their behavioral repertoire. Gains tended to occur shortly after initiating exposure and response prevention, or towards the end of acute treatment. This may suggest the benefit of earlier initiation of exposure and response prevention in treatment (session two/three versus session four) and/or the need to extend overall treatment course for some youth as they may require more exposure and response prevention trials for sufficient therapeutic learning. For those who experienced a reversal of improvement, youth still improved overall. Thus, presence of this should not necessarily shift treatment focus and/or modality, but for many cases requires continuing the intervention course. Presence of depression, SSRI use, and reduced insight was associated with incidence of reversal of gains suggesting that these youth may be at greater risk for experiencing treatment losses perhaps secondary to varying perspective into the content of their symptoms, reduced energy to combat symptoms and/or hopelessness for a positive outcome. Collectively, these data suggest that: (1) sudden gains occur with some frequency, (2) that it is not uncommon for youth to experience a return of symptoms (especially in the presence of depression and/or reduced insight), and (3) if a reversal or loss of improvement does occur, it does not indicate poor prognosis. On this note, youth who experienced sudden gains did not differ on any clinical variables relative to those without sudden gains.

There were few factors that predicted occurrence of sudden gains during treatment, and sudden gains were not associated with augmentation of CBT with DCS. Although race and depressive symptoms were significantly associated with sudden gains, these were no longer significant after correcting for multiple comparisons using the Benjamini-Hochberg procedure. This suggests that few baseline variables predict the presence of sudden gains well. Indeed, sudden gains may likely be more attributed to specific aspects of the therapeutic process and implementation of exposures not captured at baseline. Future research should investigate the relationship between specific therapeutic factors and the onset of sudden gains in CBT. On a positive note, OCD symptom severity was not predictive of sudden gains. Therefore even those patients with severe OCD symptoms at pre-treatment may still experience dramatic symptom improvement from CBT.

There are several study limitations. First, several clinical constructs were not examined that may be related to sudden gains such as externalizing symptoms, which has been associated with attenuated response (Garcia et al., 2010; Storch et al., 2008; Torp et al., 2015). Second, we did not assess OCD symptomology at every treatment session, resulting in some variation in time periods between evaluations. On balance, this allows for greater confidence that the gains were more persistent in nature versus transient, but makes it somewhat more difficult to interpret the timing of some sudden gains. Third, in light of the significant treatment response observed on average, differences in those who achieved sudden gain versus those who did not may be obscured. Fourth, although clinicians were well trained and supervised, there is the possibility that a sudden gain reflected a scoring error generated by an optimistic or inaccurate report on a “good day/interval” and then a return to the previous trajectory at the next visit. Finally, our sample was relatively homogeneous in terms of race/ethnicity and setting (i.e., OCD specialty clinics), which may reduce power to find effects and limit result generality.

Within these limitations, these data suggest that the course of CBT for pediatric OCD is variable with many children experiencing sudden gains, but a sizable percentage having a more linear course. No baseline constructs were associated with presence of sudden gains, making it difficult to discern who these may affect. However, the association with sudden gains highlights the significance of exposure and response prevention in the overall CBT package and that some youth may require extended treatment course. Interestingly, the reversal of gains was relatively common and associated with reduced insight; yet, most of these children continued to improve over time suggesting that the treatment focus/approach need not necessarily be changed if a child experiences a transient return of symptoms during CBT.

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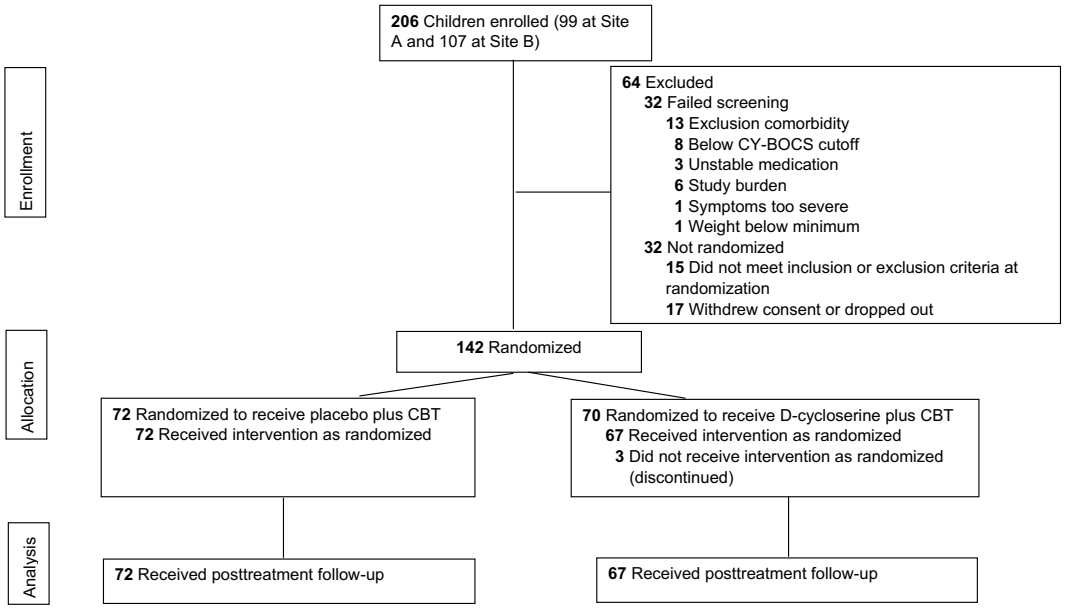
Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jbtep.2019.03.003.

Appendix

Table A1
CONSORT Checklist

Section/Topic	Item No	Checklist item	Report on page
Title and abstract			
	1a	Identification as a randomized trial in the title	3
	1b	Structured summary of trial design, methods, results, conclusions	2
Introduction			
Background and objectives	2a	Scientific background and explanation or rationale	4–5
	2b	Specific objectives or hypotheses	5
Methods			
Trial design	3a	Description of trial design (such as parallel, factorial) including allocation ratio	7–8
	3b	Important changes to methods after trial commencement (such as eligibility criteria), with reasons	NA
Participants	4a	Eligibility criteria for participants	5–6
	4b	Settings and locations where the data were collected	7
Interventions	5	The interventions for each group with sufficient details to allow replication, including how and when they were actually administered	7–8
Outcomes	6a	Completely defined pre-specified primary and secondary outcome measures, including how and when they were assessed	6–8
	6b	Any changes to trial outcomes after the trial commenced, with reasons	NA
Sample size	7a	How sample size was determined	5
	7b	When applicable, explanation of any interim analyses and stopping guidelines	NA
Randomization:			
Sequence generation	8a	Method used to generate the random allocation sequence	7–8
	8b	Type of randomization; details of any restriction (such as blocking and block size)	7–8
Allocation concealment mechanism	9	Mechanism used to implement the random allocation sequence (such as sequentially numbered Containers), describing any steps taken to conceal the sequence until interventions were assigned	7–8
Implementation	10	Who generated the random allocation sequence, who enrolled participants, and who assigned participants to interventions	8
Blinding	11a	If done, who was blinded after assignment to interventions (for example, participants, care providers, those assessing outcomes) and how	8
	11b	If relevant, description of the similarity of interventions	NA
Statistical methods	12a	Statistical methods used to compare groups for primary and secondary outcomes	8–9
	12b	Methods for additional analyses, such as subgroup analyses and adjusted analyses	NA
Results			
Participant flow	13a	For each group, the numbers of participants who were randomly assigned, received intended treatment, and were analyzed for the primary outcome	Figure A1
	13b	For each group, losses and exclusions after randomization, together with reasons	Figure A1
Recruitment	14a	Dates defining the periods of recruitment and follow-up	5, Figure A1
	14b	Why the trial ended or was stopped	Figure A1
Baseline data	15	A table showing baseline demographic and clinical characteristics for each group	Table 1
Numbered analyzed	16	For each group, number of participants (denominator) included in each analysis and whether the analysis was by original assigned groups	Figure A1
Outcomes and estimation	17a	For each primary and secondary outcome, results for each group, and the estimated effect size and its precision (such as 95% confidence interval)	9-12, Table 2, Table 3
	17b	For binary outcomes, presentation of both absolute and relative effect sizes is recommended	NA
Ancillary analyses	18	Results of any other analyses performed, including subgroup analyses and adjusted analyses, distinguishing pre-specified from exploratory	NA
Harms	19	All important harms or unintended effects in each group (for specific guidance see CONSORT for harms)	Figure A1
Discussion			
Limitations	20	Trial limitations, addressing sources of potential bias, imprecision, and, if relevant, multiplicity of analyses	13–14
Generalizability	21	Generalizability (external validity, applicability) of the trial findings	12–14
Interpretation	22	Interpretation consistent with results, balancing benefits and harms, and considering other relevant evidence	12–13
Other information			
Registration	23	Registration number and name of trial registry	3
Protocol	24	Where the full trial protocol can be accessed, if available	NA
Funding	25	Sources of funding and other support (such as supply of drugs), role of funders	15



Note: Details of original study recruitment and retention are shown (see Storch et al., 2016). CBT indicates cognitive behavior therapy; CY-BOCS, Children's Yale-Brown Obsessive Compulsive Scale

Fig. A1. Consort Diagram of Original Study

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