

Heart Rate Variability (HRV) and Posttraumatic Stress Disorder (PTSD): A Pilot Study

Gabriel Tan · Tam K. Dao · Lorie Farmer ·
Roy John Sutherland · Richard Gevirtz

Published online: 3 August 2010
© Springer Science+Business Media, LLC 2010

Abstract Exposure to combat experiences is associated with increased risk of developing Post Traumatic Stress Disorder. Prolonged exposure therapy and cognitive processing therapy have garnered a significant amount of empirical support for PTSD treatment; however, they are not universally effective with some patients continuing to struggle with residual PTSD symptoms. Heart rate variability (HRV) is a measure of the autonomic nervous system functioning and reflects an individual's ability to adaptively cope with stress. A pilot study was undertaken to determine if veterans with PTSD (as measured by the Clinician-Administered PTSD Scale and the PTSD Checklist) would show significantly different HRV prior to an intervention at baseline compared to controls; specifically, to determine whether the HRV among veterans with PTSD is more depressed than that among veterans without PTSD. The study also aimed at assessing the feasibility, acceptability, and potential efficacy of providing HRV biofeedback as a treatment for PTSD. The findings suggest that implementing an HRV biofeedback as a treatment for PTSD is effective, feasible, and acceptable for veterans. Veterans with combat-related PTSD displayed significantly

depressed HRV as compared to subjects without PTSD. When the veterans with PTSD were randomly assigned to receive either HRV biofeedback plus treatment as usual (TAU) or just TAU, the results indicated that HRV biofeedback significantly increased the HRV while reducing symptoms of PTSD. However, the TAU had no significant effect on either HRV or symptom reduction. A larger randomized control trial to validate these findings appears warranted.

Keywords Heart rate variability · Posttraumatic stress disorder

Introduction

Post Traumatic Stress Disorder (PTSD) is an anxiety disorder resulting from exposure to a traumatic event in which both of the following occurred: (1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of the self or others; and (2) the person's response involved intense fear, helplessness or horror. In addition to exposure, the diagnosis according to the Diagnostic and Statistical Manual of Mental Disorders-IV-TR requires the presence of three clusters of symptoms: persistent re-experiencing of the events, persistent avoidance of the stimuli associated with the trauma or general emotional numbing, and persistent symptoms of increased arousal (American Psychiatric Association 2000).

A substantial portion of veterans exposed to combat or warzone stressors develop PTSD. For Vietnam and Gulf War veterans, the prevalence of PTSD is estimated to be 30 and 10%, respectively (Kang et al. 2003; Schlenger et al. 1992). For current deployments in Iraq and Afghanistan,

G. Tan · L. Farmer · R. J. Sutherland
Michael E. DeBakey Veterans Affairs Medical Center,
Houston, TX, USA

T. K. Dao (✉) · R. J. Sutherland
University of Houston, 491 Farish Hall, Houston,
TX 77004, USA
e-mail: tkdao@uh.edu

G. Tan · T. K. Dao · R. J. Sutherland
Baylor College of Medicine, Houston, TX, USA

R. Gevirtz
Alliant International University, San Diego, CA, USA

the prevalence is less clear (Friedman 2004), however, some have estimated it to be around 17% (Hoge et al. 2004). More recent PTSD rates among active troops are estimated as 16.7%, and even higher among reservists at 24.5% (Milliken et al. 2007). The cost of suffering from a mental disorder such as PTSD can be quite staggering for individuals, their families and society. PTSD is associated with a number of personal health and associated economic costs (Foa et al. 2007; O'Donnell et al. 2005; Schnurr and Green 2004). It is estimated that the cost of PTSD in United States is similar to those of major depression and ranges from \$45–50 billion dollars per year (Kessler 2000).

A number of psychosocial interventions, in addition to pharmacotherapy, have been developed and tested to treat PTSD. Among them, cognitive-behavioral approaches such as prolonged exposure therapy (PE; Foa and Rothbaum 1998) and cognitive processing therapy (CPT; Resick and Schnicke 1992) have garnered a substantial amount of empirical support (Foa et al. 1999, 2005; Nishith et al. 2002; Monson et al. 2006; Resick et al. 2008). The mechanisms of action in both of these treatments differ in that PE focuses on exposure to the traumatic event to extinguish conditioned fears while CPT emphasizes cognitive restructuring to correct faulty thinking patterns. A multidimensional meta-analysis of the psychotherapies for PTSD concluded that: “The majority of patients ...recover or improve...however; exclusion criteria and failure to address polysymptomatic presentations render generalizability to the population of PTSD patients indeterminate...The majority of patients post-treatment continues to have substantial residual symptoms, and follow-up data beyond very brief intervals have been largely absent...” (pp. 214, Bradley et al. 2005). Furthermore, while mental exposure and cognitive restructuring are effective channels through which to target PTSD symptoms, a third channel that has received less attention in the PTSD treatment literature is the psychophysiological system.

Psychophysiology of PTSD

It has been well established that exposure to extreme traumatic events can lead to complex physiological abnormalities (Cohen et al. 1997), resulting in symptoms such as elevated heart rate, increased heart rate responses to physical stressors, and increased blood pressure (Cohen et al. 1997; Pitman et al. 1987), which correspond at the physiological level to the PTSD re-experiencing and increased arousal diagnostic criteria. These symptoms often persist long after the precipitating stressor has been removed (Milliken et al. 2007). These observed changes at the physiological level have led to the proposition that autonomic function, including the reactions of the sympathetic nervous system, may be altered in PTSD patients. In

fact, Orr and Roth (2000) found that physiological reactivity can discriminate between 80 and 100% of persons with and without PTSD. In addition, a meta-analysis of 122 studies investigating psychophysiological variables suggests PTSD is associated with elevated psychophysiology, particularly evident in heart rate and skin conductance in resting baseline studies; heart rate and skin conductance in startle studies; eye blink (EMG); and heart rate in standardized trauma cue studies (Pole 2007).

Elevated psychophysiology is suggestive of autonomic nervous system dysfunction. Autonomic nervous system dysfunction among patients with PTSD is characterized by excess sympathetic nervous system activity or ineffectual parasympathetic activity (i.e., hyperarousal, difficulty with responsiveness to stressors, as well as an impaired relaxation response). These psychophysiological processes suggest that autonomic dysfunction may be a potential pathogenic mechanism in the etiology and maintenance of PTSD, and thus a means of intervention. In fact, Van der Kolk (2006) has argued that somatic therapies may be effective to target the psychophysiological underpinnings of PTSD as cortical executive functions appear suppressed compared to limbic activation for some patients.

Heart Rate Variability and Autonomic Nervous System Function

Heart rate variability (HRV) has been used as an indicator of autonomic nervous system function in many settings (Appelhans and Luecken 2006). HRV can be measured by both instantaneous heart rate and beat-to-beat alterations (R–R intervals) in heart rate (Appelhans and Luecken 2006). Heart rate (HR) does not remain constant but fluctuates around a mean value. This fluctuation is influenced by the autonomic nervous system, which regulates HR through the sympathetic and parasympathetic nervous systems (Cohen et al. 1999). Over time, these cyclic changes in sinus rate are termed heart rate variability (Akselrod et al. 1981). Low HRV has been associated with excessive cardiac sympathetic modulation, inadequate parasympathetic modulation, or both (Task Force 1996). HRV biofeedback is designed specifically to target autonomic reactivity and has become the conventionally accepted term to describe such variations in modulation of the different systems.

Current research suggests that each individual has a resonant frequency at which HRV is the greatest. For instance, Vaschillo et al. (2002) reported that breathing at resonant frequency stimulates the baroreflex, which produces high amplitude heart rate and blood pressure oscillation due to resonance characteristics of the cardiovascular system. The resonant frequency in humans occurs between 0.075 and 0.120 Hz with individual resonant frequency

averaging around 0.092 Hz, which corresponds to 5.5 breaths per minute (Vaschillo et al. 2002). At resonant frequency, heart rate and blood pressure oscillate 180° out of phase, while heart rate and respiration oscillate in phase with each other (Vaschillo et al. 2002). The consequent resonance effects produce very large increases in both HRV and baroreflex gain. Thus, the central focus of heart rate variability biofeedback is to train individuals to learn how to control their breathing with the goal of slowing down the breathing rate to the frequency at which the amplitude of HRV is maximized.

In summary, we know that there is considerable evidence of empirically-supported PTSD treatment options available. However, we do not know whether HRV biofeedback can improve patients' PTSD symptoms by focusing on modification of physiological reactivity. Thus, the goal of this pilot study was threefold. First, we compared baseline HRV in two groups of patients: (1) combat PTSD veterans and (2) patients without PTSD. Second, we examined the effect of HRV biofeedback on PTSD symptoms. Third, we examined the feasibility and acceptability of HRV biofeedback as a treatment for combat-related PTSD symptoms.

We hypothesize that, at baseline, patients diagnosed with combat-related PTSD will have lower HRV compared to subjects with no PTSD diagnosis. This hypothesis reflects the expectation that patients with combat-related PTSD will show greater autonomic nervous system dysregulation compared to patients without combat-related PTSD. We also hypothesize that HRV biofeedback will improve PTSD symptoms, particularly decrease the hyperarousal symptoms. This hypothesis is based on previous findings by Lehrer et al. (2000) and reflects the expectation that improvements in PTSD symptoms could occur through modulations of the autonomic nervous system.

Methods

Participants

Institutional Review Board approval was obtained from the hospital facility as well as from the authors' University Human Subject's Committee. A convenience sample of 20 veterans with PTSD was recruited from the Michael E DeBakey VA Medical Center via one of the following channels: veterans routinely screened by the OEF/OIF outreach staff from the Trauma Recovery Program (TRP), posted flyers and announcements displayed throughout the hospital, or referrals from the two Veterans Centers in Houston. In addition to the 20 participants with PTSD, ten volunteers were recruited. These ten subjects denied any

history of PTSD or other psychiatric disorders. The inclusion criteria for the PTSD group consisted of a diagnosis of combat-related PTSD and agreement to adhere to protocol requirements. The exclusion criteria was a presence of severe psychopathology that would preclude adherence to protocol procedures (e.g., actively psychotic, active substance abuse), significant cognitive deficits (i.e., Mini Mental State Examination score below 17 or equivalent), or previous participation in another study involving HRV.

Measures

Heart Rate Variability

A power spectral analysis of HRV and a packet of standardized psychometric instruments consisting of the Clinician-Administered PTSD Scale (CAPS; Weathers and Litz 1994) and the PTSD Checklist-Specific (PCL-S; Weathers et al. 1993) were used in the study. These measures were administered before and after the completion of the 8-week treatment protocol.

Power Spectral Analysis of HRV Power spectral analysis (PSA) assesses the quantitative contribution of high frequency (HF; 0.15–0.5 Hz), low frequency (LF; 0.04–0.15 Hz), and very low frequency (VLF; 0.01–0.04 Hz) components to the total variance, or “power” of heart rate. The index of particular interest was the SDNN (standard deviation of the beat-to-beat interval averaged over successive 5 min intervals; often also referred to as SDANN). The assessment of heart rate variability was gathered via recordings of blood volume pulse (BVP) and respiration using the Thought Technology Infiniti system (See also www.thoughttechnology.com). The BVP and respiration sensors were hooked up to the participants at sites recommended by the manufacturer for HRV analysis. Study recordings were made in the Psychophysiology Lab at the Michael E. DeBakey VA Medical Center (MEDVAMC).

For the respiration measure, the BVP was recorded according to recommended procedures. An elastic band of the respirometer was adjusted to a snug, but comfortable tightness around participants' upper abdomen. The BVP sensor was fitted to the index finger of the right hand. Participants were then instructed to maintain open eyes and avoid moving their wrists while the research assistant read excerpts from a collection of pleasant (relatively neutral) travel stories. This is a common HRV experimental paradigm design to mimic normal waking state levels of arousal. HRV was recorded for 15 min for each participant. At the end of the session the recordings were coded and saved for subsequent analysis. Movement artifacts were automatically removed by the Infiniti software from the session overview which provides a display of the total

session of respiration and BVP data. This is accomplished by using a Boolean Rejection algorithm that deletes sequential pulse waves that were 20% above or below the previous sequence.

Posttraumatic Stress Disorder

The Clinician-Administered PTSD Scale (CAPS) The CAPS (Weathers and Litz 1994) is a 30-item structured interview and can be used to make a current (past month) or lifetime diagnosis of PTSD or to assess PTSD symptoms over the past week. Questions target the impact of symptoms on social and occupational functioning, improvement in symptoms since a previous CAPS administration, overall response validity, overall PTSD severity, and frequency and intensity of five associated symptoms (guilt over acts; survivor guilt; gaps in awareness; depersonalization; and derealization). The CAPS asks patients a variety of questions relating to frequency and intensity of the 17 PTSD symptoms. For instance, interviewees are asked to rate the frequency of PTSD symptoms on a 0–4 scale with 0 being “Never” and 4 being “Daily or almost every day.” They are also asked to rate how much distress or discomfort these memories cause them on a 0–4 scale with 0 being “Mild, minimal distress or disruption of activities” and 4 being “Extreme, incapacitating distress, cannot dismiss memories, unable to continue activities.” The CAPS is composed of three subscales; re-experiencing, avoidance and numbing, and hyper arousal. Cronbach’s alpha for internal consistency has been found to range from .73 to .85 for intensity of PTSD symptom criteria (Litz et al. 2000). Interrater reliability was found to range from .92 to .99 for frequency and intensity (Weathers and Litz 1994). The CAPS has been shown to demonstrate convergent validity with self-report measures of PTSD (r ’s range from .70 to .84) and combat exposure ($r = .42$) and predictive validity with heart rate reactivity in response to a combat-related priming event (Litz et al. 2000; Weathers and Litz 1994). The three subscales have demonstrated high internal consistency with alphas of .88 for re-experiencing, .87 for avoidance and numbing, .88 for hyperarousal (Weathers and Litz 1994).

The PTSD Checklist-Specific (PCL-S) The PCL-S (Weathers et al. 1993) consists of 17 items which correspond to the DSM-IV symptoms of PTSD. The Specific version of the PCL was chosen to correspond to the method of the CAPS which involves assessment of PTSD symptoms associated with an identified index trauma. Examinees are instructed to indicate how much they have been bothered by each symptom in the past month using a 5-point scale with 1 being “not at all” to 5 being “extremely.” A sample item of the PCL-S will ask a patient to

rate how much they are bothered by “repeated, disturbing memories, thoughts or images of a stressful military experience.” The PCL has shown to have good internal consistency in Vietnam and Persian Gulf veterans, victims for motor vehicle accidents, and sexual assault survivors with reliability coefficients ranging from .77 to .93 (Weathers et al. 1993). A number of studies have examined the diagnostic efficiency of the PCL and found sensitivity ranging from .78 to .82 and specificity ranging from .84 to .86 using a cutoff of .50 in predicting PTSD diagnosis (Ruggiero et al. 2003; Weathers et al. 1993). Convergent and discriminant validity has also been demonstrated for the PCL (Weathers et al. 1993).

Procedure

After obtaining informed consent, all participants were asked to complete a standard battery of measures that included the Clinician-Administered PTSD Scale (CAPS), PTSD Checklist (PCL-S), and HRV assessment using spectral analysis (previously described under “measures”). The 20 participants with PTSD were then randomly assigned to one of two groups: The Experimental Group (EXP) was provided the HRV biofeedback treatment in addition to treatment as usual (TAU) while the Control Group (CON) received only TAU. No attempt was made to control the type and amount of TAU for both groups. They were informed of services available at the MEDVAMC Trauma Recovery Program and/or the local Veteran Centers and to continue their treatment if they were already enrolled.

Participants in the EXP group was administered a total of 8 weekly sessions of 30 min HRV biofeedback as per Lehrer’s protocol (Lehrer et al. 2000). The protocol consisted of a total of 8 sessions including the administration of the pre and post HRV measurements. After obtaining the baseline HRV (see Lehrer et al. 2000 for a more detailed description), participants’ resonant frequency were determined using a pacer stimulus. To determine each participant’s optimal resonant frequency, the pacer was set at different frequencies (e.g., 6.5, 6, 5.5, 5, 4.5 breaths/min), and participants were asked to breath for 2 min. Breathing for 2 min allowed for computation of frequency spectra from at least ten breaths at each frequency to determine personal resonant frequency. Heart rate oscillation amplitudes were measured resulting in a series of highest to lowest frequency HRV. Subjects were then asked to continue breathing at their personal resonant frequency for 20 min to produce maximal increases in amplitude of HRV. Subjects were cautioned to breathe slowly and naturally, allowing for longer exhalation than inhalation in order to avoid hyperventilation. After completion of the session, participants were provided with a CD which

corresponded to their specific resonant frequency for home practice. They were instructed to listen to the CD and pace their breathing to flow with the rhythms of the CD for 20 min, twice daily. After completion of the biofeedback treatment for the EXP group or 8 weeks after the initial assessment for the CON group, all participants were asked to repeat the questionnaire packet and the post treatment spectral analysis of their HRV was computed.

Data Analysis

To address the first hypothesis that patients diagnosed with combat PTSD will have lower HRV at baseline compared to subjects that were not diagnosed with combat PTSD, we computed two separate *t*-tests. In the first *t*-test, we compared HRV measure for patients diagnosed with PTSD to available age and gender controlled norms. In the second *t*-test, we compared HRV measure for patients in the PTSD group versus “normal” volunteers.

To address the hypothesis that HRV biofeedback will decrease PTSD symptoms, we followed the analytic plan recommended by Keppel and Wickens (2004) for situations with clear directional hypotheses. Based on the hypothesized simple effects (skipping testing the interaction), four separate paired sample *t*-tests (or simple effects analyses) were computed. The first *t*-test compared baseline and post baseline CAPS and PCL-S scores for the EXP group. The second *t*-test compared baseline and post baseline CAPS and PCL-S scores for the CON group. The third *t*-test compared mean change scores on the CAPS and PCL-S for the EXP and CON groups. In the last *t*-test, we compared CAPS subscales scores in the EXP and CON groups.

Analysis of HRV Measures

Heart rate data was analyzed using the standard deviation of sequential interbeat intervals averaged over 5 min epochs (SDANN). This measure was chosen as a measure of HRV because it is considered the most straightforward and is the most commonly used metric of HRV (Task Force 1996; Nolan et al. 2008). The raw inter-beat intervals (IBI) data was exported from the Infiniti software and visually scanned in the Biosignal Analysis Program (copyrighted @ Biosignal Analysis and Medical Imaging Group 2008). IBIs that were greater or less than 20% of the previous value were averaged based on the surrounding values as recommended in the International Task Force guidelines (Task Force 1996). In addition, visual inspection was used to detect any ectopic or “bad” beats and editing was done as above. The final data was analyzed using the Biosignal software with Smoothing Priors as a detrending algorithm (interpolation rate of 2 Hz).

Feasibility of this treatment was assessed based on the ease of recruitment, treatment adherence, and treatment completion rates. Treatment adherence was assessed by examining adherence to treatment instruction and home practice. Treatment completion was determined by computing the percentage of participants who completed the treatment by attending all sessions out of all who agreed to participate. Acceptability was assessed by informal structured interview with participants after the treatment was over regarding level of satisfaction, duration number of sessions, convenience, clarity of information, and utility of the treatment.

Results

Characteristics of the Samples

The PTSD group ranged in age from 24 to 62 years (mean age = 36, SD = 13.1) and 100% were male ($n = 20$). The ethnic composition of the group consisted of 40% Caucasian ($n = 8$), 35% African American ($n = 7$), 20% Hispanic ($n = 4$) and 5% Asian ($n = 1$). The EXP group ($n = 10$) ranged in age from 24 to 67 years (mean age = 45) and 90% were male ($n = 9$). The CON group ($n = 10$) ranged in age from 24 to 62 years (mean age = 39, SD = 12.9) and 70% were male ($n = 7$). There were 7 Vietnam era vets and 13 OEF/OIF vets in the sample (with 5 Vietnam vets randomly assigned to EXP and 2 to CON groups).

Group Equivalence Among Samples

Table 1 contains descriptive statistics comparing selected demographic variables for the different samples. Marital status was the only demographic variable that was statistically significant suggesting that there was significant difference between the frequencies of the different classification levels between the PTSD and the Normal groups.

Group Equivalence on Treatment Factors

To assess equivalence in treatment related variables between the PTSD EXP and CON groups, chi square tests were performed on the frequencies of individual and group therapy sessions, and number of psychiatrist contacts during the 8-week period of the study. For individual therapy, the chi square test indicated no significant difference, $\chi^2(1, n = 20) = 1.98, p = .15$. A chi square test comparing frequency of group therapy session “attended” to “not attended” indicated no significant difference $\chi^2(1, n = 24) = .01, p = .94$ as well between the 2 groups. In regards to psychiatrist contact appointments, there was

Table 1 Group equivalence testing across demographic variables

	PTSD (<i>n</i> = 20)		Normal (<i>n</i> = 10)		<i>p</i> -Value	EXP (<i>n</i> = 10)	CON (<i>n</i> = 10)	<i>p</i> -Value
Age	40.7 (16.9)		32.4 (32.4)		.156	44.3 (19.0)	37.1 (14.9)	.383
Ethnicity					.240 ^a			.294 ^b
White	5 (27.8%)		5 (50.0%)			1 (11.1%)	4 (44.4)	
Other	13 (72.2%)		5 (50.0%)			8 (88.9%)	5 (55.6%)	
Marital status					.016 ^b			.968 ^b
Single	5 (27.8%)		8 (80.0%)			2 (22.2%)	3 (33.3%)	
Other	13 (72.2%)		2 (20.0%)			7 (77.8%)	6 (66.7%)	

^a Chi square calculated for significance testing

^b Fisher's exact test calculated for significance testing

Table 2 Comparison of SDNN (standard deviation of beat-to-beat interval) between veterans with PTSD and "normal" control

	PTSD (<i>n</i> = 20)		Normal (<i>n</i> = 10)		<i>p</i> -Value	Cohen's <i>d</i>
	Mean	SD	Mean	SD		
SDNN	48.10	47.87	138.70	47.87	<.001	1.89

Cohen's *d* calculated using pooled standard deviation and weighted for unequal samples sizes

no significant difference between the EXP and CON groups, $\chi^2(1, n = 23) = .290, p = .59$.

Depressed HRV Among Veterans with PTSD

Table 2 compares the average SDANN index score for the 20 veterans diagnosed with PTSD and the average SDANN for the ten subjects in "Normal" control group. The difference was statistically significant suggesting that the veterans diagnosed with PTSD display lower HRV when compared to those without PTSD. This difference represents a large, clinically significant effect ($d = 1.89$).

Efficacy of the HRV Biofeedback Treatment

Table 3 provides the results for paired sample *t*-tests examining differences between baseline and post treatment scores on the CAPS and the PCL-S for the EXP group. The

Table 3 Mean baseline and post baseline scores on the CAPS and PCL-S for the EXP group

	Baseline		Post baseline		<i>p</i> -Value	Cohen's <i>d</i>
	Mean	SD	Mean	SD		
CAPS	86.41	19.32	71.24	18.51	<.001	.80
PCL-S	64.82	7.43	54.43	11.53	.035	1.08

EXP Patients diagnosed with PTSD and given HRV biofeedback plus treatment-as-usual. Cohen's *d* calculated using pooled standard deviation and weighted for unequal samples sizes. CAPS Clinician Administered PTSD Scale, PCL-S PTSD check list

10 subjects in the EXP group had an average score of 86.41 (SD = 19.32) on the CAPS at baseline and an average score of 71.24 (SD = 18.51) at post treatment. This difference was statistically significant, $t(9) = 6.81, p < .0001, d = .80$. Similarly, on the PCL-S, average scores of 64.82 (SD = 7.43) and 54.43 (SD = 11.53) on the PCL-S found at baseline and post treatment respectively resulted in a statistically significant reduction in symptoms, $t(9) = 2.47, p = .035, d = 1.08$.

Table 4 provides the results for paired sample *t* tests examining differences between baseline and post treatment scores on the CAPS and the PCL-S for the CON group. Subjects in the CON group had an average CAPS baseline score of 89.13 (SD = 24.32) and an average CAPS post treatment score of 80.80 (SD = 25.23). The difference was not statistically significant, $t(9) = 1.51, p = .163$. The same 10 subjects had an average PCL-S score of 62.74 (SD = 12.71) and an average PCL-S post treatment score of 61.74 (SD = 10.72). This difference was also not statistically significant, $t(9) = .234, p = .820$.

Table 5 provides the results for the between group analysis of the EXP and CON groups. The mean differences between baseline and post treatment scores on the CAPS and the PCL-S were 15.23 (SD = 7.14) and 10.41 (SD = 13.32) respectively for those in the EXP group, and 8.33 (SD = 17.34) and 1.01 (SD = 13.54) respectively for those in the CON group. The mean change scores for the CAPS and PCL-S were not significantly different between

Table 4 Mean baseline and post baseline scores on the CAPS and PCL-S scores for the CON group

	Baseline		Post baseline		<i>p</i> -Value	Cohen's <i>d</i>
	Mean	SD	Mean	SD		
CAPS	89.13	24.32	80.80	25.23	.163	.33
PCL-S	62.74	12.71	61.74	10.72	.820	.09

CON patient diagnosed with PTSD and given treatment-as-usual. Cohen's *d* calculated using pooled standard deviation and weighted for unequal samples sizes

Table 5 Mean change scores on CAPS and PCL-S between EXP and CON groups

	EXP group (<i>n</i> = 10)		CON group (<i>n</i> = 10)		<i>t</i> -Statistics	<i>p</i> -Value	Cohen's <i>d</i>
	Mean ^a	SD	Mean ^a	SD			
CAPS	15.23	7.14	8.33	17.34	1.17	.266	.52
PCL-S	10.41	13.32	1.01	13.54	1.57	.135	.70

EXP patients diagnosed with PTSD and given HRV biofeedback plus treatment-as-usual, CON patient diagnosed with PTSD and given treatment-as-usual

^a Mean, Mean change scores between baseline and post baseline

Table 6 Between group comparison of CAPS subscales for the EXP and CON groups

CAPS subscales	EXP group (<i>n</i> = 10)		CON group (<i>n</i> = 10)		<i>t</i> -Statistics	<i>p</i> -Value	Cohen's <i>d</i>
	Mean	SD	Mean	SD			
Re-experiencing	20.03	3.64	27.74	6.82	1.80	.082	1.42
Avoiding/numbing	32.12	5.01	38.73	8.31	2.21	.044	.963
Hyperarousal	40.91	8.61	43.62	4.50	.912	.392	.393

EXP patients diagnosed with PTSD and given HRV biofeedback plus treatment-as-usual, CON patient diagnosed with PTSD and given treatment-as-usual

the EXP and CON groups, $t(18) = .266$, $p = .266$ for the CAPS, and $t(18) = .135$, $p = .135$.

Table 6 provides the results for the between group comparison of CAPS PTSD diagnostic symptom categories for the EXP and the CON groups. The mean difference between the EXP (Mean = 20.3; SD = 3.64) and CON (Mean = 27.74; SD = 6.82) groups on the re-experiencing subscale was not statistically significant, $t(18) = 1.80$, $p = .081$ (two-tailed), $d = 1.42$. The mean difference between the EXP (Mean = 32.12; SD = 5.01) and CON (Mean = 38.73; SD = 8.31) groups on the avoiding/numbing subscale was statistically significant, with the EXP group reporting significantly less avoidance/numbing symptoms than the CON group, $t(18) = 2.21$, $p = .044$ (two-tailed), $d = .963$. The mean difference between the EXP (Mean = 40.91; SD = 8.61) and CON (Mean = 43.62; SD = 4.50) groups on the hyperarousal subscale was also not statistically significant, $t(18) = .912$, $p = .392$ (two-tailed), $d = .393$.

Clinical Significance

These preliminary results, if replicated, would constitute a fairly meaningful reduction in PTSD symptoms. For example the biofeedback group reduced their CAPS scores by 18% compared to 9% for the TAU controls. The resulting score of 71.24, while still elevated, would indicate symptom reduction that would be noticeable to the patient. The 15 point reduction in the CAPS score for the biofeedback group would most of the best treatment outcome studies in the literature (Hofmann and Smits 2008). In a meta analysis by Hofmann and Smits (2008) the average

Hedges g (comparable to our d scores) was .64 for six well controlled studies. Thus our values (.80 for the CAPS and 1.08 for the PCL) compare favorably.

Freed et al. (2009) used a Preference-weighted Health Status (PWHS) measure to assign clinical meaning to PCL scores. A change of .041 units was considered clinically important. Based on our PCL improvements, the, our differential PWHS was .06, a large improvement in overall health status.

Feasibility of the Study

Our findings indicate that subjects in the study had no problems adhering to the treatment instructions. They followed instructions during face-to-face sessions and completed their homework in a timely manner. Only one subject prematurely withdrew from the study due to transportation problems. Thus, 95% of subjects completed treatment protocol. Subjects also reported that the instructions were clear and they had no problems attending the required number of sessions.

Acceptability of the Study

Immediately after the study, nine out of ten subjects reported that they were satisfied with the treatment and its benefits. After 6 months, informal follow-up structured phone interviews were conducted with subjects to assess treatment benefits. Nine out of ten of those interviewed re-iterated their approval of the treatment as well as their benefits. These subjects reported using the breathing techniques they learned to successfully reduce their PTSD

symptoms. Five out of 9 of these subjects reported that they continue to try to breathe at their resonant frequency daily. Three out of 9 of these subjects reported using their breathing technique when they encounter stressful situations or when they have increase PTSD symptoms, particularly when they are irritable or having difficulty concentrating. Overall, subjects reported average ratings of 8 out of 10, with 10 being very satisfied with the treatment. Furthermore, when asked about preference of this treatment over medications, nine out of ten subjects indicated that they prefer this treatment over medications. Their reasons included statements such as medications “don’t work” to “This treatment has helped me teach myself how to control my own PTSD symptoms.”

Discussion

The results of this pilot study indicated that veterans with combat-related PTSD exhibited significantly depressed (i.e., less responsive) HRV, a manifestation of ANS dysregulation compared to those without PTSD. The study also showed that HRV biofeedback was both feasible and acceptable to the veterans for their PTSD treatment.

The pilot study also examined the potential efficacy of HRV biofeedback as a treatment for PTSD. Our findings indicated that veterans receiving the 8 sessions (total 6 face-to-face treatment hours) of HRV biofeedback plus TAU showed significant reductions in PTSD symptoms post-treatment (on both CAPS and PCL measures) while those receiving only TAU did not. The symptom reductions were particularly evident in the avoidance/emotional numbing cluster of the CAPS. Improvement in avoidance and emotional numbing symptoms could constitute a major step towards recovery, whether by means of the biofeedback intervention investigated in this pilot study or as an adjunct to exposure-based or cognitive restructuring-based models. In addition, improvement in these symptoms may benefit social reintegration, a task identified as of high importance in facilitating long-term veteran readjustment. As there has been no published data to date to support the relationship between the avoidance/numbing cluster of PTSD symptoms and functioning and reintegration of our returning troops back into society, our findings of that our intervention not only reduced PTSD symptoms but particularly improved the avoidance/numbing symptoms merit further investigation.

Although the PTSD EXP group showed significant reductions in PTSD symptoms, the comparison in symptom reduction between the PTSD EXP and CON groups was not statistically significant (which may be due to the small sample size), the change was in the right direction as revealed by the moderate effect sizes of changes in overall PTSD symptoms (Cohen’s $d = 0.52$ – 0.70 for CAPS and

PCL-S respectively). While the findings are encouraging, a larger clinical trial is needed to confirm the efficacy of this intervention. The results are also limited because only finger pulse data was available to assess HRV. ECG measures should be gathered to substantiate these findings.

The findings of this pilot study are consistent with other research studies indicating that HRV biofeedback training provides a reduction in psychiatric symptoms associated with trauma (Karavidas et al. 2007; Zucker et al. 2007). The findings further indicate that it is feasible and acceptable to veterans to conduct such an intervention among those with combat-related trauma.

Given that PTSD symptoms encompass symptoms such as increased arousal/hypervigilance, intense fear and horror and persistent avoidance behavior, it would appear that this treatment which focuses on decreasing physiological arousal/hypervigilance could be useful either alone or in combination with existing psychosocial treatments for PTSD that target cognition and behavior (as in CBT and CPT). The implications of our findings require further investigation as to whether they would be useful in addition to exposure-based models (as in PE), as exposure-based models utilize confrontation of trauma memories and are based on emotional habituation to feared stimuli. When offering PE, clinicians are trained to attend to the level of engagement individuals have with their trauma memories for appropriate emotional processing. For example, over-engagement (i.e., unstoppable crying, rocking, dissociative re-experiencing of the event) interferes with this task, and under-engagement (i.e., mental or emotional avoidance of the event) does not elicit enough emotions for successful processing and exposure. It may be that this type of biofeedback training could help “prepare” individuals to better regulate their emotional engagement during such a meaningful confrontation of their trauma memories. Perhaps combination treatments targeting ANS dysfunction and exposure or cognitive restructuring could provide a stronger and more robust and synergistic effect on reducing the symptoms of PTSD and their persistence.

References

- Akselrod, S., Gordon, D., Ubel, F., Shannon, D., Barger, A. C., & Cohen, R. J. (1981). Power spectral analysis of heart rate fluctuation: A quantitative probe of beat-to-beat cardiovascular control. *Science*, *213*, 220–222.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th Ed., text revision). Washington, DC: Author.
- Appelhans, B. M., & Luecken, L. J. (2006). Heart rate variability as an index of regulated emotional responding. *Review of General Psychology*, *10*, 229–240.
- Biosignal Analysis and Medical Imaging Group. (2008). Biosignal Analysis Program.

- Bradley, R., Greene, J., Russ, E., Dutra, L., & Westen, D. (2005). A multidimensional meta-analysis of psychotherapy for PTSD. *American Journal of Psychiatry*, *162*, 214–227.
- Cohen, H., Kotler, M., Matar, M., Kaplan, Z., Miodownik, H., & Cassuto, Y. (1997). Power spectral analysis of heart rate variability in posttraumatic stress disorder patients. *Biological Psychiatry*, *41*, 627–629.
- Cohen, H., Matar, M., Kaplan, Z., & Kotler, M. (1999). Power spectral analysis of heart rate variability in psychiatry. *Psychotherapy and Psychosomatics*, *68*, 59–66.
- Foa, E. B., Dancu, C. V., Hembree, E. A., Jaycox, L. H., Meadows, E. A., & Street, G. P. (1999). A comparison of exposure therapy, stress inoculation training, and their combination for reducing posttraumatic stress disorder in female assault victims. *Journal of Consulting and Clinical Psychology*, *67*, 194–200.
- Foa, E. B., Hembree, E. A., Cahill, S. P., Rauch, S. A., Riggs, D. S., Feeny, N. C., et al. (2005). Randomized trial of prolonged exposure for posttraumatic stress disorder with and without cognitive restructuring: Outcome at academic and community clinics. *Journal of Consulting and Clinical Psychology*, *73*, 953–964.
- Foa, E. B., Hembree, E. A., & Rothbaum, B. O. (2007). *Prolonged exposure therapy for PTSD: Emotional processing of traumatic memories, therapist guide*. New York: Oxford University Press.
- Foa, E. B., & Rothbaum, B. O. (1998). *Treating the trauma of rape: Cognitive behavioral therapy for PTSD*. New York: Guilford Press.
- Freed, M. C., Yeager, D., Liu, Xian., Gore, K., Engel, C., et al. (2009). Preference-weighted health status of PTSD among veterans: Outcome for cost-effectiveness analysis using clinical data. *Psychiatric Services*, *60*, 1230–1238.
- Friedman, M. J. (2004). Acknowledging the psychiatric cost of war. *New England Journal of Medicine*, *351*, 75–77.
- Hofmann, S. G., & Smits, J. A. (2008). Cognitive-behavioral therapy for adult anxiety disorders: A meta-analysis of randomized placebo-controlled trials. *Journal of Clinical Psychiatry*, *4*, 621–632.
- Hoge, C. W., Castro, C. A., Messer, S. C., McGurk, D., Cotting, D. I., & Koffman, R. L. (2004). Combat duty in Iraq and Afghanistan, mental health problems, and barriers to care. *New England Journal of Medicine*, *351*, 13–22.
- Kang, H. K., Natelson, B. H., Mahan, C. M., Lee, K. Y., & Murphy, F. M. (2003). Post-traumatic stress disorder and chronic fatigue syndrome-like illness among Gulf War veterans: A population based survey of 30, 000 veterans. *American Journal of Epidemiology*, *157*, 141–148.
- Karavidas, M. K., Lehrer, P. M., Vaschillo, E., Vaschillo, B., Marin, H., Buyske, S., et al. (2007). Preliminary results of an open-label study of heart rate variability biofeedback for the treatment of major depression. *Applied Psychophysiology and Biofeedback*, *32*, 19–30.
- Keppel, G., & Wickens, T. (2004). *Design and analysis: A researchers' handbook*. Upper Saddle River, NJ: Pearson/Prentice Hall.
- Kessler, R. C. (2000). Posttraumatic stress disorder: The burden to the individual and to society. *Journal of Clinical Psychiatry*, *61*, 4–12.
- Lehrer, P., Vaschillo, E., & Vaschillo, B. (2000). Resonant frequency biofeedback training to increase cardiac variability: Rational and manual for training. *Applied Psychophysiology and Biofeedback*, *25*, 177–191.
- Litz, B. T., Orsillo, S., Kaloupek, D., & Weathers, F. W. (2000). Emotional-processing in PTSD. *Journal of Abnormal Psychology*, *109*, 26–39.
- Milliken, C. S., Auchterlonie, M. S., & Hoge, C. W. (2007). Longitudinal assessment of mental health problems among active and reserve component soldiers returning from the Iraq war. *JAMA*, *298*, 2141–2148.
- Monson, C. M., Schnurr, P. P., Resick, P. A., Friedman, M. J., Young-Xu, Y., & Stevens, S. P. (2006). Cognitive processing therapy for veterans with military-related posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *74*, 898–907.
- Nishith, P., Resick, P. A., & Griffin, M. G. (2002). Pattern of change in prolonged exposure and cognitive-processing therapy for female rape victims with posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *70*, 880–886.
- Nolan, R. P., Jong, P., Barry-Bianchi, S. M., Tanaka, T. H., & Floras, J. S. (2008). Effects of drug, biobehavioral and exercise therapies on heart rate variability in coronary artery disease: a systematic review. *European Journal of Cardiovascular Prevention & Rehabilitation*, *15*, 386–396.
- O'Donnell, M. L., Creamer, M., Elliott, P., & Atkin, C. (2005). Health costs following motor vehicle accidents: The role of posttraumatic stress disorder. *Journal of Traumatic Stress*, *18*, 557–561.
- Orr, S. P., & Roth, W. T. (2000). Psychophysiological assessment: Clinical applications for PTSD. *Journal of Affective Disorder*, *61*, 225–240.
- Pitman, R. K., Orr, S. P., Foa, E. B., de Jong, J. B., & Claiborn, J. M. (1987). Psychophysiological assessment of posttraumatic stress disorder imagery in Vietnam combat veterans. *Archives of General Psychiatry*, *44*, 970–975.
- Pole, N. (2007). The psychophysiology of post traumatic stress disorder: a meta-analysis. *Psychological Bulletin*, *133*, 725–746.
- Resick, P. A., Galovski, T. E., Uhlmansiek, M., Scher, C. D., Clum, G. A., & Young-Xu, Y. (2008). A randomized clinical trial to dismantle components of cognitive processing therapy for posttraumatic stress disorder in female victims of interpersonal violence. *Journal of Consulting and Clinical Psychology*, *76*, 243–258.
- Resick, P. A., & Schnicke, M. K. (1992). Cognitive processing therapy for sexual assault victims. *Journal of Consulting and Clinical Psychology*, *60*, 748–756.
- Ruggiero, K. J., Del Ben, K., Scotti, J. R., & Rabalais, A. E. (2003). Psychometric properties of the PTSD Checklist-civilian version. *Journal of Traumatic Stress*, *16*, 495–502.
- Schlenger, W. E., Kulka, R. A., Fairbank, J. A., Hough, R. L., Jordan, B., Marmar, C., et al. (1992). The prevalence of post-traumatic stress disorder in the Vietnam generation: A multimethod, multisource assessment of psychiatric disorder. *Journal of Traumatic Stress*, *5*, 333–363.
- Schnurr, P. P., & Green, B. L. (2004). Understanding relationships among trauma, post-traumatic stress disorder, and health outcomes. *Advances in Mind-Body Medicine*, *20*, 18–29.
- Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. *European Heart Journal*, *17*, 354–381.
- Thought Technology .com. (2009). *Thought technology infinity system*. Available at: www.thoughttechnology.com. Retrieved July 1, 2009.
- Van Der Kolk, B. A. (2006). Clinical implications of neuroscience research in PTSD. *Annals of the New York Academy of Sciences*, *1071*, 277–293.
- Vaschillo, E., Lehrer, P., Rische, N., & Konstantinov, M. (2002). Heart rate variability biofeedback as a method for assessing baroreflex function: A preliminary study of resonance in the cardiovascular system. *Applied Psychophysiology and Biofeedback*, *27*, 1–27.
- Weathers, F. W., & Litz, B. T. (1994). Psychometric properties of the Clinician-Administered PTSD Scale, CAPS-1. *PTSD Research Quarterly*, *5*, 2–6.
- Weathers, F. W., Litz, B. T., Herman, D. S., Huska, J. A., & Keane, T. M. (1993, October). *The PTSD checklist (PCL): Reliability, validity, and diagnostic utility*. Paper presented at the meeting of the International Society for Traumatic Stress Studies, San Antonio, TX.
- Zucker, T. L., Samuelson, K. W., Muench, F., Greenberg, M. A., & Gevirtz, R. N. (2007). The effects of respiratory sinus arrhythmia biofeedback on heart rate variability and posttraumatic stress disorder symptoms: a pilot study. *Applied Psychophysiology and Biofeedback*, *34*, 134–156.