Assessment of Psychophysiological Stress Reactions During a Traumatic Reminder in Patients Treated With EMDR

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This study investigates changes of stress-related psychophysiological reactions after treatment with EMDR. Sixteen patients with posttraumatic stress disorder (PTSD) following type I trauma underwent psychometric and psychophysiological assessment during exposure to script-driven imagery before and after EMDR and at 6-month follow-up. Psychophysiological assessment included heart rate (HR) and heart rate variability (HRV) during a neutral task and during trauma script listening. PTSD symptoms as assessed by questionnaire decreased significantly after treatment and during follow-up in comparison to pretreatment. After EMDR, stress-related HR reactions during trauma script were significantly reduced, while HRV indicating parasympathetic tone increased both during neutral script and during trauma script. These results were maintained during the follow-up assessment. Successful EMDR treatment may be associated with reduced psychophysiological stress reactions and heightened parasympathetic tone.

Keywords: PTSD; EMDR; psychophysiology; heart rate; parasympathetic nervous system

Exaggerated psychophysiological arousal and increased startle reactions are constituent symptoms for the diagnosis of posttraumatic stress disorder (PTSD). Empirical data actually demonstrate elevated psychophysiological baseline parameters and excessive psychophysiological reactivity in patients who have PTSD (Orr & Roth, 2000). In this vein, several studies report elevated baseline HR in Vietnam veterans with PTSD (Gerardi, Keane, Cahoon, & Klauminzer, 1994; Keane et al., 1998) as well as in victims of motor vehicle accidents (Blanchard et al., 1996) when compared with normal controls. In addition, exaggerated startle reactions to loud tones or other distressing stimuli (Metzger et al., 1999; Shalev et al., 2000) and diminished habituation to repeated stimuli presentation (Shalev, Orr, Peri, Schreiber, & Pitman, 1992) have been found consistently. However, the most intense psychophysiological reactions are known to be elicited by reminders of individual traumatic memories (e.g., audiotaped trauma scripts), which therefore have been suggested to act as a specific diagnostic indicator of PTSD (Keane et al., 1998; Pitman, Orr, Forgue, de Jong, & Claiborn, 1987). From this perspective, there is strong supporting evidence for including psychophysiological measures into the outcome assessment for psychotherapeutic treatment in patients with PTSD.

Psychophysiological Outcome

Studies investigating the effects of cognitive-behavioral exposure treatment for Vietnam veterans with PTSD (Boudewyns & Hyer, 1990; Pitman et al., 1996) and civilian participants with PTSD (Shalev, Orr, & Pitman, 1992) have found decreased psychophysiological arousal during exposure to the traumatic memory after successful treatment. In addition, a series of treatment studies administering EMDR included psychophysiological measures. Three EMDR studies found treatment-related reductions of psychophysiological arousal in response to script-driven imagery (Carlson, Chemtob, Rusnak, Hedlund, & Muraoka,

 DOI: 10.1891/1933-3196.1.1.15

1998; Renfrey & Spates, 1994; Rogers et al., 1999). These studies had small sample sizes ranging from 6 to 8 patients treated with EMDR. Two studies found evidence for a pre- to posttreatment habituation of heart rate and electromyogram (Boudewyns, Stwertka, Hyer, Albrecht, & Sperr, 1993; Forbes, Creamer, & Rycroft, 1994), but these reductions in autonomic activity could not be attributed to treatment effects. Again, both studies employed relatively small sample sizes of 9 and 8 patients, respectively, in the treatment group. Finally, in a single-session design with continuous monitoring of autonomic variables, Wilson, Silver, Covi, and Foster (1996) reported a pre- to posttreatment reduction of HR and galvanic skin reactions in 17 patients treated with EMDR.

Measurement of Parasympathetic Tone

Autonomic regulation has recently become a focus of interest in psychophysiology since newer methods allow a quantification of the influence of both branches (parasympathetic and sympathetic) of the autonomic nervous system (Cacioppo et al., 1994). HR fluctuations related to inspiration and expiration, which are known as respiratory sinus arrhythmia (RSA), are highly correlated with the parasympathetic activity on the sinoatrial node of the heart. Studies administering pharmacological blockades demonstrate that cardiac parasympathetic tone is closely related to RSA (Akselrod et al., 1981; Cacioppo et al., 1994). Low parasympathetic tone has been identified not only as a risk factor for cardiovascular disease (Bonaduce et al., 1999) but also as a concomitant of affect-dysregulation and stress-related psychiatric diseases, such as depressive disorders and anxiety disorders (Gorman & Sloan, 2000). There is increasing evidence that low parasympathetic tone is an indicator for prefrontal cortex hypofunction associated with disinhibited defensive circuits and a dominance of amygdala-generated affect and distress (Thayer & Brosschot, 2005).

Initial studies in patients with PTSD found reduced parasympathetic tone when compared to controls (Cohen et al., 1997; Rothbaum, Kozak, Foa, & Whitaker, 2001). Low parasympathetic tone was also found to be associated with prolonged psychophysiological arousal in patients with PTSD during script-driven trauma imagery (Sack, Hopper, & Lamprecht, 2004). One study reported a normalization of parasympathetic tone during pharmacological treatment of PTSD by selective serotonin reuptake inhibitor–antidepressants (Cohen, Kotler, Matar, & Kaplan, 2000). The only study investigating the effects of psychotherapeutic treatment for PTSD on autonomic regulation (Nishith et al., 2003) found a pattern of enhanced parasympathetic tone during REM sleep after successful cognitive behavioral therapy.

The primary aim of the present study was to assess the feasibility of measuring autonomic stress reactivity during the course of treatment. We hypothesized that successful EMDR treatment would be associated with diminished HR reactions during a traumatic reminder, and that a posttreatment increase in parasympathetic tone would be observed.

Method

Participants

Sixteen outpatients (10 women, 6 men) who inquired about possible treatment for trauma-related psychological problems at a specialized trauma clinic participated in the study. All patients experienced Type I traumatizations in adulthood and fulfilled diagnostic criteria for PTSD as assessed by the PTSD module of the structured clinical interview for the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–IV*; First, Spitzer, Gibbon, & Williams, 1996). Five patients were victims of assault violence, 3 patients had motor vehicle accidents, 3 patients experienced sudden death of a relative, 3 female patients were victims of rape, and 2 patients reported work accidents. Exclusion criteria for participation in the study were severe dissociative symptoms, represented by a score greater than 30 on the Dissociative Experiences Scale (Bernstein & Putnam, 1986), as well as serious traumatizations during childhood. After a detailed clinical interview (which often took several sessions) the application of EMDR was proposed.

Participants ranged in age from 26 to 56 years, with a mean age of 40.5 years (*SD* 8.4); 9 were married, 6 single, and 1 divorced. Seven participants reported 13 years of formal scholarly education, 5 participants reported 10 years, and 4 participants reported 9 years. The ethnic background of all patients was White. When assessed with a diagnostic checklist (Hiller, Zaudig, & Mombour, 1995), 1 patient endorsed symptoms consistent with a diagnosis of agoraphobia and undifferentiated somatoform disorder; the other patients had no comorbid diagnosis.

Treatment

Trauma therapy with EMDR was carried out by the authors of the study strictly following the manualized standard protocol (Shapiro, 1995), although treatment adherence was not assessed. All therapists had completed EMDR Level II training and had several

years of experience in applying EMDR. Duration of the therapy followed patients' individual needs and was terminated when the participant rated the level of distress associated with the traumatic memory at a 0 or 1 on the Subjective Units of Distress (SUD) scale (Wolpe, 1969). Treatment focused on past trauma only. A mean number of 4.7 EMDR-treatment sessions (range 1 to 8 sessions) was administered.

EMDR is an information processing therapy (Shapiro, 2001; Shapiro & Maxfield, 2002) combining multiple brief exposures to traumatic memories with eye movements or other forms of bilateral stimulation (e.g., alternating tapping on hands or alternating clicking tones). The patient attends to past and present experiences in brief sequential doses while simultaneously focusing on an external stimulus (e.g., following the therapists moving fingers with the eyes). Then the patient is instructed to let new material become the focus during the next set of stimulation. This procedure is repeated until the trauma-associated distress is reduced to a minimum.

Assessment Procedures

The first author of the study conducted all diagnostic assessment including Structured Clinical Interview for DSM-IV Disorders–PTSD ratings. Psychophysiological testing was carried out before the first treatment session, 1 week after treatment, and during a 6-month follow-up. Trauma related symptoms were assessed with the Impact of Event Scale (IES; Horowitz, Wilner, & Alvarez, 1979) and the Posttraumatic Stress Diagnostic Scale (PDS; Foa, 1995) on each testing occasion. The IES is a standardized 15-item questionnaire asking for symptoms of reexperiencing and avoiding related to traumatic experiences. The IES is a well-validated and widely used questionnaire for assessing symptom severity in trauma-related disorders. The validated German version was used (Ferring & Fillipp, 1994). The PDS asks participants to rate the extent to which they experience each PTSD symptom specified in *DSM–IV*, ranging from 0 (never) to 3 (5 times per week or more/nearly always), and yields scores for total symptom severity and intrusions, avoidance, and hyperarousal subscales. Unpublished data from previous studies of our research group have shown acceptable psychometric properties of the German translation of the PDS.

Instruments and Script-Driven Imagery

An individual trauma script was prepared for each patient. The procedure initially described by Pitman, Orr, Forgue, de Jong, and Claiborn (1987) was modified using script-reading periods of 2-min duration instead of the typical 30 s, since pilot work indicated that some patients needed more than 30 s to get access of their traumatic memory. All trauma scripts were prepared by the first author (M. S.) and were described in the present tense and first person, sequentially unfolding details of the most disturbing traumatic event. Scripts were then read to the patient to check for any inconsistencies with his/her memories. A 2-min recording of the script was made on audiotape. Psychophysiological assessment was conducted in a second session, approximately 1 week after script preparation, which took place in the therapeutic environment familiar to the patients. Participants were seated in a comfortable chair and asked to sit still during the recording procedure. After electrocardiogram (ECG) electrode placement and a 5-min adaptation period, a sequence of five scripts was played back via tape recorder in a fixed order: (1) 2-min scripted relaxation exercise followed by a 1-min break; (2) 2-min neutral script of imagining washing dishes followed by 1-min break; (3) 2-min trauma script followed by a 5-min break; (4) repeat of relaxation script/exercise and 1-min break; and (5) repeat of neutral script. Levels of subjective discomfort (SUD) on a scale ranging from 0 (no distress at all) to 10 (the highest possible distress) were immediately assessed at the end of the trauma script. The ethics committee of Hanover Medical School, Germany, approved the design of the study. All participants gave their informed consent.

ECG signals were obtained via three commercial disposable Ag-AgCl electrodes placed on the chest; they were recorded in a miniaturized amplifier (Par-Port, Par-Elektronik, Berlin, Germany). Sampling rate of ECG data for acquisition of interbeat intervals (IBIs) was 1000 Hz. Data were transferred to a PC, and a time series of interbeat intervals was generated. Time series analysis was conducted to calculate RSA according to the procedure developed by Porges and Bohrer (1990). According to published recommendations (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996), RSA was defined as variations of interbeat intervals of HR in the frequency band between 0.12 and 0.40 Hz. This frequency band selectively reflects the activity of efferent fibers from the parasympathetic system originating in the nucleus ambiguus and is characterized by a respiratory rhythm. MXedit software (Delta-Biometrics, Bethesda, MD, USA) was used to visually display the heart period data, to edit outliers, and to quantify the heart period and the vagal tone index. Processing included resampling of HR period data every 500 ms and a detrending procedure with moving polynomial filter (3rd order 21-point).

Finally, a band pass filter was administered to restrict data to the frequency range of respiratory associated arrhythmia. The analysis represents the variance of the residual series output from the detrending algorithm and is reported in units of $\ln(ms)^2$. Except for frequently premature heartbeats in one case, which therefore had to be excluded, all ECG data were free from artifacts, and no further corrections were required.

Data Reduction and Statistical Analyses

Mean values of HR were calculated for the first 60 s of each script. RSA was computed for 2-min periods during both neutral scripts and trauma script. Mean HR and mean RSA from both neutral scripts served as comparison. After testing for normal distribution, analyses of variance over time were carried out for all variables with paired *t* tests (two tailed). Significance levels were set at .05 for all statistical analyses, which were performed using the SPSS 10 statistical package (SPSS Inc., Chicago, IL, USA).

Results

Dropouts

Two of the 16 participants (13%) dropped out during treatment phase. The (male) patient with comorbid anxiety disorder reported an intolerable increase of anxiety symptoms following the first EMDR session; therefore, trauma exposure had to be stopped and replaced by stabilization. The second (female) patient noted no subjective improvement during the first 2 therapy sessions and terminated thereafter. One patient had to be excluded from psychophysiological data analysis due to frequent premature heartbeats. Due to organizational shortcomings, one patient did not complete the posttreatment assessment but did finish follow-up, and another patient did not complete follow-up. One patient completed psychophysiological data at follow-up, but questionnaire data were lost. There were no significant differences in pretreatment questionnaire measures or in levels of psychophysiological response between dropouts or participants with data loss ($N = 5$) and participants completing posttreatment as well as follow-up assessment ($N = 11$).

Questionnaires

Pre- versus posttreatment and pretreatment versus follow-up comparisons revealed highly significant decreases in posttraumatic symptoms as measured by IES and by PDS. While pretreatment scores of IES and PDS indicated pathological symptom severity (cutoff IES: 27, PDS: 1.2) posttreatment and follow-up mean scores of both questionnaires in our sample reached subclinical levels. Comparison of posttreatment versus 6-month follow-up showed no significant differences in all questionnaires (see Table 1 for details).

Effect sizes for comparison of treatment effects were computed as standardized mean differences by dividing the difference of pre- and posttreatment mean values by the square root of pooled squared standard deviations. For the comparison of pre- versus posttreatment and pretreatment versus follow-up, the following effect sizes resulted: Impact of Event Scale: 1.80 and 1.74, respectively; Posttraumatic Diagnostic Scale: 1.21 and 1.31, respectively. The highest treatment effects were found for IES-intrusion, with effect sizes of 2.01 and 1.73, respectively, and PDS-intrusion, with 1.30 and 1.58, respectively.

TABLE 1. Psychometric Variables

	Pre ($N = 16$)	Post ($N = 12$)	Follow-Up ($N = 11$)	Comparison Pre/Post		Comparison Pre/Follow-Up	
	Mean (*SD*)	Mean (*SD*)	Mean (*SD*)	*t* (*df*)	Significance	*t* (*df*)	Significance
IES total score	47.3 (13.9)	19.6 (17.3)	22.6 (14.7)	5.1 (11)	$p < .001$	5.3 (10)	$p < .001$
IES intrusions	25.3 (5.7)	9.9 (9.7)	11.7 (10.3)	6.1 (11)	$p < .001$	4.5 (10)	$p = .001$
IES avoidance	22.0 (11.5)	9.7 (9.6)	10.8 (7.1)	2.9 (11)	$p = .015$	4.2 (10)	$p = .002$
PDS total score	1.75 (0.51)	1.00 (0.74)	0.95 (0.74)	5.5 (11)	$p < .001$	6.0 (10)	$p < .001$
PDS intrusions	1.92 (0.52)	1.05 (0.83)	0.95 (0.73)	5.1 (11)	$p < .001$	4.9 (10)	$p = .001$
PDS avoidance	1.77 (0.68)	0.89 (0.72)	0.96 (0.90)	5.0 (11)	$p < .001$	5.3 (10)	$p < .001$
PDS arousal	1.51 (0.58)	1.10 (0.79)	0.93 (0.73)	2.7 (11)	$p = .021$	3.4 (10)	$p = .007$

Note. SD = standard deviation; IES = Impact of Event Scale; PDS = Posttraumatic Diagnostic Scale. Statistical comparison: paired *t* test (two tailed).

Subjective Distress During Trauma Script

Subjective distress during presentation of the individualized trauma script decreased significantly in pre- and posttreatment comparison as well as in pretreatment and follow-up comparison. Please note, that these SUD values reflect subjective reactions during the experimental condition, and that during treatment, SUDs are not reported. Mean values are described in Table 2 for all patients who started treatment (intent-to-treat), and in Table 3 for patients who completed postassessment as well as follow-up.

TABLE 2. Subjective Distress and Psychophysiological Measures

	Pre (*N* = 15)	Post (*N* = 12)	Follow-Up (*N* = 12)	Comparison Pre/Post		Comparison Pre/Follow-Up	
Measures	Mean (*SD*)	Mean (*SD*)	Mean (*SD*)	*t* (*df*)	Significance	*t* (*df*)	Significance
HR neutral (bpm)	79.0 (10.9)	74.5 (10.2)	75.4 (9.2)	0.44 (11)	*ns*	0.58 (11)	*ns*
HR trauma script (bpm)	89.9 (15.8)	78.6 (8.9)	80.5 (8.8)	2.2 (11)	$p = .053$	2.5 (11)	$p = .031$
HR difference (bpm)	10.9 (10.3)	4.2 (4.8)	5.1 (5.7)	3.3 (11)	$p = .007$	2.4 (11)	$p = .030$
RSA neutral [ln(ms)²]	5.0 (1.0)	5.3 (1.1)	5.9 (1.2)	– 1.7 (11)	$p = ns$	– 3.7 (11)	$p = .004$
RSA trauma script [ln(ms)²]	4.5 (1.4)	5.4 (1.4)	5.6 (1.2)	– 2.8 (11)	$p = .018$	– 2.8 (11)	$p = .018$
RSA difference [ln(ms)²]	– .49 (0.92)	0.12 (0.82)	– .31 (0.67)	2.0 (11)	$p = .071$	0.59 (11)	*ns*
SUD (0–10)	6.4 (2.0)	3.5 (1.9)	3.5 (2.2)	4.8 (11)	$p < .001$	4.2 (11)	$p = .002$

Note. SD = standard deviation; SUD = subjective units of distress during trauma script; HR = heart rate; RSA = respiratory sinus arrhythmia; HR difference = HR trauma script – HR neutral; RSA difference = RSA trauma script – RSA neutral. Statistical comparison: paired *t* test (two tailed).

TABLE 3. Subjective Distress and Psychophysiological Measures (Completers Only)

	Pre (*N* = 11)	Post (*N* = 11)	Follow-Up (*N* = 11)	Comparison Pre/Post		Comparison Pre/Follow-Up	
Measure	Mean (*SD*)	Mean (*SD*)	Mean (*SD*)	*t* (*df*)	Significance	*t* (*df*)	Significance
HR neutral (bpm)	74.5 (8.6)	74.4 (10.7)	74.1 (8.4)	0.07 (10)	*ns*	0.25 (10)	*ns*
HR trauma script (bpm)	86.6 (16.9)	78.9 (9.3)	79.6 (8.7)	1.8 (10)	$p = .110$	2.2 (10)	$p = .051$
HR difference (bpm)	12.1 (10.9)	4.5 (4.9)	5.5 (5.8)	2.9 (10)	$p = .015$	2.4 (10)	$p = .037$
RSA neutral [ln(ms)²]	5.0 (1.0)	5.4 (1.1)	5.8 (1.2)	1.6 (10)	$p = ns$	3.4 (10)	$p = .007$
RSA trauma script [ln(ms)²]	4.5 (1.3)	5.5 (1.4)	5.5 (1.3)	2.4 (10)	$p = .040$	2.6 (10)	$p = .027$
RSA difference [ln(ms)²]	– 0.52 (0.97)	0.06 (0.82)	– 0.32 (0.71)	1.6 (10)	$p = .143$	0.58 (10)	*ns*
SUD (0–10)	6.8 (2.10)	3.6 (2.0)	3.6 (2.3)	4.3 (10)	$p < .001$	4.0 (10)	$p = .003$

Note. SD = standard deviation; SUD = subjective units of distress during trauma script; HR = heart rate; RSA = respiratory sinus arrhythmia; HR difference = HR trauma script – HR neutral; RSA difference = RSA trauma script – RSA neutral. Statistical comparison: paired *t* test (two tailed).

Psychophysiological Measures

At all assessment points, HR increased significantly during trauma-script exposure when compared with the neutral condition (see Table 2). Pre- versus posttreatment comparison showed a significant reduction of trauma-script-induced HR reactions, which was also found in pretreatment versus follow-up comparison. RSA scores showed significant improvement—indicating higher parasympathetic tone—at posttreatment for the trauma-script condition and during follow-up for both the neutral condition and trauma script. There were no significant correlations between the reported changes in psychophysiological reactivity and treatment outcome as indicated by reduction of IES or PDS at posttreatment or follow-up.

A graphical display of the time course of HR immediately before and during listening to the individual trauma script confirmed the statistical finding of heightened psychophysiological reactivity at pretreatment assessment as compared to posttreatment and follow-up (see Figure 1).

Discussion

In our sample of patients with PTSD after adulthood single trauma, EMDR treatment was followed by a significant reduction of trauma-related symptoms, which was maintained in a 6-month follow-up. Psychophysiological arousal during presentation of an individualized trauma script, one of the main characteristics of PTSD, was significantly reduced in pre- versus posttreatment comparison as well as in pretreatment follow-up comparison. Following treatment with EMDR, HR acceleration to trauma script was significantly reduced at posttreatment and at follow-up. Subsequently, patients reported a significant decrease of their subjective distress during trauma-script presentation. RSA indicating parasympathetic tone increased significantly over the course of treatment both during the neutral condition script and during trauma script.

Observed effect sizes in terms of symptom reduction (IES pre- versus posttreatment and pretreatment versus follow-up: 1.75 and 1.69, respectively) were relatively high also when compared with results of other PTSD treatment studies (Van Etten & Taylor,

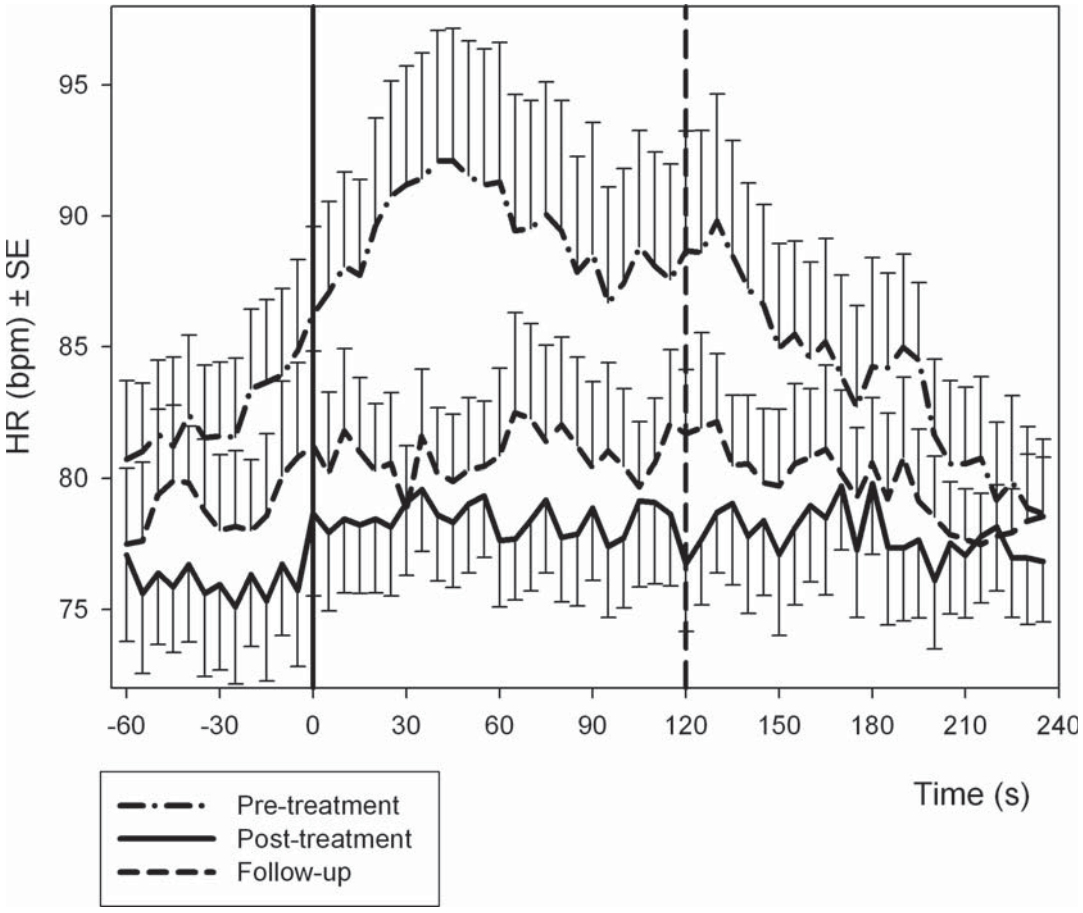

FIGURE 1. Time course of heart rate (± *SE*) immediately before and during trauma script (0 to 120 s).

1998). The largest treatment effects were found for intrusive symptoms. Particular efficacy of the EMDR treatment in improving intrusive symptoms has also been previously reported (Lee, Gavriel, Drummond, Richards, & Greenwald, 2002).

The validity of our results has certain underlying methodological restrictions. First, our study had a relatively small sample size of only 16 patients with PTSD, which was further reduced by 2 dropouts and partially missing data from 3 participants. Second, because no control group was assessed, effects of repeated measurement of psychophysiological reactions could not be controlled. Although the question of whether repeated presentation of an individual trauma script would per se be associated with psychophysiological habituation has not been sufficiently examined empirically, the reproduction of cardiovascular reactions to stressors seems to be generally high, as studies in patients with panic disorders and generalized anxiety disorders demonstrate (Allen, Sherwood, Obrist, Crowell, & Grange, 1987; Eckman & Shean, 1997). Third, since our equipment did not allow the recording of breathing rates, RSA analyses did not control for possible respiratory influences as recommended in recent guidelines (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). The generalizability of this study is further limited because no reassessment with diagnostic interview at post-treatment or follow-up was included. However, reduction of PTSD symptom levels below norms as measured by standardized questionnaires indicates that EMDR treatment in our study was an effective intervention.

Even after consideration of the above-discussed limitations, especially concerning the lack of an appropriate control group, the results of our study demonstrate the feasibility of measuring psychophysiological stress reactions during the course of treatment sessions. As our results indicate, a reduction of trauma symptoms and of subjective distress during a traumatic reminder might be accompanied by a normalization of psychophysiological reactivity during trauma script. Memories that were previously trauma triggering seem—at least in part—to have lost their pathological impact, according to our patients' reports. In fact, some patients were surprised when the audiotaped presentation of their traumatic memory failed to evoke any distressing reaction. We did not find a significant association between changes in psychophysiological reactivity and treatment outcome. However, due to the small sample size, the statistical power of our study is probably insufficient to reliably answer this question.

This is the first study showing a relation between RSA and EMDR treatment. The results of our study have to be considered as preliminary and should be interpreted with caution. However, they indicate that successful resolution of traumatic memory may be associated with an increase in parasympathetic tone, not only during confrontation with a reminder of the traumatic memory but also during a baseline control condition. The observed increase of RSA both during the control condition and during trauma script indicate higher levels of parasympathetic tone after therapy, which may be associated with better capacities to regulate psychophysiological stress reactions.

The adaptive information processing model (Shapiro, 2002), predicts that successful processing of implicit traumatic memory, with EMDR treatment, will result in integration of the memory's somatic component, resulting in a reduction of physiological reactivity. Some support for this hypothesis was found in our study, with treatment leading to enhanced psychophysiological regulation capacities—during confrontation with a trigger of the traumatic memory—as well as to a reduction of psychobiological markers of chronic stress. Furthermore, these findings fit with the idea that trauma integration might lead to a restoration of inhibitory circuits responsible for regulation of limbic-generated arousal and anxiety (Thayer & Brosschot, 2005).

Although this study relied on trauma treatment with EMDR, we do not expect the resulting psychophysiological treatment effects to be specifically related to this therapy. We anticipate that equivalent outcomes would result from any successful treatment of PTSD that produced traumatic memory integration and an extinction of the traumatic fear structure and associated dysfunctional cognitions (Foa & Kozak, 1986). Our results, therefore, supplement the findings from Nishith et al. (2003) reporting significant decreased sympathetic predominance during REM sleep in cognitive behavioral therapy treatment responders. We are convinced that the assessment of parameters of autonomic regulation during the course of psychotherapeutic treatment provides a promising field of research at the interface between body and psyche, and that further research will offer new insights into possible neurobiological mechanisms underlying trauma-related disorders.

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