The relationship between posttraumatic stress disorder (PTSD) and parasympathetic nervous system (PNS) was investigated during a resting baseline period and two 4-minute laboratory speech tasks. Participants were 20 women with PTSD and 20 age- and gender-matched controls. PNS cardiac control was measured as the high-frequency (0.12-0.40 Hz) component of heart rate variability (HF-HRV) using power spectrum analysis. Participants with PTSD had significantly greater reductions in HF-HRV during the speech tasks (trauma recall: M = -0.84, SD = 0.71; mental arithmetic: M = -0.64, SD = 0.56) than controls (trauma recall: M = -0.33, SD = 0.53; mental arithmetic: M = -0.25, SD = 0.41), $p = .03$. PTSD was related to the magnitude of decrease in PNS cardiac control during stress in women.
AUTONOMIC NERVOUS SYSTEM FUNCTIONING IN POSTTRAUMATIC STRESS DISORDER AT REST AND DURING STRESS: THE ROLE OF THE PARASYMPATHETIC NERVOUS SYSTEM

A thesis submitted
to Kent State University in partial fulfillment of the requirements for the degree of Master of Arts

by

Therese Keary

December, 2008
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>LIST OF FIGURES</td>
<td>v</td>
</tr>
<tr>
<td>INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>METHODS</td>
<td>13</td>
</tr>
<tr>
<td>RESULTS</td>
<td>26</td>
</tr>
<tr>
<td>DISCUSSION</td>
<td>38</td>
</tr>
<tr>
<td>REFERENCES</td>
<td>45</td>
</tr>
<tr>
<td>APPENDIX A – SONG SELECTIONS</td>
<td>58</td>
</tr>
</tbody>
</table>
LIST OF FIGURES

Figure | Page |
-------|------|
1 | Mean LF-HRV values throughout the laboratory protocol by group ............34 |
2 | Mean HF-HRV values throughout the laboratory protocol by group ...........35 |
3 | Mean LF/HF ratios throughout the laboratory protocol by group ............36 |
4 | Changes in HF-HRV from baseline to each speech task by group ...........37 |
INTRODUCTION

Posttraumatic Stress Disorder (PTSD) is an anxiety disorder that can develop following exposure to a terrifying event or trial in which death or serious injury occurred or was threatened. The estimated lifetime prevalence of PTSD among adults in the United States is approximately 7-8% (American Psychiatric Association, 2000; Kessler et al., 1995; Kessler et al., 2005). A growing body of research evidence indicates that PTSD confers an increased risk of cardiovascular disease (Beckham et al., 2002; Boscarino & Chang, 1999; Boscarino, 2004; Boscarino, 2006; Buckley et al., 2004; Kubzansky et al., 2007; Schnurr et al., 2000). Previous research had indicated that blood pressure (BP) and heart rate (HR) tend to be higher among individuals with PTSD relative to individuals without PTSD (for reviews, see Blanchard, 1990; Buckley & Kaloupek, 2001). A substantial literature also documents that individuals with PTSD often exhibit dysregulation in ANS functioning, both in terms of basal levels (“tone”) and changes in response to stress-related cues (for a review, see Friedman & Schnurr, 1995). This is not surprising given criterion D of the Diagnostic and Statistical Manual of Mental Disorders, fourth edition, text revision (DSM-IV-TR; American Psychiatric Association, 2000), which specifies that persistent symptoms of hyperarousal must be present for a diagnosis of PTSD.

Hyperarousal: The Autonomic Nervous System
The autonomic nervous system (ANS) regulates the body’s internal environment. The ANS consists of two anatomically separate divisions, the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). The SNS governs functions related to arousal and energy expenditure, while the PNS controls the body’s vegetative, restorative, and growth functions. Both branches seek to maintain cardiovascular homeostasis on a short-term basis (i.e., beat-to-beat) in response to internal and environmental challenges, such as physical demands and emotional distress (Akselrod et al., 1981; Porges, 1995). Most organs, including the heart, are innervated by the vagus nerve, which controls PNS functioning. Increased vagal efferent outflow promotes a reduction in heart rate, whereas decreased vagal outflow promotes an increase in heart rate.

SNS activity can be measured with a variety of methods, including plasma norepinephrine assays, urinary norepinephrine excretion, direct nerve recordings via microneurography and noradrenaline radiotracer, neural imaging techniques, and pre-ejection period via impedance cardiography (Grassi et al., 1998; Grassi & Esler, 1999; Mancia et al., 1998; Schachinger et al., 2001). A common limitation of measuring SNS activity is that there is no single index that summarizes SNS activity (Sherwood & Hughes, 2004; Brownley et al., 2000). There are three non-invasive measures of PNS activity: heart rate variability (HRV), baroreceptor sensitivity (BRS), and heart rate recovery (HR recovery). HR recovery, as indexed by the rate of HR return to baseline following the termination of exercise, is predominantly mediated by vagal activation during the first 30-60 seconds (Shetler et al., 2001). Investigations examining HR
recovery have mainly been confined to the cardiology literature, although some recent investigations have reported on the relationship between HR recovery and depression symptoms (Hughes et al., 2006a; Hughes et al., 2008).

HRV refers to cyclic variations in heart rate, or more precisely, variations in beat-to-beat (i.e., RR) intervals. The high frequency (HF) component of HRV (i.e., 0.15-0.40 Hz; HF-HRV) is generally considered to be an index of parasympathetic cardiac control (Akselrod et al., 1981; Cacioppo et al., 1994). HF-HRV is also known as respiratory sinus arrhythmia (RSA), as HF-HRV values are associated with the rate and volume of respiration (Grossman et al., 2004; Grossman & Taylor, 2007). The low frequency component (i.e., 0.04-0.15 Hz; LF-HRV) is considered to be an aggregate reflection of both PNS and SNS activity (Burr, 2007). The ratio of LF-HRV to HF-HRV power (LF/HF ratio) represents, to some extent, sympathovagal balance (Pagani et al., 1986), with higher ratios indicating inadequate cardiac parasympathetic tone and/or excessive cardiac sympathetic activity. The very low frequency (≤0.04 Hz; VLF) component is of HRV is typically obtained from 24-hour Holter recordings instead of laboratory-based psychophysiology investigations. The physiological correlates of VLF are largely unknown (Task Force of the European Society of Cardiology the North American Society of Pacing, 1996).

BRS is a vagal index of the functioning of a reflexive regulation of blood pressure via baroreceptors located within the carotid arteries and aorta. Arterial baroreflexes contribute substantially to PNS tone (LaRovere et al., 1995). BRS and HF-HRV are significantly correlated (Mancia et al., 1986; Reyes del Paso et al., 1996). However, HRV
analysis is better suited for documenting changes in PNS activity, and thus, may be considered a within-subjects measure appropriate for evaluating how the PNS modulates cardiovascular activity (Berntson et al., 1997). BRS analysis, in contrast, outperforms HRV when attempting to document an absolute level of PNS input to the heart (“tone”). For example, BRS has been demonstrated to be a better predictor of PNS tone during beta-adrenergic blockade, when HR is predominantly under vagal control (Reyes del Paso et al., 1996). It should be acknowledged that non-invasive indices of HRV (including HF-HRV and BRS) are imperfect measures of parasympathetic tone. Invasive pharmacological autonomic blockade studies provide the best estimates of the relative contributions of each of autonomic branches to cardiac functioning (Berntson et al., 1994). However, non-invasive measures of autonomic control are important because pharmacological blockades in human psychophysiological studies are often not feasible or desirable (Berntson et al., 1994)

Strong parasympathetic modulation of HR (i.e., ample HF-HRV and/or BRS) helps to maintain cardiovascular control in terms of beat-to-beat adjustments to environmental demands, whereas reduced parasympathetic modulation of HR inhibits an individual’s ability to adapt (Porges & Byrne, 1992). Strong parasympathetic cardiac control also helps to maintain the electrical stability of the heart, and for example protects against fatal ventricular arrhythmias (Billman et al., 1984). HRV estimates have already been shown to offer prognostic information regarding mortality in a population-based sample of elderly participants (Tsuji et al., 1994), chronic heart failure patients (Nolan et
For several decades, the two branches of the ANS were classically considered to operate in opposition to one another. Consequently, researchers historically operated under the assumption that the doctrine of autonomic reciprocity best described the interaction between the SNS and PNS. This doctrine postulated that the SNS and PNS were subject to reciprocal central control, such that increased activity in one branch was associated with decreased activity of the other. While considerable evidence supports the concept of a reciprocal, opponent organization of the ANS, more recent research has indicated that autonomic control cannot be sufficiently viewed as a single continuum that exists between SNS and PNS dominance. As Porges (1995) observed, there are situations in which both the SNS and PNS are activated (e.g., sexual arousal) or inhibited (e.g., anesthesia), and there are some disease states (e.g., diabetes) that are characterized by reduced parasympathetic tone without a reciprocal sympathetic activation.

A more contemporary conceptualization is that autonomic control of the heart can be depicted in a bivariate (sympathetic activation x parasympathetic activation) autonomic space. According to this two dimensional representation, the two branches of the ANS can vary reciprocally, independently, or coactively (Berntson et al., 1991; Berntson et al., 1993). Depending upon the relative input of the SNS and PNS at any given moment, the cumulative effect of ANS functioning can be either excitatory or inhibitory in nature. Accordingly, there may be three ways in which elevations of
cardiovascular activity may occur: 1) via increases in SNS activity or 2) via decreases in PNS activity, or 3) a combination of both increased SNS activity and decreased PNS activity (DeMeersman & Stein, 2007).

**PTSD: The SNS**

Initially, the negative cardiovascular outcomes associated with elevated basal cardiovascular arousal in PTSD (Blanchard, 1990; Buckley & Kaloupek, 2001) were typically explained in terms of elevated sympathetic nervous system (SNS) activity. This initial conceptualization reflected a principal focus on the sympathetic branch of the ANS in the PTSD psychophysiology research.

**PTSD: The PNS**

Investigations of the role of the parasympathetic activity in the cardiovascular functioning of individuals with PTSD are warranted for several reasons. As stated above, contemporary conceptualizations of the ANS indicate that reduced parasympathetic activity may help to account for the higher HR and BP observed in PTSD (Blanchard, 1990; Buckley & Kaloupek, 2001). In addition, prolonged autonomic imbalance is frequently associated with poor cardiovascular health. For example, reduced parasympathetic tone has been specifically implicated in cardiovascular risk factors, disease processes, and outcomes (for a review, see Thayer & Lane, 2007). However, there have been surprisingly few studies of PNS involvement in cardiovascular functioning among individuals with PTSD. In the literature comparing PTSD patients
with healthy controls, there are five studies employing HRV (Blechert et al., 2007; Cohen et al., 1997; Cohen et al., 1998; Cohen et al., 2000; Sahar et al., 2001), two investigations utilizing BRS (Hughes et al., 2006b; Hughes et al., 2007), and no studies that have used HR recovery. There are no published reports that measured parasympathetic activity with both HF-HRV and BRS in PTSD samples.

**HRV investigations.** The first study of PNS functioning in PTSD compared nine PTSD patients (six men, three women) and nine healthy controls (matched for age, sex, and smoking status) during a 20 minute resting period (Cohen et al., 1997). Results indicated that the PTSD group had higher HR, lower total HRV, higher LF-HRV, higher LF/HF ratio, and lower HF-HRV relative to the control group. These results were interpreted as evidence that the basal autonomic state of PTSD may be characterized by increased sympathetic and decreased parasympathetic tone. The investigators did not report whether or not participants in the control group had a trauma exposure history.

A subsequent manuscript based on the same sample reported the results of autonomic responses to a 20-minute speech stress task (Cohen et al., 1998). Participants in the PTSD group were asked to recount the triggering traumatic event presumed to underlie the disorder, while participants in the control group recounted a significant stressful negative life event. Results indicated that there were no significant changes in either HF-HRV or LF-HRV between the resting baseline and speech task in the PTSD group. The investigators postulated that this lack of response may be attributed to the state of chronic autonomic overstimulation, which restricts the capacity to respond further during stress. The investigators also suggested that the absence of change in sympathetic
tone during the speech task may be due to a ceiling effect associated with the use of HRV analysis. Results of the Cohen et al. (1998) study also indicated that neither the PTSD group nor the control group exhibited significant changes in HR during the speech task. Given that the typical stress response to a trauma-relevant speech task is increased HR (Orr et al., 1998), a lack of response may merely reflect a failure of the laboratory stress task to evoke any response (e.g., failed manipulation check). In addition, it should be noted that a long verbal narrative may not be well-suited for eliciting and evaluating cardiovascular responses to psychological stressors. The largest HR response to a laboratory stress task involving speaking is usually observed near the beginning of the task (i.e., during the first 2-3 minutes). The 20-minute speech task employed in the Cohen et al. (1998) study may have allowed for habituation to occur. Nonetheless, the atypical cardiovascular responses during the laboratory stress task obscures interpretation of the results.

Both of these first two investigations by Cohen and colleagues (1997; 1998) had a basal HR effect size difference (i.e., Cohen’s $d$ of 3.23) that was approximately 9 and 7.5 times the effect sizes obtained in a meta-analytic investigation which compared individuals with PTSD to non-PTSD trauma-exposed controls and non-trauma controls, respectively. As noted by Sahar (2001), the control group in these two investigations had a very low mean HR (i.e., 61.9 bpm) that is quite similar to the HR of a group of highly fit collegiate cross-country runners (Hatfield et al., 1998). Accordingly, as Sahar et al. (2001) proposed, the substantial difference in basal HR may have been the consequence of atypically high PNS tone among the control participants, rather than low PNS tone.
among the PTSD participants. Given the small samples, it is also possible that sampling error may have influenced the pattern of results obtained in these investigations.

A third study of PNS functioning in PTSD compared 14 PTSD patients, 11 panic disorder (PD) patients, and 25 matched controls during a 15-minute resting period and a 15-minute speech task (Cohen et al., 2000). Participants in the PTSD group were asked to recount the triggering traumatic event presumed to underlie the disorder, participants in the PD group described a typical panic attack, and participants in the control group recounted a significant stressful negative life event. Results indicated that both patient groups had elevated resting HR and LF-HRV and lower HF-HRV compared to controls. HR and LF-HRV in the PTSD group were higher than the PD group at trend levels, while HF-HRV in the PTSD group was lower than the PD group at trend levels (all \( p = 0.065 \)). However, the PTSD group, in contrast to both the PD and control groups, failed to respond to the speech task with increases in HR and LF-HRV or with decreases in HF-HRV. The group-specific nature of the recall task raises some concern regarding whether the degree of stress was equivalent for all three groups. Despite this, the investigators (Cohen et al., 1998; Cohen et al., 2000) once more attributed the observed lack of response in the PTSD group to a state of chronic autonomic overstimulation, which restricts the capacity of the ANS to respond further during stress. Again, the 15-minute speech task employed in the Cohen et al. (2000) study may have allowed for habituation to occur.

A fourth study of PNS functioning in PSTD was conducted by a separate group of researchers (Sahar et al., 2001). Fourteen males with PTSD and 15 trauma-exposed
controls (matched for gender, age, type of trauma, time since the most recent traumatic event, and lifetime exposure to traumatic events) were compared during a 7-minute resting baseline period and a 3.5-minute mental arithmetic task. Results indicated that groups did not differ on resting measures of heart rate and HF-HRV. Results also indicated that there was a divergence in HF-HRV response to the mental arithmetic task, such that the PTSD group exhibited the typical decrease in HF-HRV, whereas the controls demonstrated an increase in HF-HRV. In other words, the HF-HRV response evidenced by the PTSD group suggested a larger decrease in PNS activity than the control group. It should also be noted that the laboratory stress task was not trauma related and was much shorter in duration than the speech stress task employed in the Cohen et al. studies (1998; 2000).

In the largest study to date, Blechert et al. (2007) examined 23 PTSD patients, 26 PD patients, and 32 healthy controls during a 5-minute resting baseline and a 5-minute stress period. The investigators of this study examined the autonomic response to a different type of stress paradigm (i.e., a threat of shock) in order to attempt to equalize the laboratory stress task experience across groups. Results indicated that the PTSD group had lower resting HF-HRV than both the PD and control groups. Both patient groups had higher resting HR compared to the control group. No reactivity was obtained in either HR or HF-HRV during the threat of shock period, so reactivity could not be compared between groups.

_BRS investigations._ There have been two other relevant studies that have examined the relationship between PTSD and parasympathetic nervous system
functioning via examination of BRS. The first BRS study compared 80 PTSD patients and 50 controls (all current smokers) during a 5-minute resting baseline period (Hughes et al., 2006b). Results indicated that women with PTSD, but not men with PTSD, exhibited resting lower BRS than controls. The investigators noted that the discrepant findings for men and women may not reflect a genuine gender difference, but rather as may have been due to sample characteristics (i.e., the men in the study were older, more likely to be veterans, more likely to have combat experience, more likely to be heavy smokers than women).

In a later investigation of BRS, Hughes et al. (2007) compared women in four diagnostic groups [PTSD without major depressive disorder (MDD), PTSD with comorbid MDD, MDD without PTSD, and controls with neither psychiatric diagnosis] during a 5-minute resting baseline period and an approximately 6-minute anger recall speaking task. Results indicated both PTSD groups (with and without MDD) exhibited lower resting BRS than the control group. For reactivity analyses, the three psychiatric groups were collapsed into one category. Results of the reactivity analyses indicated that the magnitude of the decrease in BRS values was smaller during the anger recall task for psychiatric patients than controls. However, when resting BRS scores were controlled for, the relationship between diagnostic category and change in BRS no longer remained significant; this result likely indicated the presence of a floor effect of baseline BRS levels among the psychiatric groups.
The majority of the evidence reviewed here suggests that, in addition to elevated sympathetic activity, PTSD may be characterized by altered PNS functioning. However, the results of these studies are not entirely consistent.

*The Present Study*

The primary purpose of the present study was to examine the relationship between PTSD diagnosis and ANS functioning at rest and during stress. The present study focused primarily on the role of the PNS and is a case-control investigation comparing individuals with PTSD and individuals who are free from major psychopathology using a laboratory mental stress protocol. The protocol included a 20-minute resting baseline, two 4-minute stressogenic speech tasks, and two 10-minute resting recovery periods. The resting baseline period allowed for an examination of basal PNS functioning. The two separate speech tasks included in the protocol allowed for comparison of a trauma-relevant and non-trauma related neutral psychological stressors. It was hypothesized that:

*Hypothesis 1.* Participants with PTSD will evidence lower parasympathetic activity relative to controls during the resting baseline period.

*Hypothesis 2.* All participants will evidence decreases in PNS activity as a function of the two stressogenic speech tasks.

*Hypothesis 3.* Participants with PTSD will evidence larger decreases in parasympathetic activity during both stressogenic speech tasks as compared to control participants.
METHOD

Participants

Participants were 40 women (20 participants with PTSD and 20 control participants without PTSD). Women with PTSD were recruited from the Center for the Treatment and Study of Traumatic Stress (CTSTS), an outpatient ambulatory psychological clinic located at St. Thomas Hospital in Akron, Ohio. Patients being treated at the CTSTS were contacted only if they provided signed consent to be contacted for research and provided their phone numbers. Control participants were recruited from advertisements and from referrals from PTSD participants. Control participants were matched with the PTSD patients for age and gender.

Inclusion and exclusion criteria. To qualify, participants had to be female and between 18 and 64 years of age. Prospective participants were excluded if they: 1) were taking beta-blockers 2) had diabetes, 3) had a history of heart problems or conditions (including a heart murmur or congenital heart disease), or 4) had body mass index (BMI) greater than 35. Individuals with BMI values greater than 35 were excluded from the study because of the difficulty obtaining psychophysiology measurements from these individuals. No attempt was made to alter any participant’s medication regimen for the purposes of the present study. For the participants with PTSD, there were no specific exclusion criteria with respect to comorbid Axis-I diagnoses or use of psychiatric
medications. However, control participants did not meet lifetime diagnostic criteria for PTSD or any other mood or anxiety disorders.

Recruitment Procedures. Prospective participants were contacted via telephone for recruitment. During the telephone contact, a brief description of the study’s purpose and methods were given to the prospective participants. Interested prospective participants were asked to answer a brief series of pre-screening questions in order to exclude individuals with diabetes, heart conditions, and chronic diseases. Prospective participants were informed that all of the answers to the pre-screening questions would remain confidential and would only be made available as necessary to the immediate members of the research team. All participants were asked provide information regarding their sociodemographic and medical characteristics; see the sociodemographic and medical characteristics sections below.

Measures

Sociodemographic characteristics. The following sociodemographic characteristics were assessed: age, race, education level, and perceived socioeconomic status. Age (years) was represented as a continuous variable. Race included the categories of non-Hispanic White, African-American, and other (which consisted of Asian, Native Hawaiian or Pacific Islander, Native American or Alaska Native, and Hispanic). Education level was based on the number of completed years of formal education and was represented as a continuous variable. Marital status consisted of two categories: currently married and not currently married. Perceived socioeconomic status (SES) was
assessed using the MacArthur Scale of Subjective Social Status (Adler et al., 2000). This instrument was developed to assess subjective perceptions of social status by using a 10-point self-anchoring visual scale. In a pictorial format, the scale presents two social ladders which represent standing within one’s local community (i.e., the community ladder) and within society (i.e., the traditional SES ladder). Participants are asked to place and “X” on the ladder rung on which they feel they stand. Higher scores indicate higher perceived rank of the participant’s SES relative to the income, education, and occupational prestige. The scale has demonstrated adequate test-retest reliability (Operario et al., 2004).

Medical characteristics. Height (inches) and weight of each participant were measured at the beginning of the protocol using a standard balance beam scale (438 Detecto Eye-Level Physician Scale, Detecto, Webb City, MO). Weight was a continuous variable measured in pounds. Body mass index (BMI) was a continuous variable calculated from the patient’s height and weight [i.e., BMI = (weight in pounds / (height in inches)^2) x 703]. Medication was represented by two classes: psychiatric medications and cardiovascular medications. Patients were coded as currently taking or not currently taking medications in each of the two classes.

Psychological Assessment Instruments. All participants completed several modules of the Structured Clinical Interview for DSM-IV (SCID; First et al., 1996), a standardized interview designed to obtain Axis I diagnoses. The SCID assesses Axis I psychopathology diagnoses that are commensurate with criteria outlined in the Diagnostic and Statistical Manual of Mental Disorders, fourth edition, text revision
(DSM-IV-TR; American Psychiatric Association, 2000). Administration of the SCID was used to provide information on whether participants met *DSM-IV* criteria for PTSD and other axis I disorders. The specific modules administered for this study were: Major Depressive episode, Manic/ Hypomanic episode, Dysthymic Disorder, Alcohol Abuse/Dependence, Substance Abuse/ Dependence, Panic Disorder, Social Phobia, Specific Phobia, Obsessive Compulsive Disorder, PTSD, Generalized Anxiety Disorder, Anorexia Nervosa, and Bulimia Nervosa.

The Beck Depression Inventory II (BDI; Beck et al., 1979) is a widely used and well-validated measure used to assess depressive symptomatology during the two weeks prior to participation in the study. Each of the twenty-one items on the BDI consists of four statements representing increasing degrees of severity with scores ranging from 0 to 3. Total scores on the BDI can range from zero (no depression) to a maximum score of 63 (severe state of depression). A meta-analysis of the BDI’s internal consistency estimates produced mean alpha coefficients of 0.86 and 0.81 for psychiatric and non-psychiatric patients, respectively (Beck et al., 1988). Studies of the concurrent validity of the BDI with other self-rating scales and clinical ratings of depression yield moderate to high correlation coefficients, with mean coefficients ranging from 0.58 to 0.79 (Beck et al., 1988; Richter et al., 1998).

The PTSD Symptom Scale (PSS-SR; Foa et al., 1993) is a self-report inventory consisting of 17 items which were derived from the *Diagnostic and Statistical Manual of Mental Disorders, third edition, revised* (DSM-III-R; American Psychiatric Association, 1987) symptom criteria of PTSD. The PSS-SR has demonstrated sound psychometric
properties among similar, trauma-based populations (Foa et al., 1993). More specifically, the PSS-SR has demonstrated high internal consistency (Cronbach’s alpha = .91), as well as respectable one month test-retest reliability (r = .74).

The Traumatic Stress Schedule (TSS; Norris, 1990) is a short screening instrument that is used to detect the occurrence and impact of traumatic events. Items on this scale were derived from the DSM-III-R criterion A1 of PTSD. The one month test-retest reliability for the event portion of this instrument is .88 and the symptom portion is moderately reliable, with Cronbach’s alpha equal to .76 (Norris & Hamblen, 2004).

*Physiological Assessment Instruments.* Height (inches) and weight (pounds) measurements of each participant were taken at the beginning of the protocol using a standard balance beam scale (438 Detecto Eye-Level Physician Scale, Detecto, Webb City, MO). These measurements were used to calculate BMI. Individuals with BMI values greater than 35 were excluded from the study because individuals with such high BMIs have less accurate cardiac impedance measurements.

For the purposes of the present study, the following physiological variables were measured: systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), mean arterial pressure (MAP), respiration rate, the low frequency component of HRV (LF-HRV), the high frequency component of HRV (HF-HRV), and the ratio of LF-HRV to HF-HRV (LF/HF).

Blood pressure (BP) refers to the amount of force exerted by the blood on the walls of the blood vessels. BP is numerically expressed in millimeters of mercury (mm Hg) as the ratio of systolic blood pressure (SBP) over diastolic blood pressure (DBP).
SBP indicates the amount of force when the heart pushes blood out into the arteries (i.e., when the heart contracts). DBP indicates the amount of force when the heart is filling with blood (i.e., when the heart is at rest). Normotensive values of BP are those that are less than 120 mm Hg systolic and less than 80 mm Hg diastolic (Chobanian et al., 2003). MAP refers to the average pressure within an artery over a complete cycle of one heartbeat. Blood pressure was assessed every minute throughout the laboratory protocol using an Accutor Plus Oscillometric blood pressure device (Datascope Corp, Mahwah, NH). The blood pressure assessments yielded SBP, DBP, and MAP.

The electrocardiographic (ECG) signal was recorded from two disposable electrodes (Conmed, Utica, NY) attached to the participant’s chest in a modified lead-II configuration. The ECG was sampled continuously at 500 Hz with a BIOPAC systems MP150WSW A/D converter version 3.8.1 (Goleta, CA). AcqKnowledge Software (BIOPAC systems, Goleta, CA) and appropriate signal transducers were used to acquire and display signals on a Dell personal computer (PC). Each minute of ECG data was visually inspected offline, and data reasonably free from artifacts after the application of a high-pass digital filter were included for analysis. AcqKnowledge software was used to detect R-waves and to obtain a point event series of successive response-response (R-R) intervals, from which the beat-to-beat heart series was computed. Following R-wave detection, the interbeat interval (IBI) series was generated and saved for analysis in Nevrokard software version 6.6.5 (Medistar, Inc., Ljubljana, Slovenia). Each data file was again visually inspected for artifacts, which were manually corrected. Power spectral densities of the R-R intervals were computed in Nevkard using a fast Fourier transform,
which decomposes the variance in the frequency domain (ms²/Hz). Consistent with published guidelines for frequency-domain computations of HRV (Task Force of the European Society of Cardiology the North American Society of Pacing, 1996), spectral power was divided into LF-HRV (0.04-0.15 Hz) and HF-HRV (0.15-0.40 Hz). A ratio of LF-HRV to HF-HRV (i.e., LF/HF) was calculated; this ratio represents, to some extent, sympathovagal balance (Pagani et al., 1986). LF-HRV and HF-HRV values were calculated as the natural log of the area under the power spectrum (ms²) within the corner frequencies of the band-pass filter.

Mean HR was calculated as 60,000 divided by the mean of the heart period time series (ms²). Respiratory amplitude signals were sampled continuously at 500 Hz using a Biopac band strain gauge respirometer placed around the participant’s chest. The mean respiratory rate was calculated using AcqKnowledge software.

Procedure

The study protocol was approved by Institutional Review Boards of both Kent State University and Summa Health System.

Pre-study instructions. Participants were directed not to exercise and not to drink alcohol or caffeine the day of their participation in the study. HR can be influenced by physical exertion (e.g., from exercise) and alcohol/caffeine consumption. Although some medications may also affect HR, participants were permitted to take their prescription medications as directed by their physician. Medication use was assessed. The participants were also permitted to smoke as they normally did until they entered the hospital for assessment.
Informed consent. Upon arrival at the laboratory, each participant was presented with a written informed consent form that provided a detailed description of the procedure. Before participants gave their signed consent to participate, participants were provided with a full verbal description of the study’s procedure and they were given the opportunity to have questions or concerns addressed. A second signed consent form was obtained from each participant to audiotape the SCID and the trauma recall stress task (described below). A third signed consent form was obtained that granted permission to review the participant’s Summa Health System medical records.

Physiology laboratory protocol. Participants were seated upright in a phlebotomy chair. To minimize any potential anticipatory stress, a detailed description of the procedure was given before the patient was connected to the physiological measurement equipment. Spot electrodes were attached directly on the skin above two areas: 1) the left clavicle, and 2) the lower right side (just below the rib cage). Prior to placement of spot electrodes, the skin contact point was wiped with alcohol and lightly exfoliated with NuPrep skin prepping gel (Bio-Medical Instruments, Inc., Warren, MI) to facilitate signal acquisition. A standard inflatable BP occlusion cuff was placed around the upper portion of the participant’s non-dominant arm. Next, a strain gauge respirometer was placed around the chest above the participant’s clothing. Finally, a small microphone was taped to the participant’s chest to collect heart sounds.

Resting baseline. Data collection began with a 20-minute resting baseline period. Participants were instructed to sit quietly and avoid crossing their legs while resting measures were obtained. The lights to the laboratory were dimmed and a selection of
relaxing classical music was played from a CD; see Appendix for the song selection list. This period of resting baseline was intended to allow participants to acclimate to the laboratory setting and to become accustomed to being attached to the measurement equipment.

Following the 20-minute baseline period, participants were given instructions regarding the first of two stressogenic challenge tasks, a trauma-neutral task and a trauma-relevant task. These standardized mental stressors provide a controlled means of examining the effect of mental stress on physiological variables. For example, these controlled stressors can lead to increases in blood pressure, heart rate, cardiac output, and cardiac contractility within minutes (Bacon et al., 2006). Each speech stress task lasted four minutes. The sequential presentation of the two stressogenic challenge tasks was counterbalanced to control for order effects.

Trauma-neutral task. The trauma-neutral task was a mental arithmetic stressor in which participants were asked to perform a serial seven subtraction task orally, beginning with a three-digit number provided by the technician. If a participant produced an incorrect answer, the study technician provided the correct answer, and then the participant was asked to continue with the correct answer.

Trauma-relevant task. For the trauma-relevant task, participants were asked to recall and describe in vivid detail a traumatic event that they have experienced. This task was modeled on prolonged exposure treatments for PTSD (Foa et al., 1993). The technician asked participants to describe the trauma that led them to seek treatment and/or to describe the trauma that they specifically mentioned during the PTSD module.
of the SCID. Control participants who had not experienced a traumatic event were instructed to describe a past significant stressful negative life event (Cohen et al., 1998; Cohen et al., 2000). Participants were asked to close their eyes during their verbal recall to reduce distractibility and to describe their experiences in the present tense. If a participant stopped speaking spontaneously, she was prompted by the technician to provide further details.

Recovery periods. The stressogenic tasks were separated by a 10-minute relaxation period to permit recovery of cardiovascular responses. The participants were again asked to sit quietly and avoid crossing their legs. Following the second stressogenic task, participants had a final 10-minute resting period where they were asked to sit quietly so that their cardiovascular activity can return to basal levels.

Debriefing. Upon completion of the laboratory protocol, participants were fully debriefed regarding the purposes of the research project. Participants who met criteria for hypertension (i.e., 140 or greater mm Hg SBP and/or 90 or greater mm Hg DBP) (Chobanian et al., 2003) during the resting baseline phase of the protocol were encouraged to contact their personal physician for further evaluation. Because the study measured several of the risk factors for coronary heart disease (e.g., high BP, increased age, male gender, African American race, and high BMI), the study technician reviewed each participant’s set of risk factors and provided them with a sheet listing the risk factors measured during the study. In addition, all participants were given a pamphlet guide (American Heart Association, 2003) that more fully discusses the risk factors for coronary heart disease. Following debriefing, participants were excused.
**Analytic Strategy**

All statistical analyses were performed using Statistical Package for the Social Sciences (SPSS) software, version 15 (Chicago, IL, 2007). Descriptive analyses (i.e., frequencies and percentages, means and standard deviations) were used to assess the characteristics of the PTSD and control participants. In preliminary analyses, the PTSD and control participants were compared with respect to sample characteristics (e.g., age, race, education, perceived SES, weight, BMI, smoking status, current medications, BDI total score, PSS-SR total score, and SCID diagnoses). Sample characteristics of the PTSD and control participants were compared using independent sample t-tests for continuous variables and chi-square analyses for categorical variables. The criterion for statistical significance was set at \( p < .05 \) for all analyses.

To increase reliability, values for each of the physiological indices (i.e., SBP, DBP, HR, MAP, respiration rate, LF-HRV, HF-HRV, and LF/HF ratio) were averaged over the last 4 minutes of the resting baseline, all four minutes of both of the stressogenic speech tasks, and the last 4 minutes of both resting recovery periods. Both the LF- and HF-HRV indices were log transformed.

**Baseline analyses.** A series of independent samples \( t \) tests on each of the physiological indices were conducted to examine group (PTSD vs. Control) differences in blood pressure and heart rate variability.

**Primary analyses.** A series of 2 (PTSD vs. Control) x 2 (task order) x 5 (phases of the protocol) mixed model repeated measures ANOVAs were conducted for each of the
physiological indices to examine whether or not the counterbalancing method negated any potential confound regarding how the stress tasks were ordered. This was followed by a series of (2 PTSD presence/absence) x 2 (task order) x 5 (phases) ANOVAS were conducted to verify that each of the physiological parameters changed significantly throughout the laboratory protocol and to determine whether or not there were any significant group by phase interactions.

Two types of planned comparisons were conducted if a physiological parameter changed significantly throughout the laboratory protocol (i.e., significant effect of phase). The contrasts were intended to clarify the nature of the changes. First, simple contrasts were conducted to compare the mean of each task and recovery phases to the mean of a reference phase, which was the resting baseline period. This allowed for cardiovascular reactivity during stress tasks to be documented, and also evaluated whether physiological responses during recovery phases had returned to near baseline levels. Second, repeated contrasts were conducted to compare the mean of each phase to the mean of the subsequent phase.

Reactivity analyses. For each physiological index with a significant group by phase interaction, two separate task reactivity scores were calculated by subtracting initial baseline values from each task value. A series of 2 (PTSD vs. Control) x 2 (traumatic recall vs. mental arithmetic) ANOVAs were conducted to test group differences in reactivity.

For all analyses involving more than two levels of a repeated factor, probability values were adjusted for violations of sphericity by using the Greenhouse-Geisser
correction for degrees of freedom. Greenhouse-Geisser statistics are reported as epsilon (\(\varepsilon\)) statistics.
RESULTS

Description of the Sample

The sample included 40 women (20 participants with PTSD and 20 control participants without PTSD). As can be seen in Table 1, groups did not differ in terms of age, race, perceived community SES, marital status, weight, BMI, and current cardiovascular medication use. Control participants completed significantly more years of education (M = 15.9, SD = 2.0) than participants with PTSD (M = 13.5, SD = 2.5), t(38) = -3.35, p ≤ .01. Participants in the control group were free of current psychiatric diagnoses. Participants with PTSD were significantly more likely to currently smoke (χ² = 8.53, p ≤ .01) and use psychiatric medications (χ² = 32.7). Control participants had a significantly higher perceived U.S. SES (M = 5.7, SD = 1.3) than participants with PTSD (M = 4.0, SD = 2.3), t(28.71) = 2.91, p ≤ .01.

As expected, the groups differed on both psychometric scores. The PTSD group had significantly higher scores on the BDI (M = 26.6, SD = 14.2) than control group (M = 4.6, SD = 5.7), t(24.94) = 6.45, p ≤ .01. Mean BDI scores for the PTSD and control groups were very similar to those obtained in similar investigations (Blechert et al., 2007; Sahar et al., 2001). In addition, the PTSD group had significantly higher scores on the PSS-SR (M = 32.1, SD = 12.0) than the control group (M = 7.9, SD = 7.4), t(31.77) = 7.70, p ≤ .01.
Additional descriptive statistics for the clinical and demographic characteristics of both groups are presented in Table 1.

Table 1. Clinical and demographic characteristics by PTSD diagnosis (N = 40)

<table>
<thead>
<tr>
<th></th>
<th>PTSD (n = 20)</th>
<th>Control (n = 20)</th>
<th>Group Comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sociodemographic characteristics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age M (SD)</td>
<td>37.0 (11.8)</td>
<td>37.1 (12.9)</td>
<td>t (38) = -0.013</td>
</tr>
<tr>
<td>Non-Hispanic White n (%)</td>
<td>17 (85%)</td>
<td>16 (80%)</td>
<td>(\chi^2 = 0.17)</td>
</tr>
<tr>
<td>Education M (SD)</td>
<td>13.5 (2.5)</td>
<td>15.9 (2.0)</td>
<td>t (38) = -3.35**</td>
</tr>
<tr>
<td>Perceived SES</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Community M (SD)</td>
<td>4.95 (2.4)</td>
<td>6.1 (1.5)</td>
<td>t (31.97) = -1.74</td>
</tr>
<tr>
<td>United States M (SD)</td>
<td>4.0 (2.3)</td>
<td>5.7 (1.3)</td>
<td>t (28.71) = -2.91**</td>
</tr>
<tr>
<td>Married n (%)</td>
<td>5 (25%)</td>
<td>22 (55%)</td>
<td>(\chi^2 = 3.75)</td>
</tr>
<tr>
<td>Medical characteristics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight, pounds M (SD)</td>
<td>152.9 (36.6)</td>
<td>148.9 (31.6)</td>
<td>t (38) = 0.365</td>
</tr>
<tr>
<td>BMI M (SD)</td>
<td>26.7 (6.3)</td>
<td>24.4 (4.4)</td>
<td>t (38) = 1.34</td>
</tr>
<tr>
<td>Smoke n (%)</td>
<td>9 (45%)</td>
<td>1 (5%)</td>
<td>(\chi^2 = 8.53**)</td>
</tr>
<tr>
<td>Psychiatric Medication n (%)</td>
<td>18 (90%)</td>
<td>0 (0%)</td>
<td>(\chi^2 = 32.7**)</td>
</tr>
<tr>
<td>Cardiovascular Medication n</td>
<td>0 (0%)</td>
<td>1 (5%)</td>
<td>(\chi^2 = 1.03)</td>
</tr>
<tr>
<td>Psychological characteristics</td>
<td>BDI total score M (SD)</td>
<td>PSS-SR M (SD)</td>
<td>( t ) (24.94) = 6.45**</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-----------------------</td>
<td>--------------</td>
<td>--------------------------</td>
</tr>
<tr>
<td>Major Depressive Episode</td>
<td>10 (50%)</td>
<td>0 (0%)</td>
<td>( \chi^2 = 13.33** )</td>
</tr>
<tr>
<td>Current Manic/ Hypomanic</td>
<td>1 (5%)</td>
<td>0 (0%)</td>
<td>( \chi^2 = 1.03 )</td>
</tr>
</tbody>
</table>

| Episode                       |                       |              |                          |                          |
| Dysthymic Disorder            | 2 (10%)               | 0 (0%)       | \( \chi^2 = 2.11 \)      |                          |
| Alcohol Abuse                 | 0 (0%)                | 0 (0%)       | \( \chi^2 = \text{n/a} \) |                          |
| Alcohol Dependence            | 0 (0%)                | 0 (0%)       | \( \chi^2 = \text{n/a} \) |                          |
| Substance Abuse               | 0 (0%)                | 0 (0%)       | \( \chi^2 = \text{n/a} \) |                          |
| Substance Dependence          | 1 (5%)                | 0 (0%)       | \( \chi^2 = 1.03 \)      |                          |
| Panic Disorder                | 6 (30%)               | 0 (0%)       | \( \chi^2 = 7.06** \)    |                          |
| Social Phobia                 | 2 (10%)               | 0 (0%)       | \( \chi^2 = 2.11 \)      |                          |
| Specific Phobia               | 2 (10%)               | 0 (0%)       | \( \chi^2 = 2.11 \)      |                          |
| OCD                           | 1 (5%)                | 0 (0%)       | \( \chi^2 = 1.03 \)      |                          |
| PTSD                          | 19 (95%)              | 0 (0%)       | \( \chi^2 = 36.19** \)   |                          |
| Current GAD                   | 1 (5%)                | 0 (0%)       | \( \chi^2 = 1.03 \)      |                          |
| Anorexia Nervosa              | 0 (0%)                | 0 (0%)       | \( \chi^2 = \text{n/a} \) |                          |
| Bulimia Nervosa               | 0 (0%)                | 0 (0%)       | \( \chi^2 = \text{n/a} \) |                          |

\( * p < .05, ** p \leq .01 \)
Abbreviations. PTSD = posttraumatic stress disorder; BMI = body mass index; BDI = Beck Depression Inventory; PSS-SR = PTSD Symptom Scale; OCD = Obsessive Compulsive Disorder; SCID = Structured Clinical Interview for DSM-IV.

Notes. One participant in the PTSD group did not meet full criteria for current PTSD as she was near the end of treatment; however, this one participant had met full SCID criteria for PTSD during her initial intake evaluation. In the investigation by Sack et al. (2004), the PTSD sample included two participants who satisfied criteria for partial PTSD.

The degrees of freedom for all $\chi^2$ analyses are 1 (row), 1 (column).

Trauma History

Participants experienced a variety of traumatic exposure histories, and several experienced more than one. For the control participants, 14 (70%) reported a trauma history. For the sample, by group, the following types of trauma were endorsed: robbery/mugging/hold up (9 PTSD, 2 control); beaten/attacked (15 PTSD, 4 control); unwanted sexual activity by force or threat (PTSD 17, 1 control); death of close friend or family member via accident, homicide or suicide (10 PTSD, 8 control); injury or property damage due to fire (4 PTSD, 2 control); injury or property damage due to severe weather, natural disaster or human-made disaster (5 PTSD, 2 control); involved in a motor vehicle accident serious enough to cause injury (8 PTSD; 6 control); witnessed someone seriously injured or killed (10 PTSD, 2 control); serious physical injury as a result of non-motor vehicle related accident (6 PTSD, 3 control); and other terrifying or shocking experience (9 PTSD, 6 control).
Baseline Analyses

Mean (SD) baseline values of SBP, DBP, HR, MAP, respiration rate, LF-HRV, HF-HRV, and LF/HF ratio for the PTSD and control groups are presented in Table 2. Results of the series of independent samples \( t \) tests on each of the baseline physiological indices indicated that there was no significant effect of group membership (all \( p \) values <.05). Respiration rate is sometimes useful in the interpretation of HF-HRV because respiratory parameters (e.g., rate and depth) can confound the relationship between HF-HRV and cardiac vagal tone (Grossman et al., 2004; Grossman & Taylor, 2007). However, since respiration rate did not differ between groups at baseline and was not significantly correlated with HF-HRV during any phase of the laboratory protocol, no further results regarding respiration rate are reported.
Table 2. *Mean Values (SD) of Cardiovascular Measures at Each Phase by Diagnostic Group (N= 40)*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Trauma Recall</th>
<th>Recovery 1</th>
<th>Mental Arithmetic</th>
<th>Recovery 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SBP</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>111.6 (20.3)</td>
<td>124.9 (22.4)</td>
<td>116.6 (19.7)</td>
<td>117.9 (19.6)</td>
<td>112.3 (18.3)</td>
</tr>
<tr>
<td>Control</td>
<td>106.4 (11.7)</td>
<td>119.2 (14.4)</td>
<td>108.9 (12.9)</td>
<td>115.0 (13.0)</td>
<td>106.5 (10.4)</td>
</tr>
<tr>
<td><strong>DBP</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>67.6 (14.2)</td>
<td>77.3 (14.7)</td>
<td>70.8 (13.8)</td>
<td>72.6 (13.7)</td>
<td>67.4 (15.0)</td>
</tr>
<tr>
<td>Control</td>
<td>66.6 (10.7)</td>
<td>73.9 (10.8)</td>
<td>66.1 (8.2)</td>
<td>72.0 (9.8)</td>
<td>64.1 (8.0)</td>
</tr>
<tr>
<td><strong>Heart rate</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(beats/min)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>73.5 (12.6)</td>
<td>81.7 (11.5)</td>
<td>73.6 (12.7)</td>
<td>78.3 (11.5)</td>
<td>74.0 (12.3)</td>
</tr>
<tr>
<td>Control</td>
<td>70.1 (12.9)</td>
<td>77.6 (13.4)</td>
<td>71.0 (13.3)</td>
<td>75.6 (13.0)</td>
<td>72.0 (11.4)</td>
</tr>
<tr>
<td><strong>MAP</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>84.6 (17.2)</td>
<td>95.4 (17.8)</td>
<td>88.3 (16.2)</td>
<td>90.1 (16.2)</td>
<td>85.4 (16.1)</td>
</tr>
<tr>
<td>Control</td>
<td>81.1 (11.0)</td>
<td>92.0 (12.5)</td>
<td>82.5 (10.2)</td>
<td>88.4 (11.3)</td>
<td>80.6 (9.1)</td>
</tr>
<tr>
<td><strong>LF-HRV (ln)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>3.8 (0.5)</td>
<td>4.3 (0.2)*</td>
<td>3.9 (0.3)</td>
<td>4.1 (0.3)</td>
<td>3.9 (0.4)</td>
</tr>
<tr>
<td>Control</td>
<td>3.8 (0.6)</td>
<td>4.1 (0.3)</td>
<td>4.0 (0.4)</td>
<td>4.0 (0.4)</td>
<td>3.7 (0.6)</td>
</tr>
<tr>
<td><strong>HF-HRV (ln)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>3.8 (0.4)</td>
<td>2.9 (0.7)</td>
<td>3.6 (0.6)</td>
<td>3.1 (0.4)</td>
<td>3.6 (0.5)</td>
</tr>
<tr>
<td>Control</td>
<td>3.6 (0.5)</td>
<td>3.3 (0.6)</td>
<td>3.4 (0.7)</td>
<td>3.4 (0.5)</td>
<td>3.6 (0.7)</td>
</tr>
<tr>
<td><strong>LF/HF ratio</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>1.4 (1.3)</td>
<td>6.4 (10.4)</td>
<td>2.1 (2.3)</td>
<td>3.4 (2.0)</td>
<td>1.8 (1.4)</td>
</tr>
<tr>
<td>Control</td>
<td>1.9 (1.6)</td>
<td>3.1 (2.7)</td>
<td>2.8 (3.1)</td>
<td>2.6 (1.9)</td>
<td>2.2 (2.4)</td>
</tr>
</tbody>
</table>

*Notes.* HR results presented here are based on analyses of interbeat interval (IBI). HRV results are expressed in normalized units.
SBP = systolic blood pressure; DBP = diastolic blood pressure; PTSD = posttraumatic stress disorder; MAP = mean arterial pressure; LF-HRV = low frequency heart rate variability; HF-HRV = high frequency heart rate variability.

* $p < .05$

**Primary Analyses**

Results of a series of 2 (PTSD vs. Control) x 2 (task order) x 5 (phases of the protocol) mixed model repeated measures ANOVAs indicated that there were no main effects for task order on any of the physiological indices. Thus, task order was not included in any of the subsequent analyses.

**Phase Analyses**

A series of (2 PTSD presence/absence) x 5 (phases) ANOVAs revealed significant effects of phase on each physiological variable (all $F$ values (4,152) > 4.6, $p$s < .05). Mean (SD) task and recovery values of SBP, DBP, HR, MAP, LF-HRV, HF-HRV, and LF/HF ratio for the PTSD and control groups are presented in Table 2.

Subsequent inspection of the means and planned contrasts indicated that SBP increased from baseline levels during both the trauma recall and mental arithmetic speech tasks ($p$s < .05) and returned to near baseline levels (i.e., did not differ from baseline, $p$ > .05) following the mental arithmetic task. Although SBP also decreased during the recovery period following the trauma recall task, it did not return to near baseline levels.
DBP increased from baseline levels during both the trauma recall and mental arithmetic speech tasks ($p < .05$) and returned to near baseline levels during both recovery periods.

HR increased from baseline levels during both the trauma recall and mental arithmetic speech tasks ($p < .05$) and decreased significantly to baseline levels following the recovery period following the trauma recall task. Although HR also significantly decreased during the recovery period following the mental arithmetic task ($p < .05$), it did not return to near baseline levels.

MAP increased from baseline levels during both the trauma recall and mental arithmetic stress tasks ($p < .05$) and returned to near baseline levels during the recovery period following the mental arithmetic task. Although MAP decreased during the recovery period following the trauma recall task ($p < .05$), it did not return to near baseline levels.

LF-HRV increased from baseline levels during the trauma recall task ($p < .05$). Although LF-HRV decreased during the recovery period following the trauma recall ($p < .05$), it did not return to near baseline levels. LF-HRV demonstrated a trend toward significantly increasing during the mental arithmetic task ($p = .06$), and decreased to near baseline levels during the recovery period following the mental arithmetic task ($p < .05$). Mean LF-HRV values throughout the five phases of the laboratory protocol are presented in Figure 1.

*Figure 1.* Mean LF-HRV values throughout the laboratory protocol by group.
HF-HRV decreased from baseline levels during both the trauma recall and mental arithmetic speech tasks and significantly increased to near baseline levels during the recovery period following the mental arithmetic task ($p < .05$). Although HF-HRV increased during the recovery period following the trauma recall task ($p < .05$), it did not return to near baseline levels. Importantly, there was a significant group by phase interaction for HF-HRV, Greenhouse-Geisser $\varepsilon (3.15, 119.54) = 2.97, p = .03$. Participants with PTSD had significantly greater reductions in HF-HRV during the speech tasks (trauma recall: $M = -0.84$, $SD = 0.71$; mental arithmetic: $M = -0.64$, $SD = 0.56$) than controls (trauma recall: $M = -0.33$, $SD = 0.53$; mental arithmetic: $M = -0.25$, $SD = 0.41$).
Mean HF-HRV values throughout the five phases of the laboratory protocol are presented in Figure 2.

*Figure 2. Mean HF-HRV values throughout the laboratory protocol by group.*

LF/HF ratio increased from baseline levels during the trauma recall task ($p < .05$), and returned to near baseline levels during the recovery period following the mental arithmetic task. LF/HF ratio demonstrated a trend toward significantly decreasing to near baseline levels during the recovery period following the trauma recall task ($p = .06$). LF/HF ratio did not significantly change during the mental arithmetic task from the recovery period following the trauma recall task. However, LF/HF ratio decreased to near
baseline levels during the recovery period following the mental arithmetic task ($p < .05$). Mean LF/HF ratios throughout the five phases of the laboratory protocol are presented in Figure 3.

**Figure 3.** Mean LF/HF ratios throughout the laboratory protocol by group.

![Graph showing Mean LF/HF ratios](image)

*Reactivity Analyses*

The change score analyses involving HF-HRV revealed a significant main effect for group, $F (1, 38)= 9.12, p < .01$. Subsequent inspection of the means indicated that the PTSD group had greater decreases in HF-HRV during both the trauma recall ($M = -0.84$, $SD= 0.71$) and mental arithmetic ($M = -0.64$, $SD= 0.56$), compared to the control group.
(M = -0.33, SD = 0.54 and M = -0.25, SD = 0.41, respectively). Changes in HF-HRV from baseline to each speech task according to group are shown in Figure 4.

*Figure 4.* Changes in HF-HRV from baseline to each speech task by group.
DISCUSSION

Summary

Results of the present study may be summarized in three statements. First, participants with PTSD did not exhibit significant lower parasympathetic activity relative to controls at rest. Second, both groups of participants demonstrated decreases in PNS activity as a function of the two stress tasks. Third, participants with PTSD evidenced larger decreases in parasympathetic activity during both tasks as compared to control participants. Each of these findings will be discussed in turn below.

Baseline Parasympathetic Functioning

Contrary to the first hypothesis, women with PTSD in the present study did not exhibit lower HF-HRV relative to age-matched controls during a resting baseline period. This result is consistent with the study by Sahar et al. (2001), which found no differences in baseline HF-HR when comparing 14 male PTSD patients and 15 trauma-exposed controls. The results of the present study differ from other investigations of resting HF-HRV among individuals with PTSD (Blechert et al., 2007; Cohen et al., 1997; Cohen et al., 1998; Cohen et al., 2000). As discussed previously, the sample of control participants in the Cohen et al. (1997; 1998) investigations had a low resting HR, and the observed difference in basal HF-HRV may have been the consequence of atypically high PNS tone among the control participants, rather than low PNS tone among the PTSD participants.
(Sahar et al., 2001). That is, the results of these studies may be sensitive to sampling and the HR obtained in each group. This study did not find a HR difference, which is consistent with not finding a HF-HRV difference between PTSD and control participants. In order to conclude that PTSD is associated with elevated HR, a meta-analysis was necessary (Buckley & Kaloupek, 2001). Given the large contribution of the PNS to resting HR, it is likely that findings for resting HF-HRV in any single study will parallel the findings for resting HR. Therefore, larger samples and eventual meta-analyses may be required before PTSD-related alterations in resting HF-HRV can be conclusively demonstrated.

The results of the present study are not entirely inconsistent with the investigations employing BRS by Hughes et al. (2006b; 2007). BRS is a better index of absolute levels of PNS activity (i.e., PNS tone) than HF-HRV analysis (Reyes del Paso et al., 1996). Thus, in theory, it is possible to obtain lower baseline BRS values among individual with PTSD and equivalent HF-HRV values. However, data are not available from this investigation to test this hypothesis, and future research using both measures would be necessary.

Parasympathetic Functioning During Stress

Consistent with the second hypothesis, all participants evidenced significant decreases in PNS activity as a function of the two stressogenic speech tasks. This observation is consistent with previous research analyzing the effects of pharmacological autonomic blockades in healthy women, which demonstrated a pattern of decreased
parasympathetic control of the heart during speech stress and mental arithmetic (Berntson et al., 1994). The results of the present study are also consistent with the investigation by Sahar et al. (Sahar et al., 2001), which found that participants with PTSD demonstrated larger decreases in PNS activity during a mental arithmetic task compared to controls. The results of the present study are not in agreement with two previous investigations, which did not obtain significant changes in HF-HRV in the PTSD group during much longer speech tasks (Cohen et al., 1998; Cohen et al., 2000).

Consistent with the third hypothesis, participants with PTSD evidenced larger decreases in parasympathetic activity during both speech tasks as compared to controls. These results suggest that individuals with PTSD experience altered parasympathetic responses to psychological stressors, regardless of whether the stressors are trauma-relevant or trauma-neutral. Although the present findings are inconsistent with two previous studies that employed a trauma recall task (Cohen et al., 1998; Cohen et al., 2000), the duration of the speech task in the present study was much shorter in duration (i.e., four minutes). As discussed previously, a long verbal narrative (i.e., 15-20 minutes) may not be well-suited for eliciting and evaluating cardiovascular responses to psychological stressors.

The results of this study are also inconsistent with the speech task reactivity findings of the investigation by Hughes et al. (2007), which indicated that the magnitude of the decrease in PNS activity (as measured by BRS levels) was smaller during the anger recall task for psychiatric patients (the PTSD without MDD, PTSD with MDD, and MDD without PTSD groups collapsed into a single group) than controls. However, two
methodological issues may account for the discrepant findings. First, the smaller
decreases in BRS during the anger recall task among the combined psychiatric group of
the Hughes et al. study may be due to the presence of a floor effect of baseline BRS
levels among the psychiatric groups.

Limitations

Limitations of the proposed study should be noted. Because the study was cross-
sectional in design, mechanisms for the observed association between PTSD and
dysregulated parasympathetic nervous system functioning cannot be specified based on
these data. It is plausible that PTSD results in attenuated HF-HRV through a
physiological effect of PTSD on the PNS. At the same time, however, it is also possible
that reduced HF-HRV is a risk factor for developing PTSD, and that group differences in
HF-HRV were pre-existing. Given that previous research has found that HR response in
the immediate aftermath of a traumatic event is predictive of subsequent development of
PTSD (Bryant et al., 2000; Bryant et al., 2003; Shalev et al., 1998), women with
diminished parasympathetic control may be at greater risk of developing PTSD.

The present study examined a group of females with PTSD, thus limiting the
generalizability of these findings to women. Women with PTSD were selected as the
focus of this study because the majority of research examining PTSD and physical health
has been conducted in male samples (Beckham et al., 2002). The present study did not
exclude PTSD patients who were taking psychiatric medications or those who with
comorbid psychiatric diagnoses; these non-restrictive criteria may increase the generalizability of the current findings.

Participants with PTSD were significantly more likely to currently smoke and use psychiatric medications than controls. HRV is influenced by many factors, including both medications and smoking status (Cohen et al., 1997). For example, treatment with non-tricyclic antidepressant medications leading to improvement in depressive symptoms has been associated with increases in HRV (Balogh et al., 1993; Glassman et al., 2007). In addition, smoking alters sympathovagal balance in favor of sympathetic predominance and is associated with decreased HRV in healthy subjects (Alyan et al., 2008; Barutcu et al., 2005). Although current smoking status and psychiatric medication use represent potential confounds to the present results, the study was not powered to assess group differences for these sample characteristics. Furthermore, these patients were sampled from an outpatient treatment facility and may be more representative of PTSD patients than unmedicated samples.

**Future Directions**

It is also possible that the results of the present study are characteristic of mental disorders in general, and are not specific to PTSD. Altered parasympathetic activity has been observed in several mental health disorders, including depression (Hughes & Stoney, 2000; Udupa et al., 2007), panic disorder (Yeragani et al., 1993), generalized anxiety disorder (Thayer 1996), schizophrenia (Zahn & Pickar, 1993), and bipolar disorder (Cohen et al., 2003). Only a handful of previous investigations have
simultaneously compared more than one diagnostic group (Blechert et al., 2007; Cohen et al., 2000; Hughes et al., 2007), and more are needed. With an exception of the investigation by Hughes et al. (2007), both the current and previous studies of PNS functioning among PTSD patients have examined samples that did not allow for the specificity of the effects of depression or anxiety to be evaluated. Thus, future investigations may wish to simultaneously compare more specific diagnostic groups, such as PTSD with comorbid Major Depressive Disorder (MDD), PTSD without comorbid Major Depressive Disorder (MDD), MDD without PTSD, and controls without either of these psychiatric diagnoses.

Participants in the present sample reported a variety of types of trauma exposure history. The relatively small sample size in the present study does not afford enough statistical power to examine whether there are differences in physiological responses according to type of trauma exposure. Future investigations may wish to examine the potential differences in physiological responses according to type and duration of trauma exposure in larger samples.

No known studies have simultaneously measured parasympathetic activity with both HF-HRV and BRS. In addition, there are no known studies employing HR recovery analysis when comparing PTSD patients and healthy controls. Thus, future studies should consider the simultaneous comparison of HF-HRV, BRS, and HR recovery indices when examining the activity of the PNS among individuals with PTSD.

Conclusions
In summary, the present study found that all participants demonstrated decreases in PNS activity as a function of two stressogenic speech tasks, and that participants with PTSD demonstrated larger reductions in HF-HRV during both speech tasks compared to control participants. This is interpreted to indicate a larger decline in parasympathetic cardiac control during stress, despite apparently similar levels of parasympathetic activity at rest. Such findings suggest that PTSD is related to the magnitude of decrease in parasympathetic cardiac control during stress in women.
REFERENCES

Reference List


Song Selections

1. String Quartet #1—Adante (Tchaikovsky) 6:38
2. The Girl with the Flaxen Hair (Debussy) 2:14
3. Fugue in C Minor (Bach) 2:52
4. The Planets—Venus (Holst) 8:03
5. Serenade for String (Antonin Dvořák) 7:71
6. String Quartet #2 (Alexander Borodin) 7:44
7. The Four Seasons (Antonio Vivaldi) 2:26