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ScienceDirect

Behavior Therapy 44 (2013) 717–730

Behavior Therapy

www.elsevier.com/locate/bt

Treating PTSD in Patients With Psychosis: A Within-Group Controlled Feasibility Study Examining the Efficacy and Safety of Evidence-Based PE and EMDR Protocols

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The present study uses a within-group controlled design to examine the efficacy and safety of two psychological approaches to posttraumatic stress disorder (PTSD) in 10 patients with a concurrent psychotic disorder. Patients were randomly assigned either to prolonged exposure (PE; *N* = 5) or eye movement desensitization and reprocessing (EMDR; *N* = 5). Before, during, and after treatment, a total of 20 weekly assessments of PTSD symptoms, hallucinations, and delusions were carried out. Twelve weekly assessments of adverse events took place during the treatment phase. PTSD diagnosis, level of social functioning, psychosis-prone thinking, and general psychopathology were assessed pretreatment, posttreatment, and at three-month follow-up. Throughout the treatment, adverse events were monitored at each session. An intention-to-treat analysis of the 10 patients starting treatment showed that the PTSD treatment protocols of PE and EMDR significantly reduced PTSD symptom severity; PE and EMDR were equally effective and safe. Eight of the 10 patients completed the full intervention period. Seven of the 10 patients (70%) no longer met the diagnostic criteria for PTSD at follow-up. No serious adverse events occurred, nor did patients show any worsening of hallucinations, delusions, psychosis proneness, general psychopathology, or social functioning. The results of this feasibility trial suggest that PTSD patients with comorbid psychotic disorders benefit from trauma-focused treatment approaches such as PE and EMDR.

Keywords: efficacy; EMDR; prolonged exposure; psychosis; PTSD

Between 50 and 98% of patients who have experienced psychotic episodes report having been exposed to one or more traumatic life events (see Read, Van Os, Morrison, & Ross, 2005, for a review). Accordingly, the prevalence of PTSD in people with psychotic disorders is relatively high, ranging from 12 to 29% (Achim et al., 2011; Buckley, Miller, Lehrer, & Castle, 2009). It is also important to mention that recent meta-analytical research (Varese et al., 2012) shows that being traumatized as a child almost triples the chance of developing psychosis. This strong association between childhood adversities and the increased risk for psychosis was found both in population-based cross-sectional studies (OR = 2.99), and in prospective studies (OR = 2.75), even after controlling for potentially confounding variables such as genetic liability. These meta-analytical findings suggest that without the causal factor of childhood trauma, there would be 33% less people suffering from psychosis.

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0005-7894/44/717–730/$1.00/0

Significant associations have been shown between type of trauma on the one hand, and hallucinations and delusions on the other (Bentall, Wickham, Shevlin, & Varese, 2012; Varese et al., 2012).

Meta-analyses (Bisson & Andrew, 2009; National Institute for Clinical Excellence, 2005) indicate that PTSD can best be treated using each of the following three treatments: (a) trauma-focused cognitive-behavioral therapy (TF-CBT; see Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010). TF-CBT can be subdivided in several effective types of treatment: prolonged exposure (PE; e.g., Foa, Hembree, & Rothbaum, 2007; Schnurr et al., 2007) and cognitive processing therapy (e.g., Resick, Nishith, Weaver, Astin, & Feuer, 2002); (b) eye movement desensitization and reprocessing therapy (EMDR; Nijdam, Gersons, Reitsma, De Jongh, & Olff, 2012; Shapiro, 2001); and (c) stress management training (e.g., Taylor et al., 2003). However, significant reductions in PTSD and associated symptom severity may also be achieved by other interventions, such as psychodynamic therapy, hypnotherapy, and supportive counseling (Bisson & Andrew, 2009; Ford, Chang, Levine, & Zhang, 2012; National Institute for Clinical Excellence, 2005).

The presence of a past or present comorbid psychotic disorder is the highest-ranking exclusion criterion found in meta-analyses of randomized clinical PTSD outcome studies (Bradley, Greene, Russ, Dutra, & Westen, 2005; Powers et al., 2010; Spinazzola, Blaustein, & Van der Kolk, 2005). Most randomized controlled trials do not report the rationale for excluding patients with psychosis (Bradley et al., 2005). Out of their concern about the potentially adverse effects of trauma treatment, 87% of clinicians also see comorbid psychosis as a contraindication for PE (Becker, Zayfert, & Anderson, 2004). In this vulnerable group—and especially in patients suffering from schizophrenia (Lothian & Read, 2002; Young, Read, Barker-Collo, & Harrison, 2001)—they fear symptom exacerbation or dropout (Becker et al., 2004; van Minnen, Hendriks, & Olff, 2010), or the induction of false memories (Read, Hammersley, & Rudegeair, 2007). On the other hand, some authors specifically encourage mental health professionals to address psychological trauma, especially when clients are suffering from the consequences of child abuse or neglect (e.g., Larkin & Morrison, 2006; Read et al., 2007).

Few explorative TF-CBT outcome studies have been conducted in patients with serious mental illness, including those with psychosis and with comorbid PTSD (Lu et al., 2009; Mueser et al., 2008; Rosenberg, Mueser, Jankowski, Salyers, & Acker, 2004). One study specifically examined the effects of PE in 20 PTSD patients with psychotic disorders (Frueh et al., 2009). They were treated in 14 preparatory sessions comprising anxiety-management training and social-skills training, followed by eight PE sessions. While PTSD symptoms did not decrease in the preparation phase, they decreased significantly during and after PE, without any adverse events being noted. Although these results were promising, the study lacked a control condition, and psychotic symptoms were not monitored. It is not known how PE affected the severity of patients' symptoms of psychosis.

With regard to EMDR, an important EMDR study addressed PTSD in 27 outpatients with a lifetime diagnosis of psychosis including schizophrenic disorder (Van den Berg & van der Gaag, 2012). Results showed that, after six 90-minute EMDR sessions, patients improved significantly with regard to PTSD symptoms, depression, anxiety, hallucinations, and self-esteem, and that no adverse events had occurred. There was no control condition.

In sum, it is suggested by the few exploratory studies that have evaluated treatment outcomes in patients with PTSD and comorbid psychosis that PE and EMDR are both safe approaches to treating PTSD in this population. The results challenge the consensus among many clinicians and researchers that PTSD treatment and research in this particular group is potentially dangerous and should therefore be avoided (van Minnen, Harned, Zoellner, & Mills, 2012).

The main aim of the present study was to replicate previous findings (Frueh et al., 2009; Van den Berg & van der Gaag, 2012) indicating efficacy of evidence-based PTSD protocols (i.e., PE and EMDR) in treating patients with comorbid PTSD and psychosis, and to extend the previous studies through the inclusion of a control condition. The second aim of the present study was to evaluate the safety of both trauma treatments. The safety effects of treatment were indexed using weekly monitoring, and pertained to several variables that are often perceived as barriers for trauma-focused treatments in patients with psychosis: hallucinations, delusions, psychotic thinking, social functioning, general psychopathology, and distress, and the occurrence of serious adverse events (e.g., self-harm and suicidal behavior). The third goal was to tentatively compare the PE and EMDR protocols in terms of their differential effects on PTSD symptoms, treatment acceptance, and safety.

Method

PARTICIPANTS

Potential candidates for our trial were recruited from a local Dutch mental health outpatient center, and referred by their therapists. Those eligible were adult patients who had suffered a severe psychotic

episode up to 3 years prior to the study, with current positive or negative psychotic symptoms remaining, and who received treatment for their symptoms of psychosis. In total, 32 patients were referred, 10 of whom declined further participation; 22 consented to an inclusion interview (see also the flowchart in Figure 1). Eligibility was established by assessing psychotic symptoms as part of a psychotic or mood disorder; PTSD symptoms were established through the Structured Clinical Interview for DSM-IV diagnoses (SCID-I; First, Spitzer, Gibbon, & Williams, 2002). PTSD diagnoses were verified using the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1990, 1995; Dutch version by Hovens, Luinge, & van Minnen, 2005). There were three exclusion criteria: acute suicidality, an IQ below 70 according to chart diagnosis or intelligence test, and poor Dutch language skills. See Fig. 1 for the patient flow, and Table 1 for the patient characteristics of the 10 patients included. During the study period, all candidates continued to receive treatment as usual (TAU) for their psychosis, aimed at stabilizing their psychiatric condition; TAU included case management and medication in all cases.

TREATMENT

Each psychological PTSD treatment (PE or EMDR) comprised a maximum of twelve 90-minute sessions. Provided a patient achieved Posttraumatic Stress Symptom Scale, Self-Report (PSS-SR) scores

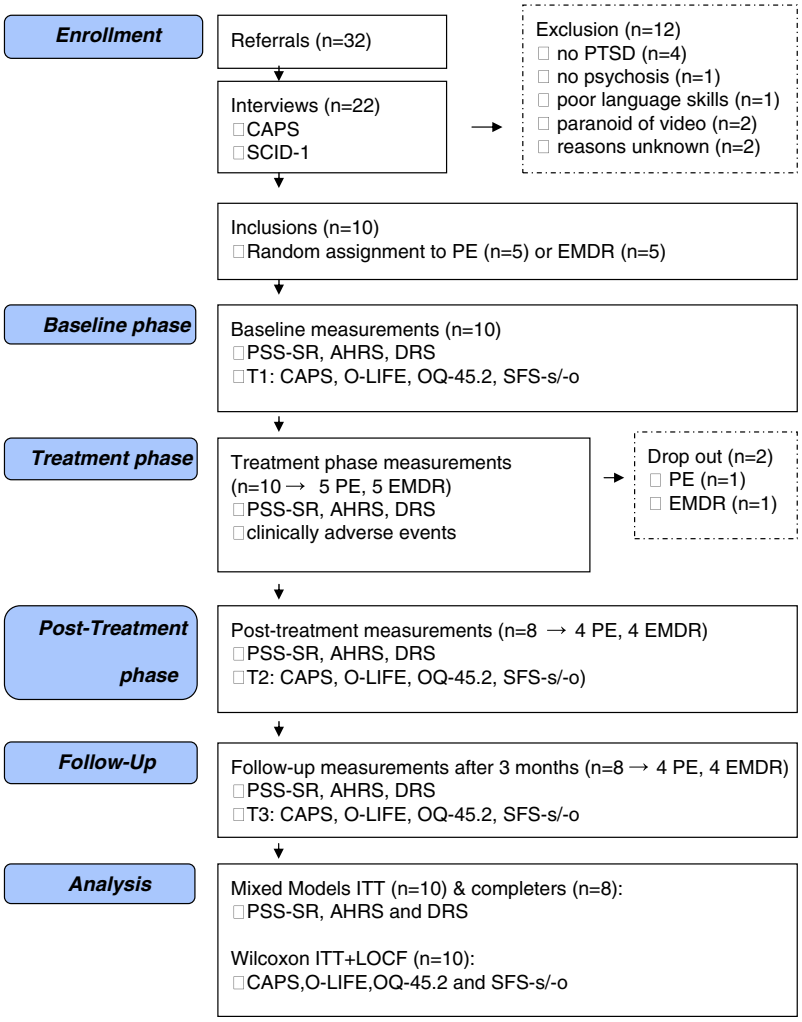

FIGURE 1 Flow diagram. *Note.* AHRS = PSYRATS Auditory Hallucination Rating Scale, CAPS = Clinician-Administered PTSD Scale, DRS = PSYRATS Delusion Rating Scale, EMDR = eye movement desensitization and reprocessing, ITT = intention to treat, LOCF =, O-LIFE = Oxford–Liverpool Inventory of Feelings and Experiences, OQ-45.2 = Outcome Questionnaire, PE = prolonged exposure, PSS-SR = Posttraumatic Stress Symptom Scale Self-Report, SCID-I = Structured Clinical Interview for DSM-IV, SFS = Social Functioning Scale.

Table 1
Patient Characteristics

Patient Number[a]	Sex	Age	Yrs of Treatment	Diagnosis	Ethnic Group	Trauma 1[b]	Trauma 2	Trauma 3
1	F	49	12	Schizophrenia	Dutch	V	CEA	n.a.
2	F	28	2	Schizophrenia	Dutch	V	n.a.	n.a.
3	F	48	>20	Psychosis NOS	Dutch	ASA	CEA	n.a.
4	F	49	>20	Psychosis NOS	Dutch	CSA	CPA	CEA
5	M	26	13	Schizophrenia	Bosnian	W	n.a.	n.a.
6	F	44	3	Schizoaffective	Dutch	CPA	CEA	n.a.
7	F	33	2	Psychosis NOS	Dutch	CPA	CEA	n.a.
8	M	48	10	Psychotic bipolar	Dutch	CSA	CEA	n.a.
9	F	56	20	Schizophrenia	Dutch	ASA	APA	CEA
10	F	55	16	Psychosis NOS	Dutch	CSA	CEA	CB

Note. APA = adult physical abuse, ASA = adult sexual abuse, CB = childhood bullying, CEA = child emotional abuse, CPA = child physical abuse, CSA = child sexual abuse, n.a. = not applicable, V = violence, W = war.
[a] Patients 1–5 received prolonged exposure and patients 6–10 received EMDR.
[b] DSM-IV-TR PTSD A-criterion: traumatic incidents (maximum of three) reported on the Clinician-Administered PTSD Scale (CAPS); there was no second or third A-criterion trauma.

below 10 in three consecutive sessions, early completion was allowed.

Both treatments were given by the first author (PdB), a licensed clinical psychologist and psychotherapist with extensive experience in the two treatment modalities. For supervision purposes, all sessions were videotaped; treatment integrity was monitored by the coauthors, both experts in their respective fields (AvM exposure; AdJ EMDR).

In the first session, the treatment rationale was presented to the patient. The target trauma was identified (or, in the case of multiple traumatic events, the trauma that would be the focus of treatment). PE and EMDR treatment were delivered according to the standardized treatment protocols outlined below.

Prolonged Exposure

In accordance with the treatment manual (Foa et al., 2007), the first PE session was dedicated to the treatment rationale, psychoeducation, and trauma identification. Next, the therapist and patient agreed on the hierarchical ordering of memories according to their relevance to the PTSD. Each subsequent session comprised 60 minutes of prolonged imaginal exposure in which the patient was helped to process the traumatic memory and emotions associated with it by describing the event to the therapist. He or she was encouraged to revisit the memories of the trauma, and to recount, in the present tense, the most frightening parts of the traumatic memory in all sensory details.

Each imaginal exposure session was recorded on audiotape, and the patient was asked to listen to that week's tape 5 days a week at home. From Session 2 in vivo exposure to feared but safe trauma-related stimuli was added. The homework assignments were discussed and assessed at the start of each session. Patients were also asked to monitor distress levels at home using the Subjective Units of Distress scale before, during, and after each exposure task, and also to record any changes in their cognitive and/or emotional responses to the stimuli they feared. The rationale behind this was that the main mechanism in PE is thought to be fear extinction: exposure enables patients to engage emotionally in the traumatic memories, and to process them by emotionally experiencing that confronting trauma stimuli in imagination and in vivo is safe (see Foa et al., 2007, for more details).

Eye Movement Desensitization and Reprocessing

EMDR is a protocolized psychotherapeutic approach intended to resolve the symptoms that can result from disturbing and unprocessed life experiences (Shapiro, 2001). Following the Dutch translation of the EMDR protocol (De Jongh & Ten Broeke, 2003) the treatment rationale, psychoeducation, and treatment planning were addressed in the first EMDR session. The subsequent sessions focused on a patient's traumatic memories. First the patient was asked to recall the memory of a particular traumatic event. He or she was then asked to concentrate on specific aspects of it, particularly (a) its most distressing "image"; (b) the cognition associated with it, that is, the patient's negative or dysfunctional belief of him- or herself; and (c) the accompanying emotions and physical responses.

At the core of the EMDR technique is the principle of taxing the working memory. In this study, this was operationalized by distracting the patients with loud audio tones (clicks) bilaterally through a headphone while they were mentally confronting the most disturbing part of the traumatic event. The patient was asked to focus on the clicks and to concurrently report emotional, cognitive, and/or somatic experiences. This procedure has been found to resolve patients' fearful and negative responses to the traumatic memories, enabling them to develop strong, positive beliefs about themselves (Jeffries & Davis, 2012).

To help foster closure, each session ends on a positive note. Homework between sessions is not a standard part of EMDR. Its underlying adaptive information processing theory (Shapiro, 2001) has been supported by experimental studies that showed that the vividness and emotionality of aversive memories was reduced by eye movements during their recall (Engelhard, van den Hout, & Smeets, 2011; Gunter & Bodner, 2008). While it has been questioned whether the effects of EMDR can be attributed to the eye movements or to exposure and cognitive restructuring (Davidson, 2001), a recent review and a meta-analysis provided evidence that eye movements and other exposure-based components have differential effects on traumatic memories (Jeffries & Davis, 2012; Lee & Cuijpers, 2013).

MEASURES AND DESIGN

Primary Outcome: PTSD Measures

To monitor weekly changes in PTSD symptoms throughout the study, the PSS-SR (Foa, Riggs, Dancu, & Rothbaum, 1993) was used. The 17 items of the PSS-SR correspond to the 17 diagnostic DSM-IV-TR criteria for PTSD. The PSS-SR total score ranges from 0 to 51. The self-report scale has been shown to have good reliability and validity (Foa et al., 1993; Dutch version: alpha = .85; Engelhard, Arntz, & van den Hout, 2007).

The CAPS (Blake et al., 1995; Dutch version: Hovens et al., 2005) was used to check the diagnostic criteria for PTSD and to assess the severity of PTSD symptoms. The CAPS rates the frequency and intensity of the DSM-IV-TR criteria; its total score ranges from 0 to 136. The reliability, validity, and sensitivity of the CAPS are good (Weathers, Keane, & Davidson, 2001; Dutch version: reliability alpha = .93 to .98; Hovens et al., 1994). The CAPS was administered at the baseline, posttreatment, and 3-month follow-up assessments.

Secondary Outcome: Safety Measures

In this study we considered treatment to be unsafe if psychotic symptoms and symptoms of general psychopathology were exacerbated, if the level of social functioning decreased, and if clinically adverse events occurred as a consequence of the intervention. We therefore checked for these signs weekly during the treatment.

Psychotic symptom severity. Psychotic symptom severity was monitored weekly by means of the Psychotic Symptom Rating Scale interview (PSYRATS; Haddock, McCannon, Tarrier, & Faragher, 1999). The interview uses the Auditory Hallucination Rating Scale (AHRS; 11 questions, total score range 0–55) to help establish the occurrence and severity of hallucinations, and the Delusion Rating Scale (DRS; 6 questions, total score range 0–30) to establish the occurrence of delusions. With regard to the hearing of voices, the AHRS assesses the frequency, duration, location, loudness, causal attribution, negative content, severity of negative content, the extent and severity of discomfort and suffering, any disruption of daily life caused by hearing voices, and any experience of control over voices. The DRS assesses the extent and duration of preoccupation with the delusion, the credibility of the delusion, the extent and severity of discomfort and suffering, and the disruption of daily life caused by the delusions. All PSYRATS items are scored from 0 (*not*) to 5 (*continuously*). Interrater reliability (AHRS alphas .78–.1.00; DRS alphas .88–.99) and validity were found to be good to excellent (Haddock et al., 1999).

Psychosis proneness. Proneness to psychosis was assessed by the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE; Mason, Claridge, & Jackson, 1995). The O-LIFE considers unusual experiences, cognitive disorganization, introvertive anhedonia, and impulsive nonconformity. Its total score ranges from 0 to 104, its test–retest reliability was found to be high (alpha > .70; Burch, Steel, & Hemsley, 1998), and its validity was shown to be good (Mason & Claridge, 2006). The O-LIFE was administered at the baseline, posttreatment, and 3-month follow-up assessments.

General psychopathology and distress. We used the Dutch version of the Outcome Questionnaire (OQ-45.2; Lambert et al., 1996) to assess psychiatric symptom distress (25 items), interpersonal relations (11 items), and social role functioning (9 items). The QQ-45.2 provides a total score that ranges from 0 to 180. The original and the Dutch versions have both been found to have high reliability and good validity (De Jong et al., 2007; Lambert et al., 1996; alpha = .68 and .95). The OQ-45.2 was administered at the baseline, posttreatment, and 3-month follow-up assessments.

Social functioning. This was checked using the Social Functioning Scale (SFS; Birchwood, Smith, Cochrane, Wetton, & Copestake, 1990), a 79-item scale designed to index social functioning in schizophrenia. It is completed independently twice: once by the patient ("self-report"), and once by someone close to the patient, for example, a family member ("other report"). The SFS total score ranges from 9 to 197; subscale raw scores may be converted to scale-score equivalents ($X = 100$, $SD = 15$). The SFS scales have been shown to be reliable (alphas .69–.78), valid, and sensitive measures of social functioning (Birchwood et al., 1990). The SFS was administered at the baseline, posttreatment and three-month follow-up assessments.

Clinically adverse events. Undesirable effects that were potentially related to the PTSD treatment were screened for every session. At the beginning of each session, the patient was asked about (a) hospital admissions, (b) suicidal behavior and nonsuicidal self-injury, (c) changes in medication (nonprescribed medication, or the need for more medication), or (d) crisis interventions provided by caregivers in the past week.

Design

The enrollment phase (Fig. 1) was completed with the random assignment of the 10 patients to either PE or EMDR treatment. Next, the $N = 10$ study was designed to allow the treatment phase, posttreatment, and follow-up scores to be compared with baseline scores. The design has two arms of measurements, with within-group controlled observations in each of the two arms: one controlled arm of 20 weeks repeated measurement, and one controlled arm with three time points of measurements (see Table 2).

In the first arm the severity of PTSD symptoms, hallucinations, and delusions were assessed repeatedly per patient in a multiple baseline design (Table 2): 20 times within a 20-week study period, and once again at 3-month follow-up. Each baseline phase length was randomly assigned to one participant in the PE treatment, and to one participant in the EMDR treatment. During treatment per patient a maximum of 12 assessments took place. On a group level this repeated measurements design plans for the comparison of 40 observations in the baseline, 120 observations in the treatment, 40 observations in the posttreatment, and 10 observations at follow-up. The grouped baseline observations served as the control condition in the comparison.

In the second arm the PTSD diagnosis (CAPS), level of social functioning (SFS), psychosis-prone thinking (O-LIFE), and general psychopathology (OQ-45.2) were assessed at three single time points: at baseline, after treatment, and at 3-month follow-up (Table 2). Adverse events were monitored in each session throughout the treatment phase.

Table 2
Measurement Planning in the Two Controlled Arms: (1) Repeated Measurements in a Multiple Baseline Design and (2) Measurements at Three Time Points

Arm	Planning of Measurements
1. [a]Baseline a	BB | TTTTTTTTTTT | PPPPPP | 3 months | FU
Baseline b	BBB | TTTTTTTTTTT | PPPPP | 3 months | FU
Baseline c	BBBB | TTTTTTTTTTT | PPPP | 3 months | FU
Baseline d	BBBBB | TTTTTTTTTTT | PPP | 3 months | FU
Baseline e	BBBBBB | TTTTTTTTTTT | PP | 3 months | FU
2. [b]Three time points	T1 | (*treatment*) | T2 | 3 months | T3

Note. B = baseline; 2–6 weeks, FU = follow-up; 3 months, P = posttreatment; 2–6 weeks, T = treatment; 12 weeks.
[a] Each baseline phase length was randomly assigned to one participant in the PE treatment, and to one participant in the EMDR treatment.
[b] For all $N = 10$ patients: T1 = baseline, T2 = posttreatment, T3 = 3 months follow-up.

Data Analysis

Primary outcome measures. First, changes in PTSD symptom severity as assessed with the PSS-SR were analyzed using the mixed-model procedure in SPSS, which allows all the individually varying number of observations within each phase to be entered into the analysis, producing an estimated marginal mean (EMM) for each phase. Scores obtained within the four phases (baseline, treatment, posttreatment, and follow-up) were defined as the main fixed effects. Patients were defined as the random factors within the phases, that is, each score (observation) obtained from the patients in each phase was considered as a random sample of possible scores. The random-effect covariance matrix was specified as ar1 (first-order autoregressive). To assess treatment effect on the PSS-SR, the EMMs computed for the treatment, posttreatment, and follow-up phases were each compared with the baseline EMM. All analyses were conducted using PASWS Statistics Version 18.0.3 (SPSS Inc., Chicago, Il). Effect sizes were calculated with the formula $r =$ (EMM baseline – EMM x)/Sd, an estimator used in parametric statistics. The individual graphs of PSS-SR symptom changes during and after the active treatment phase were inspected visually for changes relative to the baseline phase.

Second, using Wilcoxon pairwise tests, the CAPS PTSD total scores of the intention-to-treat (ITT) group at posttreatment (T2) and follow-up (T3) were compared with those at baseline (T1), and the follow-up (T3) scores were compared with posttreatment (T2) scores. The reason we used the

nonparametric Wilcoxon pairwise testing, was first because the distribution of scores did not meet the criteria for parametric testing, and second because of the small number of observations per phase (10 in T1, 8 in T2, and 8 in T3). Effect sizes were calculated with the formula $r = Z/\sqrt{N}$, which is an estimator used in nonparametric statistics (Wilcoxon tests). Third, we compared posttreatment (T2) and follow-up (T3) CAPS PTSD diagnoses with baseline values (T1).

Secondary outcome measures. To analyze the adverse effects of the treatment in terms of psychotic symptom severity as assessed with the PSYRATS (AHRS and DRS), we used the mixed-model procedure described above. We also inspected the graphs for changes in psychotic symptoms. Adverse treatment effects on the O-LIFE, OQ-45.2, and SFS were tested using Wilcoxon pairwise tests to compare baseline (T1) to posttreatment (T2) and follow-up (T3), and the follow-up (T3) scores to posttreatment (T2) scores.

Comparison of PE and EMDR. We compared PE and EMDR in terms of PTSD end-state diagnosis, treatment dropout, early completion, and serious adverse events.

Results

TREATMENT COMPLETION

Two of the 10 patients who started treatment dropped out prematurely (one in PE, one in EMDR). Two PE participants completed their treatment early (one in Session 5 and one in Session 7), as did one EMDR participant (in Session 10). The mean number of sessions was 9 in PE, and 11.5 in EMDR.

Primary Outcomes: PTSD

PSS-SR symptom severity. See Table 3 for descriptive statistics. All patients scored above the clinical cutoff score of 14 for the PSS-SR at baseline (Wohlfarth, van den Brink, Winkel, & ter Smitten, 2003); the high mean baseline PSS-SR scores indicated a severity of PTSD symptoms that is comparable with other severely mentally ill populations (e.g., Cloitre et al., 2010).

The mixed-model analysis showed that, given the TAU baseline phase as the statistical control condition, PTSD symptom severity in the ITT group had decreased significantly in the treatment phase ($p < .001$, $r = .64$), and that this effect was maintained in the posttreatment phase ($p < .001$, $r = .73$) and follow-up phase ($p < .001$). The significant F value reflects the effect of the phases, $F(3, 56.998) = 13.2$, $p < .001$.

For the completers ($N = 8$; see Table 3), the decrease in PTSD symptom severity in the treatment phase was significant, showing large treatment effects ($p < .001$, $r = 1.21$) that were sustained posttreatment ($p < .001$, $r = 1.39$) and during follow-up ($p < .001$). The significant F value reflects the effect of the phases, $F(3, 49) = 12.53$, $p < .001$.

Fig. 2 shows the individual PSS-SR graphs. After the start of treatment, PTSD symptoms decreased in Patients 1, 2, 5, 7, 8, and 9. Patients 3 and 6 dropped out. The effects in Patients 4 and 10 were ambiguous.

CAPS symptom severity. See Table 3 for descriptive statistics. At follow-up, CAPS total scores were significantly lower than the pretreatment values ($Z = –2.52$, $p = .012$, effect size $r = .63$). The posttreatment total scores had also decreased, but only reached borderline significance ($Z = –1.96$, $p = .05$, $r = .49$).

CAPS end-state functioning. At the posttreatment (T2) assessment, six of the eight completers no longer met the criteria for a PTSD diagnosis; at follow-up (T3) this was the case with seven of the eight completers.

SECONDARY OUTCOMES: SAFETY

Psychotic symptom severity. See Table 3 for descriptive statistics. The mixed-model analysis of the PSYRATS values showed no significant phase effects of PTSD treatment on the EMMs of auditory hallucinations (AHRS), ITT: $F(3, 60) = .86$, $p = .466$, completers, $F(3, 50) = .73$, $p = .54$); or on delusions (DRS), ITT: $F(3, 49) = 1.77$, $p = .165$, completers, $F(3, 48) = 1.57$, $p = .21$.

See Figs. 3 and 4 for the individual AHRS and DRS graphs. During the treatment phase, nine patients had no increase in psychotic symptoms. Note that Patients 3 and 6 dropped out of treatment. Patient 4 had a sudden increase in auditory hallucinations, which she attributed to a stressful life event in her family. Before this incident, PE had not provoked any hallucinations.

Psychosis proneness. See Table 4 for the descriptive statistics. Pre-to-post analyses (T1–T2) yielded a significant decline in psychosis-prone thinking ($Z = –2.05$, $p = .041$, $r = .65$). Changes from baseline (T1) to follow-up were not significant (T3; $Z = –1,75$, $p = .080$, $r = .55$).

General psychopathology and distress. See Table 4 for the descriptive statistics. Relative to the scores at baseline (T1), OQ-45.2 posttreatment (T2) total scores were significantly lower ($Z = –2.19$, $p = .028$, $r = .69$). So, too, were the follow-up (T3) scores ($Z = –2,37$, $p = .018$, $r = .75$).

Table 3
Estimated Marginal Means (EMM) and Standard Errors (SE) for the Mixed-Models Analysis for the Session-to-Session Analyses of PTSD (PSS-SR Self-Report) and for Verbal Hallucinations and Delusions (AHRS and DRS Interviews) During the Four Study Phases (Intention to Treat $N = 10$, Treatment Completers $N = 8$, PE and EMDR Together)

Phases of the Study								
	Baseline		Treatment		Posttreatment		Follow-up	
PSS-SR	*EMM*	*SE*	*EMM*	*SE*	*EMM*	*SE*	*EMM*	*SE*
$N = 10$	31.92	5.30	21.97**	4.67	15.67**	4.68	14.06**	4.68
$N = 8$	30.22	5.75	19.49**	5.11	14.13**	4.97	12.75**	4.94
AHRS	*EMM*	*SE*	*EMM*	*SE*	*EMM*	*SE*	*EMM*	*SE*
$N = 10$	18.71	8.19	18.06	7.27	13.89	7.17	16.65	7.09
$N = 8$	14.54	9.49	14.37	8.50	10.67	8.14	13.62	8.02
DRS	*EMM*	*SE*	*EMM*	*SE*	*EMM*	*SE*	*EMM*	*SE*
$N = 10$	6.28	2.61	5.09	2.23	1.94	2.46	1.97	2.53
$N = 8$	5.68	2.76	3.91	2.37	1.49	2.49	1.75	2.55

Note. PSS-SR = Posttraumatic Stress Symptom Scale Self-Report, AHRS = PSYRATS Auditory Hallucination Rating Scale, DRS = PSYRATS Delusion Rating Scale.
** = $p \leq .01$ relative to the baseline phase.

Social functioning. See Table 4 for the descriptive statistics. Posttreatment and follow-up values were not significantly different from those at baseline (T1, T2, T3 all $p > .05$).

Clinically adverse events. During the treatment phases, no negative effects occurred (i.e., no hospital admissions, suicidal behavior/nonsuicidal self-injury, changes in medication, or crisis interventions by caregivers).

Comparison of PE and EMDR. Of the eight completers, three patients in the PE treatment and three in the EMDR treatment no longer met the criteria for a PTSD diagnosis as assessed with the CAPS at T2 (posttreatment). At T3 (follow-up), this was four in the PE treatment and three in the EMDR treatment.

The graphs in Figs. 2, 3, and 4 show that patient response to PE and EMDR was comparable: PTSD symptoms decreased clearly after the start of treatment in three patients in both PE and EMDR (Patients 1, 2, and 5 in PE, and Patients 7, 8, and 9 in EMDR), and these effects were sustained. At first, Patient 4 (PE) improved, but this was not sustained after Session 6—possibly because, as stated above, to the occurrence of a stressful life event in the patient's family. Patient 10 (EMDR) also improved, but this was less pronounced. Symptoms of psychosis increased in one patient in PE, and in none of the patients in the EMDR.

Discussion

As in previous studies (Frueh et al., 2009; Van den Berg & van der Gaag, 2012), our results suggest that PE and EMDR—both evidence-based trauma-focused treatments—are effective in reducing PTSD symptoms in patients with a psychotic disorder. Seven of the 8 patients (87.5%) who completed the treatment, and 7 of the 10 patients starting treatment (70%) no longer fulfilled the CAPS diagnostic criteria for PTSD, showing that both PE and EMDR were highly effective. As the controlled design of the trial allowed statistical hypothesis testing of treatment efficacy, it is plausible that the significant decrease in PTSD symptoms was a result of the interventions, with effects being maintained for 3 months after the end of treatment. Comparison of the two treatments (PE and EMDR) revealed no significant or marked qualitative differences, either in terms of effect, or in terms of safety.

As stated in the introduction, many therapists tend to refrain from trauma-focused interventions for fear that such approaches are too burdensome to the psychotic patient, or are even harmful (Becker et al., 2004; van Minnen et al., 2010). As none of our patients showed a treatment-related increase in psychotic or other psychopathological symptoms, and as we found no signs of a deterioration in social functioning or of clinically adverse events, our findings demonstrate that PE and EMDR can be used safely for patients with psychosis.

Nonetheless, one patient who started treatment without psychotic symptoms reported sudden hallucinations during the second half of treatment, and a very mild delusion at one assessment. Although this patient claimed that the incidents were not related to the intervention, future studies will need to compare

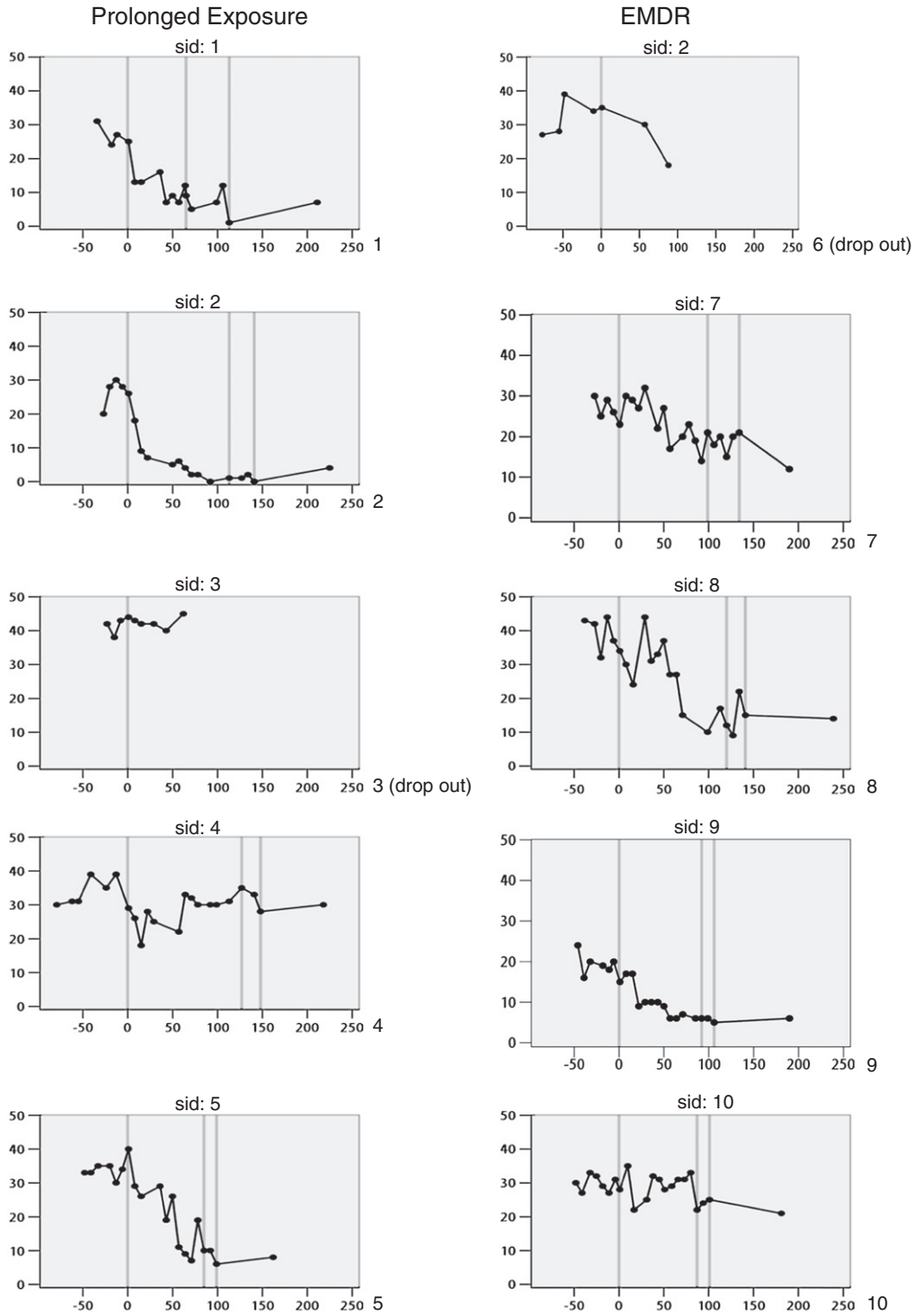

FIGURE 2 Primary outcome variable of PTSD: PSS-SR symptom severity. Session scores across baseline, treatment, posttreatment, and follow-up (phases divided by vertical lines; *x*-axis = number of days; *y*-axis = outcome scores).

the proportion of patients in whom such psychotic symptoms increase during trauma-focused treatment with the incidence of similar symptoms in patients not receiving treatment.

Overall, the significant improvements posttreatment with regard to psychosis-prone thinking and general psychopathology are interesting: they indicate that, rather than increasing, these comorbid symptoms tended to decrease together with the PTSD symptoms. Our finding that treatment had no positive effects on hallucinations and delusions (as assessed with the AHRS and DRS), may have

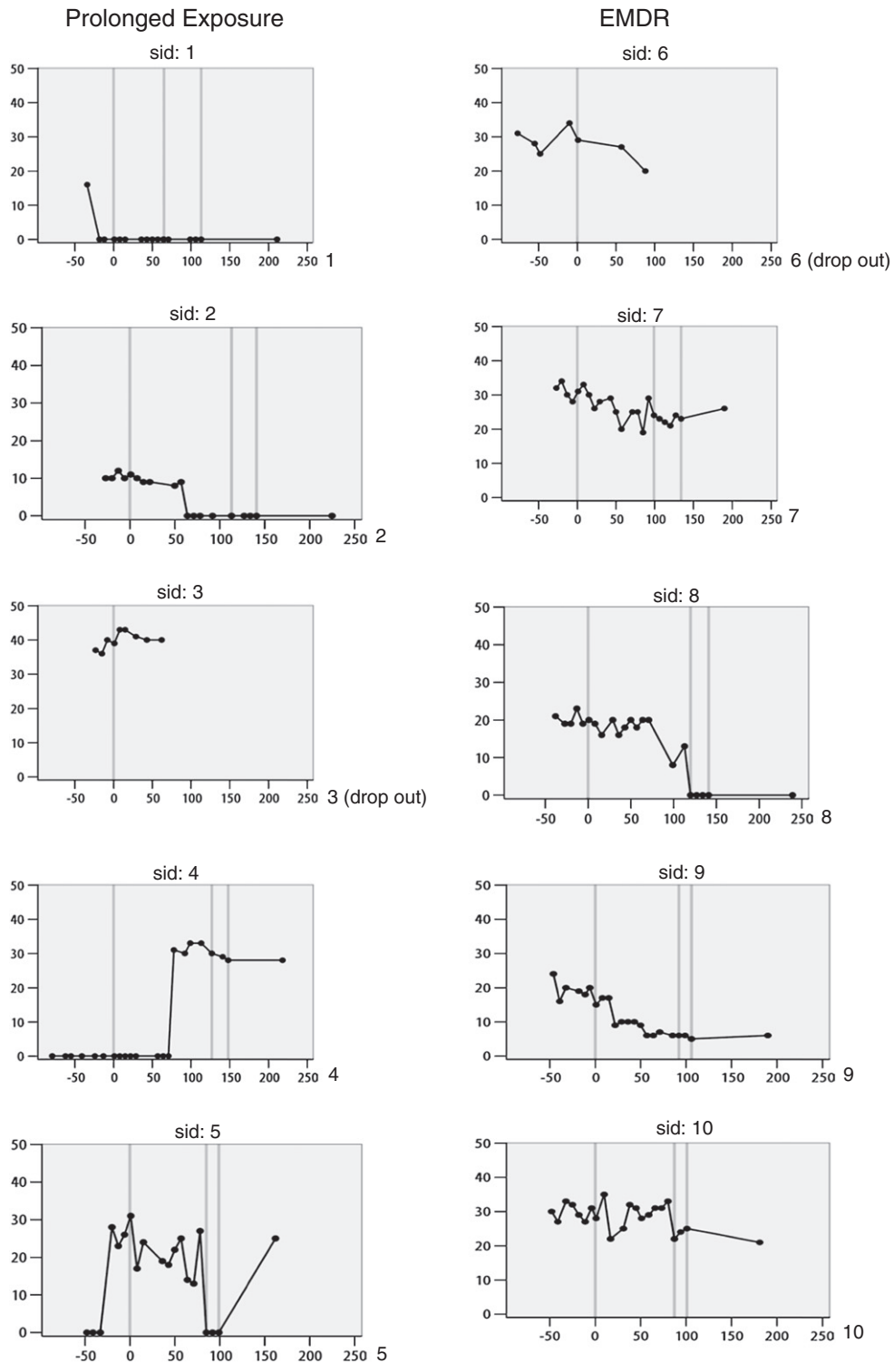

FIGURE 3 Secondary outcome safety variable of hallucinations: PSYRATS-AHRS symptom severity. Session scores across baseline, treatment, posttreatment, and follow-up (phases divided by vertical lines; *x*-axis = number of days; *y*-axis = outcome scores).

been due to a floor effect, as the overall scores on these measures were already relatively low at the start of treatment.

Both intervention modalities were well accepted, and most patients were able to comply with the treatment sessions. For trauma-focused treatments, the 20% dropout rate is acceptable (Bisson & Andrew, 2009; Hembree et al., 2003).

While one patient ended treatment prematurely due to very hostile verbal and visual hallucinations,

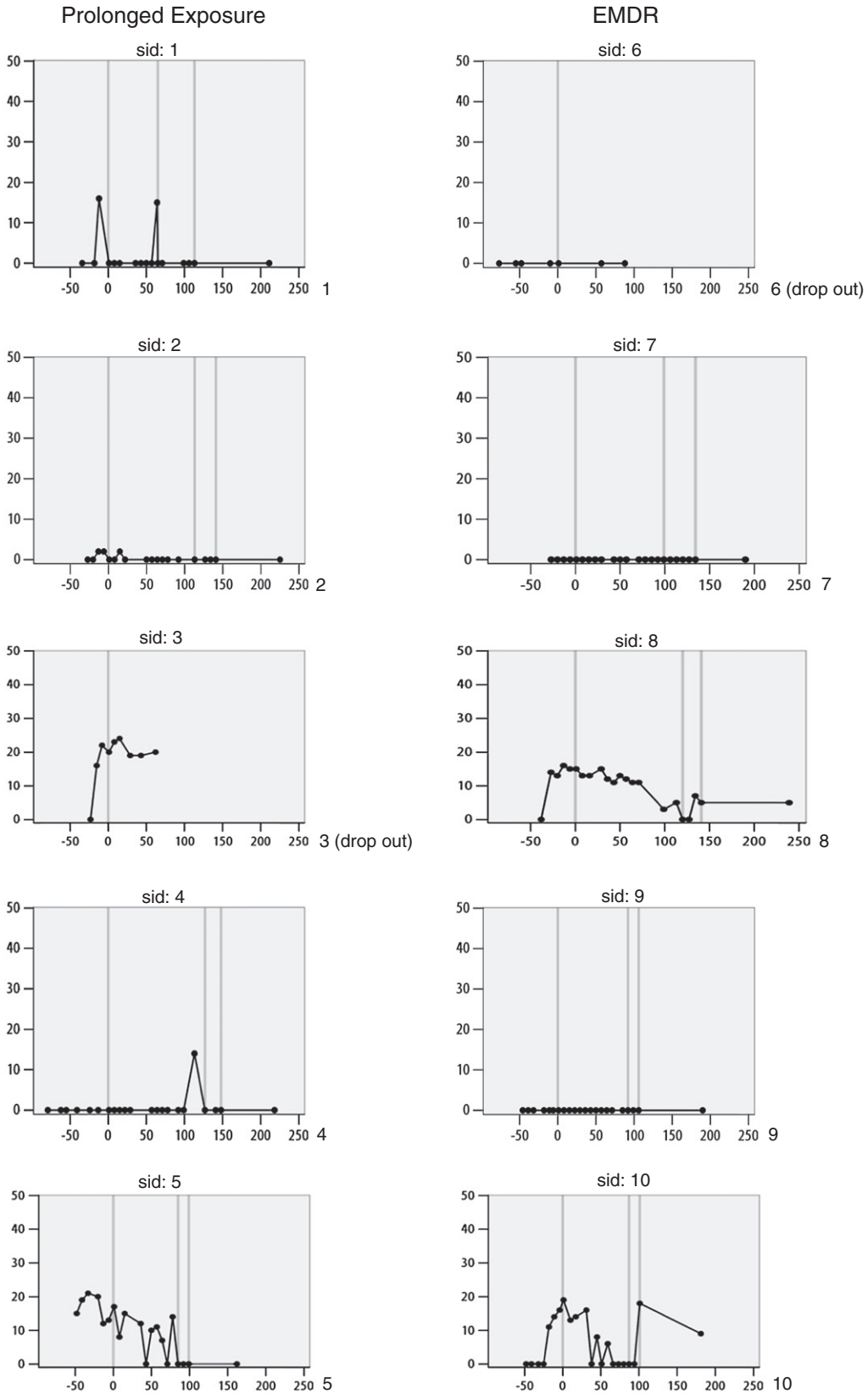

FIGURE 4 Secondary outcome safety variable of delusions: PSYRATS-DRS symptom severity. Session scores across baseline, treatment, posttreatment, and follow-up (phases divided by vertical lines; *x*-axis = number of days; *y*-axis = outcome scores).

Table 4
Means, Medians, and SDs for Wilcoxon Pairwise Tests for the Primary and Secondary Outcome Measures (Intention to Treat N = 10, PE and EMDR Together)

	T1(Baseline)			T2 (Posttreatment)			T3 (Follow-up 3 months)		
	Mean	Median	*SD*	Mean	Median	*SD*	Mean	Median	*SD*
CAPS	71.20	72.50	22.49	48.20*	35.50	38.29	37.60*	25.50	34.68
O-LIFE	51.90	58.00	19.95	45.90*	52.00	20.78	48.10	55.00	21.51
OQ-45.2	89.60	93.50	33.56	77.40*	79.00	36.86	75.30*	74.00	33.08
SFS-self	104.70	112.50	27.80	107.80	112.00	29.74	105.80	108.00	28.26
SFS-other	102.60	100.00	26.86	107.80	108.50	27.53	104.60	108.00	28.24

Note. CAPS = Clinician-Administered PTSD Scale, O-LIFE = Oxford-Liverpool Inventory of Feelings and Experiences, OQ-45.2 = Outcome Questionnaire, SFS = Social Functioning Scale.
* $p \leq .05$ relative to T1.

these hallucinations had not been provoked or intensified by the treatment: the patient had been having such hallucinations for many years, and their presence had already been established in the baseline phase. Nonetheless, her delusions did seem to have been mildly provoked by the treatment: her hallucinations had "forbidden" her to talk about the traumatic events for many years, and were now exacerbating her fear. Even though she was highly motivated, she was afraid to subject herself to treatment—a dilemma that may be specific to this particular patient population, and may be the only argument, if a crucial one—in favor of modifying the PE or EMDR protocols accordingly.

Not all patients suffered from auditory hallucinations. Hallucinations in other modalities have not been monitored. Monitoring hallucinations in all modalities, especially visual hallucinations (Bentall et al., 2012) certainly is recommendable for future research.

The protocols we used in this study were not modified for the study group. With the exception of some basic anxiety management strategies that are integral to the treatments, neither did they include any of the elements that are routinely incorporated in many PTSD approaches, such as pretreatment stabilization modules, skill-coping training, or relaxation exercises.

Our trial has a relatively low dropout. This may be explained by the absence of a preparatory phase before the actual trauma-focused interventions: a previous study (Frueh et al., 2009) showed that most patients dropped out in the skills-training phase that preceded the PE sessions.

The present study focused specifically on PE and EMDR. But we acknowledge that cognitive processing and stress management, and combinations of typical CBT interventions, may have potential for the successful treatment of PTSD in this comorbid population (see Jackson et al., 2009; Mueser et al., 2008).

Two important limitations of this study should be mentioned. The first is the small sample size, which clearly restricts the generalizability of the results. The second is the limited time frame—three months including follow-up only—this cannot show clearly whether the positive effects are sustained for a longer period.

In summary, this is the first controlled case study in which prolonged exposure and EMDR—two recommended evidence-based psychological treatments for PTSD—were successfully used in patients suffering from psychosis. It is our hope that the findings foster the inclusion of patients with psychosis in evidence-based trauma-focused treatments, both in empirical research and in clinical practice.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

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RECEIVED: September 21, 2012
ACCEPTED: July 11, 2013
Available online 27 July 2013