Journal of Traumatic Stress, Vol. 19, No. 5, October 2006, pp. 721–734 (© 2006)

Reduced Heart Rate Responding to Trauma Reliving in Trauma Survivors With PTSD: Correlates and Consequences*

Sarah L. Halligan
School of Psychology and Clinical Language Sciences, University of Reading, Reading, United Kingdom

Tanja Michael and Frank H. Wilhelm
Department of Clinical Psychology and Psychotherapy, Institute for Psychology, University of Basel, Basel, Switzerland

David M. Clark and Anke Ehlers
Department of Psychology, Institute of Psychiatry, King's College, London, United Kingdom

The authors investigated whether heart rate (HR) responses to voluntary recall of trauma memories (a) are related to posttraumatic stress disorder (PTSD), and (b) predict recovery 6 months later. Sixty-two assault survivors completed a recall task modeled on imaginal reliving in the initial weeks postassault. Possible cognitive modulators of HR responsivity were assessed; dissociation, rumination, trauma memory disorganization. Individuals with PTSD showed a reduced HR response to reliving compared to those without PTSD, but reported greater distress. Notably, higher HR response but not self-reported distress during reliving predicted greater symptom reduction at follow-up in participants with PTSD. Engagement in rumination was the only cognitive factor that predicted lower HR response. The data are in contrast to studies using trauma reminders to trigger memories, which have found greater physiological reactivity in PTSD. The authors' observations are consistent with models of PTSD that highlight differences between cued or stimulus-driven retrieval and intentional trauma recall, and with E. B. Foa and M. J. Kozak's (1986) hypothesis that full activation of trauma memories facilitates emotional processing.

Enhanced physiological reactivity in response to trauma cues appears to be a robust correlate of posttraumatic stress disorder (PTSD; Blanchard & Buckley, 1999; Orr & Roth, 2000); trauma survivors with PTSD have consistently shown higher heart rate (HR), blood pressure, skin conductance and facial electromyogram responses on exposure to trauma reminders than trauma survivors without PTSD. Such observations have replicated across trauma populations distinguished by type of exposure and chronicity of symptoms, and some authors have suggested that the specificity of enhanced responding in PTSD warrants the implementation of psychophysiological tests as a diagnostic aid (Blanchard, Kolb, & Prins, 1991; Orr & Roth, 2000).

To date, the focus of psychophysiological studies has been on involuntary access to trauma material. Studies have primarily investigated cued recall, with memories

*This article was edited by the journal's previous editor, Dean G. Kilpatrick.

This research was funded by Wellcome Prize Studentships awarded to Sarah Halligan and Tanja Michael. David Clark and Anke Ehlers were supported by Wellcome Principal Research Fellowships. We thank the Victim Support Centres for their invaluable collaboration in this research.

Correspondence concerning this article should be addressed to: Sarah L. Halligan, School of Psychology, University of Reading, 3 Earley Gate, Whiteknights, Reading RG6 6AL, United Kingdom. E-mail: s.l.halligan@reading.ac.uk.

 DOI: 10.1002/jts.20167

being automatically triggered by trauma reminders such as sounds or pictures (Blanchard et al., 1996; Blanchard, Kolb, Pallmeyer, & Gerardi, 1982; Shalev, Peri, Gelpin, Orr, & Pitman, 1997), or by highly provocative, idiosyncratic trauma-scripts, which describe the most arousing aspects of the trauma and response elements such as heart pounding or sweating (Pitman, Orr, Forgue, de Jong, & Claiborn, 1987; Shalev, Orr, & Pitman, 1993). In contrast, physiological responses during intentional recall of the trauma (i.e., self-generated, effortful retrieval) have not been systematically studied.

A closer investigation of physiological parameters during intentional trauma recall is of theoretical interest because the literature suggests a paradox of easy cue-driven retrieval of trauma memories by matching cues in PTSD on the one hand and impaired intentional access to trauma memories on the other (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000; Foa & Riggs, 1993). Numerous processes have been identified that may limit both the extent and the quality of intentional access. First, there is some empirical evidence for deficits in intentional trauma memory retrieval in PTSD (e.g., Foa, Molnar, & Cashman, 1995; Halligan, Michael, Clark, & Ehlers, 2003; for a review, see Ehlers, Hackmann, & Michael, 2004). Disorganized and incomplete intentional retrieval of trauma memories in individuals with PTSD may limit physiological responding due to a failure to access key elements of the event (Foa, Molnar, et al., 1995). Second, dissociation is common both peritraumatically and during subsequent trauma recall. As dissociation represents a separation in cognitive and emotional processes, it is likely to result in an uncoupling of physiological responses from psychological distress. Prior research has found peritraumatic dissociation to be associated with decreased psychophysiological activation in response to discussing the trauma (Griffin, Resick, & Mechanic, 1997). Finally, ruminative processes are likely to be relevant. Some studies have found that patients with generalized anxiety disorder, a condition characterized by excessive worry, show sympathetic inhibition rather than enhancement in response to stress (Hoehn-Saric, McLeod, & Zimmerli, 1989). Furthermore, worry preceding imaginal exposure to phobic events has been found to suppress cardiovascular responses to these events without reducing the extent to which they are psychologically experienced as anxiety relevant (Borkovec, Ray, & Stöber, 1998). Rumination is comparable to worry as a process, but is focused on past rather than future events (Papageorgiou & Wells, 2004); trauma-related rumination is marked in individuals with PTSD, and has been shown to predict chronic PTSD in prospective longitudinal studies (e.g., Ehlers, Mayou, & Bryant, 1998; Murray, Ehlers, & Mayou, 2002).

The degree of physiological activation during intentional trauma recall is of potential relevance to the treatment of PTSD. Foa and colleagues (Foa & Kozak, 1986; Foa, Steketee, & Rothbaum, 1989) have proposed that complete activation of the trauma fear memory network is necessary for emotional processing to occur. High psychophysiological responding is seen as an indicator of such therapeutic memory activation (Foa & Kozak, 1986; Foa, Riggs, Massie, & Yarczower, 1995; Pitman et al., 1996). Imaginal trauma exposure (or "reliving") is a treatment technique designed to facilitate emotional processing of the trauma memory (Foa & Rothbaum, 1998), requiring the patient to intentionally retrieve a detailed account of the trauma. Tentative support for the utility of psychophysiological responding as an indicator of therapeutic activation has been provided by Pitman and colleagues (Pitman et al., 1996) who examined HR responses during exposure therapy in a sample of chronic PTSD patients. Higher initial HR response during initial exposure sessions predicted a better treatment outcome in terms of a reduction in the number of intrusive memories, but was not related to other symptom measures. The small sample size ($n = 20$) and modest symptom reduction (13% overall) reported in this study mean that replication is desirable.

The current study examines psychophysiological responding during intentional recall of the trauma in victims of assault, in contrast to the prior research focus on cue-driven, triggered recall. Participants were recruited from the community shortly following the trauma and were reassessed 6 months later. Heart rate response during intentional trauma memory recall was examined in relation to the presence or absence of PTSD at first assessment and in the prediction symptoms at follow-up. Due to a lack

of information in the literature, we could not formulate a directional hypothesis with respect to psychophysiological responding in participants with PTSD versus those without PTSD. Although the literature on cue-driven retrieval would suggest a greater HR response in participants with PTSD, intentional recall may be characterized by a failure to achieve full access to emotionally arousing components of the memory trace, with concomitant lower HR. As such, conducting an examination of heart rate response during intentional trauma recall was the primary aim of this study. We further hypothesized that:

1. Assault survivors with PTSD will report greater emotional distress during intentional recall of the trauma than assault survivors without PTSD.
2. High levels of dissociation, trauma memory disorganization, and rumination will serve as barriers to emotional access and will thus be related to reduced psychophysiological responding during intentional trauma recall.
3. Following models that emphasize the need for emotional processing of the trauma, higher heart rate response to intentional trauma recall in the aftermath of the trauma will predict better outcomes at 6-month follow-up for individuals with PTSD.

METHOD

Participants

Participants were drawn from a sample of 73 victims (32 women and 41 men) of physical and sexual assault; the current study reports on 61 participants for whom HR assessments were available. Missing data were due to physical or medical exclusions ($n = 4$), recording problems ($n = 5$), and refusal to complete this aspect of the research ($n = 3$). Details of recruitment, sample characteristics, and the outcomes of cognitive assessments have been published elsewhere (Halligan et al., 2003; Michael, Ehlers, & Halligan, 2005; Michael, Ehlers, Halligan, & Clark, 2005). In brief, participants were recruited via flyers sent out by Victim Support Centres who contact all victims of crime who report the incident to the police in the UK. Respondents were sent a flyer (which contained a brief description of all study procedures) if they had been assaulted less than 3 months previously and were aged 18–75 years. They returned their contact details to us if they wished to participate. Participants were then accepted into the study subject to a telephone-screening procedure examining the following exclusion criteria: assault occurring in the context of ongoing domestic violence, a history of psychosis, or the presence of a current substance abuse problem. The UK Multicentre Research Ethics Committee approved the study, and all participants gave written informed consent prior to participation.

Self-Report Measures

Objective assault characteristics. A semistructured interview provided a comprehensive assessment of assault characteristics (Halligan et al., 2003). Objective assault severity was computed as a composite score with a possible range of 0 to 12, derived from the following indices: number of assailants, assault duration, use of verbal threat, injury severity, and weapon usage. Scores in the current sample ranged from 2 (for example, the victim of a road rage incident who was hit through her car window) to 11 (e.g., the victim of an extended physical assault by a large group of individuals armed with metal piping, resulting in multiple injuries). As the semistructured interview was completed before the assault recall task, care was taken to ensure that it did not elicit a full descriptive account of the incident. Rather, participants responded to a series of structured-response questions that asked for the specific, limited information about the characteristics of the assault detailed above.

Posttraumatic stress disorder. The Posttraumatic Stress Diagnostic Scale (PDS; Foa, 1995; Foa, Cashman, Jaycox, & Perry, 1997) is a standardized self-report measure of PTSD diagnostic criteria, including trauma characteristics, symptom severity and duration, and resultant degree of life interference. The PDS was administered with the assistance of the interviewer, who clarified items as required.

The PDS has good levels of reliability and validity and shows good diagnostic agreement with the Structured Clinical Interview for DSM-IV (Foa et al., 1997). To be given a positive diagnosis individuals needed to score at least 15 on the PDS symptom scale in addition to meeting *DSM-IV* criteria, following research indicating that the inclusion of this severity cutoff improves agreement between the PDS and diagnoses based on diagnostic interview (Foa, 1998). For participants in the current study who completed the first assessment less than 1 month following the assault a diagnosis of PTSD was assigned where all criteria were met except for *DSM-IV* Criterion E (1-month duration of symptoms). Importantly, in all cases where a positive PTSD diagnosis was initially assigned in the absence of Criterion E, PTSD was also found to be present 3 months later.

Anxiety and depression. General symptoms of depression and anxiety were measured as common responses to trauma, using the Beck Depression Inventory (BDI; Beck, Ward, Mendleson, Mock, & Erbaugh, 1961) and the State Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983), respectively. Both the BDI and the STAI are widely used scales with demonstrated reliability and validity (Beck, Steer, & Garbin, 1988; Groth-Marnot, 1990).

Persistent dissociation. The Trait Dissociation Questionnaire (TDQ; Murray et al., 2002), a reliable and valid self-report measure of dissociative symptoms, was used to assess ongoing dissociative tendencies following the assault.

Peritraumatic dissociation. The State Dissociation Questionnaire (SDQ; Murray et al., 2002) was completed by participants with respect to their experiences during the assault. This nine-item scale measuring peritraumatic dissociative experiences such as derealization, depersonalization, detachment, altered time sense, and emotional numbing shows good reliability and validity (Halligan, Clark, & Ehlers, 2002; Halligan et al., 2003; Murray et al., 2002), and correlates highly with the Peritraumatic Dissociation Scale (Marmar, Weiss, & Metzler, 1997; Rosario, Williams, & Ehlers, 2002).

Memory disorganization. Transcribed trauma narratives were analyzed phrase by phrase to give an index of disorganization; this index, based on an original analysis by Foa and colleagues (Foa, DiSavino, & Turk, 1995; Foa, Molnar, et al., 1995), has been described in detail in prior research and has previously been found to correlate with PTSD symptoms (Halligan et al., 2003). Participants also completed a questionnaire comprised of five items, which examined the extent to which they themselves believed their memory to be disorganized or incomplete (see Halligan et al., 2003). Scores on this questionnaire correlated ($r = .48$, $n = 178$) with our measure of narrative disorganization. Finally, participants rated on a 5-point scale the extent to which they deliberately left information out while recalling the assault.

Rumination. Participants completed a detailed interview relating to their current ruminative behaviors (Michael, Halligan, Ehlers, & Clark, in press); the number of hours per week spent dwelling on the assault was used to index ongoing ruminative activity.

Baseline and Assault Recall Task

Task description. Participants were sitting in a comfortable chair during the entire procedure. The experimenter was seated opposite at a distance of approximately 1.5 meters. For the baseline assessment, participants were asked to sit quietly with eyes open for 5 minutes. The experimenter kept their head lowered and avoided eye contact. After 5 minutes, participants were asked to close their eyes and imagine the assault vividly while giving a detailed verbal narrative. This task was modeled on imaginal reliving (Foa & Rothbaum, 1998). The following instructions were provided in both verbal and written form.

> With your eyes closed, I would like you to take yourself back to the time of the assault and remember it as vividly as you can. Begin just before the assault took

> place and go through everything that happened from start to finish. Try to make your account as clear as possible, describing things in the order in which you remember them happening. Include details about the surroundings, your activities, the thoughts that were going through your mind, and the way that you were feeling at each point in time.

Participants were not interrupted during the recall task. No participant failed to talk spontaneously for less than 2 minutes and all participants completed their description of the incident without prompting.

Heart rate responses. Heart rate was recorded continuously during the tasks using a Polar Vantage NV Heart Rate Monitor (Polar Electro Ltd., Kempele, Finland), consisting of a chest band and wrist receiver. The monitor was set to record HR second by second. The device has been shown to reliably assess interbeat intervals during rest as well as activity (Karvenon, Chwalbinska-Moneta, & Saynajakangas, 1984; Seaward, Sleamaker, McAuliffe, & Clapp, 1990); 10-second interval HR measurements obtained via Polar HR monitors have been found to correlate r = .99 with those obtained via electrocardiogram (Laukkanen & Virtanen, 1998; Wajciechowski, Gayle, Andrews, & Dintiman, 1991). The beginning and endpoints of each task were defined using a marker button on the receiver (which was operated by the experimenter). Recorded data was subsequently examined using Polar Precision Performance Software Version 3.0 (Polar Electro Ltd., Kempele, Finland). Artifacts in the data due to misdetected R-waves in the electrocardiogram were easily discernible as outliers from the average HR curve, and were deleted and interpolated prior to averaging using the software error correction facility. Heart rates for the last 2 minutes of the baseline task and the first 2 minutes of the assault recall task were averaged to retrieve stable estimates of HR reactivity.[1] For the majority of participants, less than 5% of HR data for these segments were interpolated due to artifacts. For two cases, more than 5% interpolation was noted (up to 10%); however, excluding these cases from analyses did not alter the findings.

Reported emotional responses. Participants rated their level of relaxation versus activation on an anchored 0 (*extremely relaxed*) to 100 (*extremely activated*) point visual-analogue scale immediately preceding the baseline assessment. Following recall, participants rated how activated or relaxed they felt during the recall task using the same scale. Participants also rated their distress and their feelings of emotional numbness during the recall task on 0–100 point visual-analogue scales (from 0 = *not at all distressed/numb* to 100 = *extremely distressed/numb*).

Procedure

Participants were initially interviewed as soon after making contact as it was possible to arrange, and in all cases within 3 months of the assault (mean elapsed time of 7 weeks). Prior to being interviewed, participants were sent a full description of all study procedures, along with self-report measures of symptoms of depression (BDI), anxiety (STAI), and dissociation (TDQ) to complete. Participants were assessed by a single researcher (either of the two senior authors, SLH or TM) in their own homes or at their local Victim Support Centre, as preferred, and sessions lasted approximately 2 hours in total. Participants began by completing a semi structured interview examining (a) sociodemographic factors, (b) objective assault severity, and (c) current medical status. This interview was placed at the start of the session to facilitate participant engagement, and to ensure a delay of approximately 30 minutes between the start of the session and the beginning of heart rate recording. Subsequently, baseline heart rate and self-reported emotional state were obtained, and the assault recall task and associated self-report measures of emotional responding were completed. Following this, participants went on to complete further assessments that have been detailed elsewhere (Halligan et al., 2003; Michael, Ehlers,

[1] In every case, HR was higher for the first 2-minutes of the recall task than for the entire task. Furthermore, when the data were analysed using HR averaged over the whole task, the results were the same as for analyses of the initial 2 mins. Only the latter findings are reported due to the problems associated with comparing HRs averaged across assessments of different duration.

& Halligan, 2005; Michael, Ehlers, Halligan, et al., 2005). After 6 months participants were reinterviewed, and their PTSD symptoms reassessed (PDS).

Statistical Analyses

Primary outcome measures were normally distributed and were examined using parametric tests. One-way repeated measures analysis of variance (2×2 ANOVA) with diagnostic group as the between subject factor (PTSD vs. non-PTSD) and time (baseline vs. trauma recall) as the within subject factor examined group differences in response to the trauma recall task. Correlational analyses examined: (a) associations between self-reported distress and HR response to recalling the assault; (b) the prediction of PTSD symptoms at 6-month follow-up by initial HR and self-reported distress; and (c) possible modulation of HR response by cognitive factors (peritraumatic and ongoing dissociation, memory disorganization, rumination). Some subsidiary analyses involved variables with nonnormal distributions (e.g., duration of the assault recall task); appropriate nonparametric tests were employed (Mann–Whitney U tests, chi square tests for categorical variables).

RESULTS

Description of the Participant Groups

The sample was comprised of 28 female and 33 male victims of assault, aged 18 to 71 years ($M = 39.0$ years, $SD = 14.4$). The majority of participants had experienced physical ($n = 59$) rather than sexual assault ($n = 2$).[2] Twenty-five participants (41%) met criteria for PTSD at first assessment (PTSD group) and 38 (59%) did not (non-PTSD group). Demographic characteristics, assault-related measures, and symptom scores are reported in Table 1. As can be seen from the table, the groups were comparable in terms of gender[3] and age, time elapsed since the assault, and assault severity. The PTSD group scored higher on all measures of psychological symptoms; depression, anxiety, and dissociation, as well as symptoms of PTSD. Fifty-eight of the original 61 participants completed the 6-month follow-up; the small number of dropouts precluded meaningful analyses of their characteristics relative to the rest of the sample.

Duration of the Narrative

All participants followed the instructions well and produced continuous assault recall narratives of at least 2 minutes duration. However, participants exhibited significant variability in the time taken to complete the narrative ($M = 8.5$, $SD = 7.4$ minutes, range = 2–40 minutes). The PTSD and non-PTSD groups did not differ significantly in the duration of their narratives, (Mann–Whitney U test, $Z = -1.28$, *ns*). In addition, there was no significant difference between the groups in terms of the number of narrative phrases produced during the recall task, Mann–Whitney U test, $Z = -0.30$, *ns*, or in the rate of phrase production during the task Mann–Whitney U test $Z = -0.66$, *ns*.

Self-Reported Responses to Recalling the Assault

Repeated-measures ANOVA examined self-reported scores on the relaxation-activation scale during the baseline period versus the assault recall task by PTSD group. The results indicated a significant main effect of time, Hotelling's Trace $F(1, 58) = 76.2$, $p < .001$, with reported activation being higher during the recall task versus the baseline period. There was also a significant time (baseline vs. assault recall) by diagnosis (PTSD vs. non-PTSD) interaction, Hotelling's Trace $F(1, 58) = 4.90$, $p = .05$, illustrated in

[2] We had no *a priori* reason to believe that PTSD to a sexual assault should be intrinsically different to that resulting from a physical assault. Furthermore, when analyses were carried out with versus without the two victims of sexual assault, the findings were the same (results not presented).

[3] Although the PTSD and no-PTSD groups had similar gender distributions, as gender is known to be associated with differences in heart rate and subjective experiences of distress all reported analyses were also repeated controlling for gender. In no instance did gender prove to be a significant covariate, and the inclusion of gender did not alter the reported findings (results not presented).

Table 1. Sample Characteristics

Characteristic	PTSD ($n = 25$)		Non-PTSD ($n = 38$)		Analysis
	M	*SD*	*M*	*SD*	
Demographics					
Proportion female; (*n, %*)	12	48.0%	16	44.4%	$\chi^2(1) = 0.08$
Age (years)	39.9	13.3	38.4	15.2	$t(59) = 0.35$
Assault					
Time since (weeks)[a]	8.5	3.0	7.3	2.8	$Z = 1.82$
Assault severity score (0–12)[a]	5.8	2.1	5.2	2.0	$Z = 1.41$
Initial psychiatric symptoms					
PDS (PTSD)	30.4	7.5	10.1	6.3	$t(59) = 11.5^{***}$
BDI (Depression)[b]	18.1	8.3	5.8	5.0	$t(30.8) = 6.27^{***}$
STAI-S (State anxiety)	41.0	8.4	32.9	10.7	$t(59) = 3.17^{**}$
STAI-T (Trait anxiety)	56.7	10.2	38.8	11.0	$t(59) = 6.17^{***}$
Follow-up symptoms					
PDS (PTSD)	20.8	7.9	5.1	6.9	$t(56) = 8.04^{***}$
BDI (Depression)[b]	12.2	6.4	3.1	3.9	$t(35.2) = 6.20^{***}$
Cognitive factors					
TDQ (Trait dissociation)[b]	57.4	27.5	37.5	17.2	$t(32.2) = 3.01^{**}$
SDQ (Peritraumatic dissociation; 0–4)[b]	1.76	1.20	0.93	0.59	$t(32.3) = 3.21^{**}$
Rumination score (0–5)[a]	2.24	1.45	0.67	1.01	$Z = 4.40^{***}$
Assault memory fragmentation[b]	0.90	1.78	−0.58	0.83	$t(30.0) = 4.29^{**}$

Note. PDS = Posttraumatic Stress Diagnostic Scale, BDI = Beck Depression Inventory; STAI-S/T = State and Trait Anxiety Inventory, state/trait version; TDQ = Trait Dissociation Questionnaire; SDQ = State Dissociation Questionnaire.

[a]Mann–Whitney U tests were reported for nonparametric data; although this test uses ranked scores, actual means and standard deviations are presented for the reader's information.

[b] *t* test corrected for unequal variances.

$^{**}p < 0.01$. $^{***}p < .001$.

Figure 1, Panel A. In accordance with predictions, follow-up *t* tests indicated that although the two groups were equivalent during baseline, $t(58) < 1$, the PTSD participants reported more activation than the non-PTSD participants in response to recalling the assault, $t(59) = 2.56$, $p < .05$.

In addition, we examined levels of self-reported distress and emotional numbing in response to recalling the assault. Participants in the PTSD group reported significantly more distress when recalling the assault than did participants in the non-PTSD group [PTSD $M = 72.0$, $SD = 21.7$; non-PTSD $M = 46.3$, $SD = 23.5$; $t(59) = 4.33$, $p < 0.001$]. However, a Mann–Whitney U test indicated no significant group difference in the amount of numbing reported during the recall task (PTSD $M = 32.6$, $SD = 35.3$; non-PTSD $M = 19.3$, $SD = 27.8$; $Z = 1.49$, *ns*).

Heart Rate Response to Recalling the Assault

Repeated-measures ANOVA examined mean HR during the baseline period versus the assault recall task by the PTSD group. The results indicated a significant main effect of time, Hotelling's Trace $F(1, 59) = 49.5$, $p < 0.001$, with HR being higher during the recall task versus the baseline period. There was also a significant time (baseline vs. assault recall) by diagnosis (PTSD vs. non-PTSD) interaction, Hotelling's Trace $F(1, 59) = 5.85$, $p < .05$, illustrated in Figure 1, Panel B. Follow-up *t* tests indicated no significant differences between the PTSD and non-PTSD groups in either baseline HR, $t(59) = 1.20$, *ns*, or HR during the recall task, $t(59) = 0.62$, *ns*. However, the HR change score was significantly lower in the PTSD group, $t(59) = 2.65$, $p = .01$.

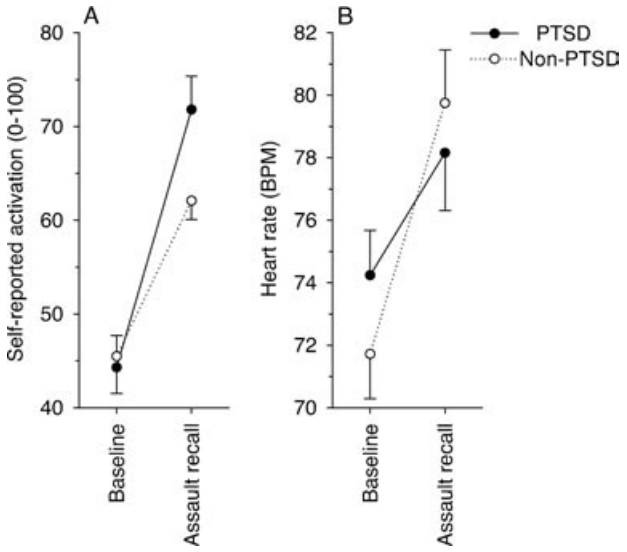

Figure 1. Self-reported (Panel A) and psychophysiological (Panel B) responding to trauma recall in assault victims with and without posttraumatic stress disorder. PTSD = Posttraumatic stress disorder; BPM = beats per minute.

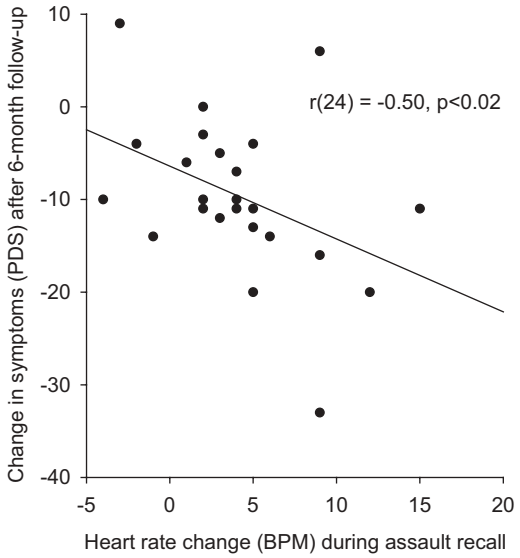

Figure 2. Association between heart rate responding to trauma recall and change in self-reported posttraumatic stress disorder symptoms over the subsequent 6 months in participants with posttraumatic stress disorder. PDS = Post-Traumatic Stress Diagnostic Scale; BPM = beats per minute.

Prediction of Symptom Change

Correlational analyses examined the prediction of symptom change (as assessed by the PDS) through the 6-month follow-up period by changes in both self-reported reactions and HR in response to recalling the assault. Analyses were carried out separately for the PTSD and non-PTSD groups due to group differences in the range of HR and symptom change. The change in self-reported activation during the recall task did not predict change in symptoms from first assessment to 6-month follow-up in either the non-PTSD ($r = -.04$, $n = 33$, *ns*) or PTSD ($r = -.04$, $n = 24$, *ns*) groups. Change in HR during the assault recall task similarly did not predict symptom change in the non-PTSD group ($r = -.05$, $n = 34$, *ns*). However, in the PTSD group there was a relatively strong, negative association between HR change in response to recalling the assault and symptom change at 6 months ($r = -.50$, $n = 24$, $p < .05$). This indicates that a greater physiological response to recalling the assault predicted a greater reduction in symptoms over the 6-month follow-up period (see Figure 2).

Associations Between Self-Report and Physiological Responses

Correlational analyses examined the relationship between HR and self-report indices of activation in response to the assault recall task, using change scores from baseline in each instance. There was no correlation between the change in self-reported activation and HR response to recalling the assault ($r = -.09$, $n = 60$, *ns*). Neither did actual HR during the assault recall task or HR change from baseline correlate with either self-reported distress or numbing while recalling the assault (rs range from $-.14$ to .07, $n = 61$, all *ns*). Correlations between HR response and dissociation (peritraumatic and ongoing), rumination, and memory disorganization (both narrative scored and self-reported) were also calculated. Of these cognitive variables, only rumination was significantly associated with HR response to the assault recall task ($r = -0.26$, $n = 61$, $p = 0.05$). Finally, we verified that participant HRs did not reflect a deliberate lack of engagement in our recall task, through examining the extent to which they endorsed deliberately omitting

aspects of the assault. No participant scored greater than 3 out of a possible 5 on this questionnaire item, and there was no correlation between reported omission and HR response (Spearman's $r = -.12$, $n = 59$, *ns*).

DISCUSSION

Assault survivors with PTSD showed smaller HR responses to intentional recall of their traumatic experience compared to those without PTSD. Furthermore, a discrepancy was observed between HR changes and self-reported levels of distress in response to recalling the trauma; trauma survivors with PTSD self-reported more distress than those without, despite the smaller physiological response. Indeed, there were no associations between HR responses and participant self-reports of subjective distress. Finally, for participants with PTSD higher physiological activation during assault recall, but not higher self-reported activation, predicted greater reduction in symptoms over the subsequent 6 months.

Our findings using intentional recall of the trauma modeled on the imaginal reliving treatment procedure contrast those obtained with prior research on physiological responses to trauma reminders. In studies that have used cue-driven retrieval of trauma memories via a range of trauma reminders (sounds, pictures, or imagery scripts), survivors with PTSD have shown greater HR responses than those without PTSD, whereas we found intentional trauma recall to provoke a lower heart rate response in those with PTSD versus without PTSD. Furthermore, previous studies using trauma reminders found that higher initial physiological responding predicted the persistence of PTSD symptoms at follow-up (Blanchard et al., 1996; Elsesser, Sartory, & Tackenberg, 2004), whereas our study using intentional, self-generated trauma recall found greater improvement in PTSD sufferers who showed high initial HR responses.

The present findings may help to dispel the tension between psychophysiological studies of responding to trauma cues, where elevated responding indicates poor adjustment, and theoretical accounts of exposure-based treatments, which hold that strong psychophysiological responding during reliving indicates more complete processing and is therefore adaptive. The need to differentiate psychophysiological responding to cue-driven versus intentional trauma recall is in line with recent theoretical models of PTSD (Brewin et al., 1996; Ehlers & Clark, 2000; Foa & Riggs, 1993; Foa et al., 1989). These models highlight the contrast between the easy triggering of intrusive trauma memories and corresponding physiological arousal by matching sensory cues, and the difficulties observed in intentionally accessing a complete, organized, and emotionally arousing memory of the trauma. Whereas studies using trauma reminders to provoke trauma memories are likely to tap into the first process, thus finding high physiological responses to reminders in PTSD, the present study appears to have tapped into the latter process by using self-generated, intentional retrieval of the trauma memory, resulting in lower physiological responsiveness. Participants with PTSD may have failed to achieve full activation of emotional components of their memory during the recall task, despite reporting significant distress. Prior research supports the proposal that limited emotional access may result in reduced physiological responding in individuals with PTSD. Griffin et al. (1997) examined psychophysiological responding to discussing the trauma in the immediate aftermath and found that individuals reporting high peritraumatic dissociation showed a decrease rather than an increase in physiological arousal. Conversely, externally expressed intensity of trauma-related emotion during reliving, as evidenced by facially expressed fear, has been found to be a positive predictor of exposure therapy efficacy (Foa, Riggs, et al., 1995).

We examined several potential cognitive contributors to the lower HR reactivity observed in PTSD participants; peritraumatic and concurrent dissociative symptoms, memory disorganization, and ruminative behavior following the trauma. Only high rumination was significantly related to lower HR response on recalling the trauma. This finding resembles those in generalized anxiety disorder, where worry has been found to alter physiological responding to emotional stimuli without altering their psychological significance, and may represent a form of avoidant coping (Borkovec & Roemer, 1995). Borkovec has argued that worry focuses processing on the verbal channel, and particularly on abstract concepts,

which has the effect of reducing the vividness of associated imagery and hence physiological responding (Borkovec et al., 1998). We have found abstract thoughts (particularly attempts to mentally undo the trauma) to be a primary component of trauma-related rumination (Michael, Halligan, et al., in press). Thus, individuals who spend large amounts of time dwelling on the assault may access trauma-related material in a less-activating manner, as has been reported with respect to worry. As rumination is an intentional cognitive process, this is pertinent to aspects of intentional trauma recall versus the cue-driven retrieval that characterizes intrusive memories. However, in light of the modest size of the correlation we observed, and the multiple correlations carried out between self-report measures and heart rate responding, the significance of this finding should not be overstated.

Dissociation and memory disorganization were not found to be associated with physiological responding to trauma recall. With respect to dissociation, our findings are at odds with those of Griffin et al. (1997), who found peritraumatic dissociation to be associated with lower heart rate responding during a trauma discussion carried out within 2 weeks posttrauma. We measured peritraumatic and ongoing symptoms of dissociation, as assumed determinants of dissociation during assault recall; this assumption may have been erroneous in our sample, where the elapsed time since the trauma was significantly longer than that reported by Griffin et al. (7 weeks on average). The failure to directly measure dissociation during trauma recall limits our ability to address this issue; although the participants' ratings of numbing also failed to correlate significantly with HR response, the extent to which emotional numbing can be equated with the broader construct of dissociation is limited (Holmes et al., 2005).

The absence of the predicted association between memory disorganization and low heart rate responding is surprising given that narrative disorganization predicted PTSD symptoms in the current sample (Halligan et al., 2003). This lack of association may reflect a general problem in assessing deficits in trauma memory recall comprehensively (see Ehlers et al., 2004 for a review). However, the failure to find an association may also be explained by further consideration of neurobiological systems implicated in trauma memories. Organizational aspects of memory, particularly the integration of sensory components and contextual setting, appear to rely on hippocampal functioning, while the fearful emotional tone is dependent on activation of the amygdala (Cahill, Babinsky, Markowitsch, & McGaugh, 1995; LeDoux, 1993, 2000; McClelland, McNaughton, & O'Reilly, 1995). It is proposed that the high levels of fear experienced during trauma heighten amygdaloid functioning and reduce hippocampal activity simultaneously, resulting in poorly organized but highly emotional memories (LeDoux, 1996; Siegel, 1996). As such, a possible confound exists in that memory disorganization may be both an indicator of highly emotionally charged memories, and therefore associated with high physiological arousal when the trauma memory is triggered (usually by external cues), but also a factor that limits memory access, leading to our hypothesis of an association with reduced physiological arousal in intentional retrieval.

The processes underlying the current observations require further investigation. In addition to possible cognitive influences on HR response, the broader biological response to recall is likely to be significant. Researchers have characterized response to stressful or traumatic events in terms of activation versus withdrawal, with the latter encompassing dissociative states (Bremner, Vermetten, Southwick, Krystal, & Charney, 1998; Kalin & Shelton, 1998; Perry, Pollard, Blackley, Baker, & Vigilante, 1995; Schore, 2002). Differences in psychobiology have been linked to these alternative states. Activated responding is an excitatory state associated with elevated secretion of noradrenaline and adrenaline; withdrawal has been linked to the secretion of endogenous opioids, promoting analgesia and immobility (Faneslow, 1986), as well as the secretion of cortisol, a steroid hormone that serves to inhibit behavior and to check adrenergic responding (Kagan, Reznick, & Snidman, 1987; Kalin, Shelton, Rickman, & Davidson, 1998; Munck, Guyre, & Holbrook, 1984; Sapolsky, Romero, & Munck, 2000). Although the activated state involves predominantly sympathetic arousal and increases in heart rate, in withdrawal the parasympathetic nervous

system is also activated, lowering heart rate. Speculatively, these differential patterns of responding could underpin the current, unexpected findings. We limited psychophysiological measurement to HR in the current study, as the response that has previously shown the largest effect sizes (Wilhelm & Roth, 1998). However, as HR is determined by both sympathetic and parasympathetic branches of the autonomic nervous system, sometimes in co-activation mode (Wilhelm, Kochar, Roth, & Gross, 2001), the physiological underpinnings of this response are complicated. The concurrent measurement of other systems in future studies would disambiguate the physiological effects that we observed.

Regardless of the process underlying the current observations, the findings indicate that not all types of access to trauma material provoke an enhanced physiological response in individuals with PTSD. However, despite their lower physiological responses, trauma survivors with PTSD in the current study self-reported experiencing significantly greater distress in response to recalling the trauma than those without PTSD; consequently, there was no association between self-report measures of distress and HR response. Although anxiety responses are held to be comprised of loosely coupled behavioral, cognitive, and physiological components that generally act in tandem (Lang, 1979), other authors have also observed dissociations between self-reported anxiety and HR responses (Griffin et al., 1997; Grossman, Wilhelm, Kawachi, & Sparrow, 2001; Orr, Pitman, Lasko, & Herz, 1993; Wilhelm et al., 2001).

Perhaps most significant is our observation that a higher HR response during trauma recall was associated with greater subsequent symptom reduction in the PTSD group. Self-reported distress, in contrast, was not related to subsequent symptomatology and may not be a good indicator of degree of emotional processing. The present findings are in line with Foa and Kozak's hypothesis that full access to the trauma memory, particularly characterized by activation of associated emotions with concomitant physiological responding, is necessary for emotional processing and thus recovery to occur (Foa & Kozak, 1986). However, it should be noted that this interpretation of our observations rests on the assumption that the elevated HR we observed in response to trauma recall was a valid indicator of emotional activation. Existing research would suggest a significant contribution of emotional access to HR responding in this context. For example, Pennebaker, Hughes and O'Heeron (1987) reported HR responses during disclosure of emotional material that were similar to our observations (7.9 BPM change in HR compared to 8.0 BPM in the current sample), and a significant emotional component in these responses was demonstrated by comparison to an equivalent, low emotion task. Thus, an emotional component to HR responding in the current study can be inferred based on the well-established relationship between HR increases and emotional states of fear and anxiety; nonetheless, the absence of a nonemotional control condition is a limitation.

Our observations are of potential relevance to the implementation of treatment practice in PTSD, as they suggest that only emotional access which is accompanied by physiological arousal may have therapeutic benefits. They further suggest that such access is not necessarily achieved during reliving episodes even though significant distress is reported, particularly for individuals who are frequently engaging in trauma-related rumination. As such, our findings provide support for the possible utility of measures of distress that assess physiological or motor–behavioral components in addition to self-report indices (Blanchard & Buckley, 1999; Orr & Roth, 2000). This conclusion is broadly supported by the observations of Pitman et al. (1996), who found preliminary evidence that indices of physiological arousal may be useful indicators of exposure treatment efficacy, but no evidence that self-reported distress is significant.

Limitations of the current research need to be considered. As already discussed, we had only one measure of physiological responding in the current study, and we did not compare trauma memory recall to recall of a nontraumatic event; both these factors limited our ability to draw strong conclusions regarding emotional arousal in PTSD versus non-PTSD groups. In addition, we found differences in the degree of HR responding in individuals with PTSD versus without PTSD, but not differences

in absolute HR either during baseline or during trauma recall, a fact that further obscures the significance of our findings. Nonetheless, we believe the contrast between the current findings, and prior reports of higher psychophysiological responding in PTSD in response to trauma cues to be theoretically important. In addition, our data point to a potential source of variation in exposure-based treatment efficacy. Further investigation of the cognitive and biological factors that determine psychophysiological responding during trauma recall may provide valuable information relating to both contraindications for exposure-based therapies in PTSD, and methods for improving the efficacy of such treatments.

REFERENCES

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. Clinical Psychology Review, 8, 77–100.

Beck, A. T., Ward, C. H., Mendleson, M., Mock, J. E., & Erbaugh, J. K. (1961). An Inventory for Measuring Depression. Archives of General Psychiatry, 4, 561–571.

Blanchard, E. B., & Buckley, T. C. (1999). Psychophysiological assessment of posttraumatic stress disorder. In P. A. Saigh & J. D. Bremner (Eds.), Posttraumatic stress disorder: A comprehensive text (pp. 248–266). Needham, MA: Allyn & Bacon.

Blanchard, E. B., Hickling, E. J., Buckley, T. C., Taylor, A. E., Vollmer, A., & Loos, W. R. (1996). Psychophysiology of posttraumatic stress disorder related to motor vehicle accidents: Replication and extension. Journal of Consulting and Clinical Psychology, 64, 742–751.

Blanchard, E. B., Kolb, L. C., Pallmeyer, T. P., & Gerardi, R. J. (1982). A psychophysiological study of post-traumatic stress disorder in Vietnam veterans. Psychiatry Quarterly, 54, 220–229.

Blanchard, E. B., Kolb, L. C., & Prins, A. (1991). Psychophysiological responses in the diagnosis of posttraumatic stress disorder in Vietnam veterans. Journal of Nervous and Mental Disease, 179, 97–101.

Borkovec, T. D., Ray, W. J., & Stöber, J. (1998). Worry: A cognitive phenomenon intimately linked to affective, physiological, and interpersonal processes. Cognitive Therapy and Research, 22, 561–576.

Borkovec, T. D., & Roemer, L. (1995). Perceived functions of worry among generalized anxiety disorder subjects: Distraction from more emotionally distressing topics? Journal of Behavior Therapy and Experimental Psychiatry, 26, 25–30.

Bremner, J. D., Vermetten, E., Southwick, S. M., Krystal, J. H., & Charney, D. S. (1998). Trauma, memory, and dissociation: An integrative formulation. In J. D. Bremner & C. R. Marmar (Eds.), Trauma, memory, and dissociation. Progress in psychiatry 54 (pp. 365–398). Washington, DC: American Psychiatric Press.

Brewin, C. R., Dalgleish, T., & Joseph, S. (1996). A dual representation theory of posttraumatic stress disorder. Psychological Review, 103, 670–686.

Cahill, L., Babinsky, R., Markowitsch, H. J., & Mcgaugh, J. L. (1995). The amygdala and emotional memory. Nature, 377, 295–296.

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. Behaviour Research and Therapy, 38, 319–345.

Ehlers, A., Hackmann, A., & Michael, T. (2004). Intrusive reexperiencing in post-traumatic stress disorder: Phenomenology, theory, and therapy. Memory, 12, 403–415.

Ehlers, A., Mayou, R. A., & Bryant, B. (1998). Psychological predictors of chronic PTSD after motor vehicle accidents. Journal of Abnormal Psychology, 107, 508–519.

Elsesser, K., Sartory, G., & Tackenberg, A. (2004). Attention, heart rate, and startle response during exposure to trauma-relevant pictures: A comparison of recent trauma victims and patients with posttraumatic stress disorder. Journal of Abnormal Psychology, 113, 289–301.

Faneslow, M. S. (1986). Conditioned fear-induced opiate analgesia: A compelling motivational state theory of stress analgesia. In D. D. Kelly (Ed.), Stress-induced analgesia (pp. 40–54). New York: The New York Academy of Sciences.

Foa, E. B. (1995). The Posttraumatic Diagnostic Scale (PDS) manual. Minneapolis, MN: National Computer Systems.

Foa, E. B. (November 1998). New developments in the treatment of PTSD. Paper presented at the annual meeting of the Association for the Advancement of Behavior Therapy, Washington, DC.

Foa, E. B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: The Posttraumatic Diagnostic Scale. Psychological Assessment, 9, 445–451.

Foa, E. B., DiSavino, P., & Turk, E. (1995). Content analysis manual. Unpublished manuscript, University of Pennsylvania.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. Psychological Bulletin, 99, 20–35.

Foa, E. B., Molnar, C., & Cashman, L. (1995). Change in rape narratives during exposure therapy for posttraumatic stress disorder. Journal of Traumatic Stress, 8, 675–690.

Foa, E. B., & Riggs, D. S. (1993). Post-traumatic stress disorder in rape victims. In J. Oldham, M. B. Riba, & A. Tasman (Eds.), Review of psychiatry (pp. 273–303). Washington, DC: American Psychiatric Press.

Foa, E. B., Riggs, D. S., Massie, E. D., & Yarczower, M. (1995). The impact of fear activation and anger on the efficacy of exposure treatment for posttraumatic stress disorder. Behavior Therapy, 26, 487–499.

Foa, E. B., & Rothbaum, B. O. (1998). Treating the trauma of rape: Cognitive-behavioral therapy for PTSD. New York: Guilford Press.

Foa, E. B., Steketee, G., & Rothbaum, B. O. (1989). Behavioral/cognitive conceptualizations of post-traumatic stress disorder. Behavior Therapy, 20, 155–176.

Griffin, M. G., Resick, P. A., & Mechanic, M. B. (1997). Objective assessment of peritraumatic dissociation: Psychophysiological indicators. American Journal of Psychiatry, 154, 1081–1088.

Grossman, P., Wilhelm, F. H., Kawachi, I., & Sparrow, D. (2001). Gender differences in psychophysiological responses to speech stress among older social phobics: Congruence and incongruence between self-evaluative and cardiovascular reactions. Psychosomatic Medicine, 63, 765–777.

Groth-Marnot, G. (1990). The handbook of psychological assessment. New York: Wiley.

Halligan, S. L., Clark, D. M., & Ehlers, A. (2002). Cognitive processing, memory, and the development of PTSD symptoms: Two experimental analogue studies. Journal of Behavior Therapy and Experimental Psychiatry, 33, 73–89.

Halligan, S. L., Michael, T., Clark, D. M., & Ehlers, A. (2003). Posttraumatic stress disorder following assault: The role of cognitive processing, trauma memory, and appraisals. Journal of Consulting and Clinical Psychology, 71, 419–431.

Hoehn-Saric, R., McLeod, D. R., & Zimmerli, W. D. (1989). Somatic manifestations in women with generalised anxiety disorder: Psychophysiological responses to psychological stress. Archives of General Psychiatry, 46, 1113–1119.

Holmes, E. A., Brown, R. J., Mansell, W., Fearon, R. P., Hunter, E. C., Frasquilho, F., et al. (2005). Are there two qualitatively distinct forms of dissociation? A review and some clinical implications. Clinical Psychology Review, 25, 1–23.

Kagan, J., Reznick, J. S., & Snidman, N. (1987). The physiology and psychology of behavioral inhibition in children. Child Development, 58, 1459–1473.

Kalin, N. H., & Shelton, S. E. (1998). Ontogeny and stability of separation and threat-induced defensive behaviors in Rhesus monkeys during the first year of life. American Journal of Primatology, 44, 125–135.

Kalin, N. H., Shelton, S. E., Rickman, M., & Davidson, R. J. (1998). Individual differences in freezing and cortisol in infant and mother Rhesus monkeys. Behavioral Neuroscience, 112, 251–254.

Karvenon, J., Chwalbinska-Moneta, J., & Saynajakangas, S. (1984). Comparison of heart rates measured by ECG and microcomputer. The Physician and Sports Medicine, 12, 65–69.

Lang, P. J. (1979). Presidential Address, 1978. A bio-informational theory of emotional imagery. Psychophysiology, 16, 495–512.

Laukkanen, R. M. T., & Virtanen, P. K. (1998). Heart rate monitors: State of the art. Journal of Sports Sciences, 16, S3–S7.

Ledoux, J. E. (1993). Emotional memory systems in the brain. Behavioural Brain Research, 58, 69–79.

Ledoux, J. E. (1996). The emotional brain. New York: Simon & Schuster.

Ledoux, J. E. (2000). Emotion circuits in the brain. Annual Review of Neuroscience, 23, 155–184.

Marmar, C. R., Weiss, D. S., & Metzler, T. J. (1997). The Peritraumatic Dissociation Scale. In J. P. Wilson & T. M. Keane (Eds.), Assessing psychological trauma and PTSD (pp. 412–428). New York: Guilford Press.

McClelland, J. L., McNaughton, B. L., & O'Reilly, R. C. (1995). Why there are complementary learning systems in the hippocampus and neocortex: Insights from the successes and failures of connectionist models of learning and memory. Psychological Review, 102, 419–457.

Michael, T., Ehlers, A., & Halligan, S. L. (2005). Enhanced priming for trauma-related material in posttraumatic stress disorder. Emotion, 5, 103–112.

Michael, T., Ehlers, A., Halligan, S. L., & Clark, D. M. (2005). Unwanted memories of assault: What intrusion characteristics are associated with PTSD? Behaviour Research And Therapy, 43, 613–628.

Michael, T., Halligan, S. L., Ehlers, A., & Clark, D. M. (in press). Rumination in posttraumatic stress disorder. Depression and Anxiety.

Munck, A., Guyre, P. M., & Holbrook, N. J. (1984). Physiological functions of glucocorticoids in stress and their relation to pharmacological actions. Endocrinology Review, 93, 9779–9783.

Murray, J., Ehlers, A., & Mayou, R. A. (2002). Dissociation and post-traumatic stress disorder: Two prospective studies of road traffic accident survivors. British Journal of Psychiatry, 180, 363–368.

Orr, S. P., Pitman, R. K., Lasko, N. B., & Herz, L. R. (1993). Psychophysiological assessment of posttraumatic stress disorder imagery in World War II and Korean combat veterans. Journal of Abnormal Psychology, 102, 152–159.

Orr, S. P., & Roth, W. T. (2000). Psychophysiological assessment: Clinical applications for PTSD. Journal of Affective Disorders, 61, 225–240.

Papageorgiou, C., & Wells, A. (2004). Nature, functions, and beliefs about depressive rumination. In C. Papageorgiou & A. Wells (Eds.), Depressive rumination: Nature, theory and treatment (pp. 3–20). Chichester: Wiley.

Pennebaker, J. W., Hughes, C. F., & O'Heeron, R. C. (1987). The psychophysiology of confession: Linking inhibitory and psychosomatic processes. Journal of Personality and Social Psychology, 52, 781–793.

Perry, B. D., Pollard, R. A., Blackley, T. L., Baker, W. L., & Vigilante, D. (1995). Childhood trauma, the neurobiology of adaptation, and "use-dependent" development of the brain: How "states" become "traits." Infant Mental Health Journal, 16, 271–289.

Pitman, R. K., Orr, S. P., Altman, B., Longpre, R. E., Poire, R. E., Macklin, M. L., et al. (1996). Emotional processing and outcome of imaginal flooding therapy in Vietnam veterans with chronic posttraumatic stress disorder. Comprehensive Psychiatry, 37, 409–418.

Pitman, R. K., Orr, S. P., Forgue, D. F., De Jong, J. B., & Claiborn, J. M. (1987). Psychophysiologic assessment of posttraumatic stress disorder imagery in Vietnam combat veterans. Archives of General Psychiatry, 44, 970–975.

Rosario, M., Williams, R., & Ehlers, A. (2002). Cognitive processing during motor vehicle accidents predicts posttraumatic stress disorder. Unpublished manuscript, Institute of Psychiatry, King's College London.

Sapolsky, R. M., Romero, L. M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. Endocrinology Review, 21, 55–89.

Schore, A. N. (2002). The effects of early relational trauma on right brain development, affect regulation and infant mental health. Infant Mental Health Journal, 22, 201–269.

Seaward, B. L., Sleamaker, R. H., McAuliffe, T., & Clapp, J. F. (1990). The precision and accuracy of a portable heart rate monitor. Biomedical Instrumentation & Technology, 24, 37–41.

Shalev, A. Y., Orr, S. P., & Pitman, R. K. (1993). Psychophysiologic assessment of traumatic imagery in Israeli civilian patients with posttraumatic stress disorder. American Journal of Psychiatry, 150, 620–624.

Shalev, A. Y., Peri, T., Gelpin, E., Orr, S. P., & Pitman, R. K. (1997). Psychophysiologic assessment of mental imagery of stressful events in Israeli civilian posttraumatic stress disorder patients. Comprehensive Psychiatry, 38, 269–273.

Siegel, D. J. (1996). Cognition, memory, and dissociation. Child and Adolescent Psychiatric Clinics of North America, 5, 509–536.

Spielberger, C. D., Gorsuch, R. L., Lushene, R. E., Vagg, P. R., & Jacobs, G. A. (1983). Manual for the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press.

Wajciechowski, J., Gayle, R., Andrews, R., & Dintiman, G. (1991). The accuracy of radio telemetry heart rate monitor during exercise. Clinical Kinesiology, 45, 9–12.

Wilhelm, F. H., Kochar, A. S., Roth, W. T., & Gross, J. J. (2001). Social anxiety and response to touch: Incongruence between self-evaluative and physiological reactions. Biological Psychology, 58, 181–202.

Wilhelm, F. H., & Roth, W. T. (1998). Taking the laboratory to the skies: Ambulatory assessment of self-report, autonomic, and respiratory responses in flying phobia. Psychophysiology, 35, 596–606.