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Exploring the Relationship Between Posttraumatic Stress Disorder Symptoms and Momentary Heart Rate Variability

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Abstract

Objective—Exposure to trauma-related cues has been associated with a prolonged decrease in heart rate variability (HRV) under laboratory conditions, however the relationship between PTSD symptoms and HRV has not been evaluated during everyday life. The present study sought to determine whether Posttraumatic Stress Disorder (PTSD) symptoms reported during everyday life were related to reduced HRV.

Methodology—Eighty-three young adults with PTSD underwent 24-hour Holter monitoring, during which PTSD symptoms were measured using ecological momentary assessment (EMA). Multilevel modeling was used to examine the association between PTSD symptom severity and low frequency (LF) and high frequency (HF) HRV.

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Conflict of Interest

We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further confirm that the order of authors listed in the manuscript has been approved by all of us.

We confirm that we have given due consideration to the protection of intellectual property associated with this work and that there are no impediments to publication, including the timing of publication, with respect to intellectual property. In so doing we confirm that we have followed the regulations of our institutions concerning intellectual property.

We further confirm that any aspect of the work covered in this manuscript that has involved either experimental animals or human patients has been conducted with the ethical approval of all relevant bodies and that such approvals are acknowledged within the manuscript.

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Results—PTSD symptoms were associated with reductions in LF HRV, independently of age and activity level. There was no significant association between PTSD symptom levels and HF HRV.

Conclusions—These results indicate that an association between momentary PTSD symptom severity and reduced LF HRV is significant and observable in young adults with PTSD. Findings highlight the need for cardiovascular screening in young adults with PTSD and early interventions that target physiological reactivity in PTSD.

Keywords

Heart Rate Variability; Posttraumatic Stress Disorder

Introduction

Posttraumatic Stress Disorder (PTSD) may be a risk factor for reduced heart rate variability (HRV). Studies of trauma-exposed patients have found that PTSD is associated with reduced parasympathetic nervous system (PSNS) control measured from both vagally mediated baroreflex sensitivity (Hughes et al, 2006; Hughes et al., 2007) and HRV (Tan et al., 2011; Minassian et al., 2014; Lakusic et al. 2007; Mellman et al. 2004).

Defined as a measure of beat-to-beat variation in heart rate, HRV is a clinically accepted index of autonomic nervous system (ANS) activity. HRV captures heart rate regulation by both the sympathetic (SNS) and PSNS divisions of the autonomic nervous system (ANS) and is one psychophysiological manifestation of stress on the human body. As a widely used marker of cardiac autonomic function, HRV is reduced in several chronic conditions such as coronary heart disease and myocardial ischemia (Dekker et al., 2000; Goldkorn et al., 2015). Both low-frequency (LF) and high-frequency (HF) components of HRV have been associated with increased cardiovascular disease (CVD) risk and new cardiac events in diabetic and CVD sufferers (Hadase et al., 2004; Liao et al., 2002; Gerritsen et al. 2001).

Although PTSD has been associated with reduced HRV (Dennis et al., 2014; Shah, 2013), it remains unclear whether PTSD is directly related to reduced PSNS through PTSD symptomatology (e.g., hyperarousal) or *via* an indirect effect of lifestyle factors (e.g., smoking; Dennis et al., 2014). Findings that PTSD is associated with an exaggerated withdrawal of PSNS activity and an excessive SNS response to trauma cues suggest that the experience of PTSD's core symptoms may directly underlie ANS dysregulation (Norte et al., 2013; Southwick et al, 1999; Kosten et al., 1987; Blanchard et al, 1991; Liberzon et al, 1999).

We sought to evaluate whether momentary PTSD symptoms were associated with reductions in ambulatory HRV in 83 young adults with current PTSD. We hypothesized that momentary PTSD symptoms experienced during daily life would be associated with reduced minute-to-minute HF and LF HRV.

Methodology

Participants

Data were from a larger study on trauma and health (Dennis et al., 2014). Exclusion criteria included age <18 or >39 years, the presence of an organic mental disorder, schizophrenia, bipolar I (mixed) and II, lifetime PTSD without current PTSD, current substance abuse/dependence, current major depressive disorder without PTSD, pregnancy, HIV/AIDS, and an uncontrolled condition. The current study was limited to participants diagnosed with PTSD ($N = 86$). Three participants using beta or calcium-channel blockers were removed owing to potential effects on HRV ($N=83$). IRB approval and informed consent were obtained. Data collection occurred over three or four visits, between 08/2008 and 07/2013.

Procedures

At the initial visit, PTSD status was assessed using a clinical structured interview, the Clinician Administered PTSD Scale (Blake et al., 1995). PTSD severity was measured using the Davidson Trauma Scale (Davidson et al., 1997) on a scale of 0, reflecting no symptoms, to 72, reflecting severe symptoms. Sleep disturbance was assessed using the Pittsburgh Sleep Quality Inventory (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Participants also reported their smoking status.

At a subsequent visit, participants were fitted with a 24-hour digital Holter monitor (Lifecard CF, Del Mar Reynolds, Irvine, CA) and provided a personal digital assistant for ecological momentary assessments (EMA) of PTSD symptoms in response to random alarms. Monitoring sessions began at 2:00 PM and lasted 24 hours. Four diary items captured the moment of DSM-IV PTSD symptom severity from 0 (“not at all”) to 4 (“extremely”). Response items were: disturbing memories, thoughts, or images of the trauma; feeling distant, emotionally numb, or cut off from other people; avoiding thoughts/activities of the trauma; and difficulty concentrating, feeling jumpy/easily startled, overly alert, or feeling irritable/angry. A total PTSD symptom score was derived from the mean of the four items. Participants recorded their current activity level: lying down, sitting, standing up, engaging in light activity (e.g., walking slowly) or heavy activity (e.g., running).

Minute-by-minute changes in LF and HF HRV were assessed by complex demodulation, a nonlinear time-domain method for assessing time-dependent changes in nonstationary oscillatory components within a predefined frequency band (Kitney, 1980). This method has been reported previously (Hayano et al, 1993). Briefly, the time-dependent changes in LF and HF amplitudes were extracted continuously by demodulating the frequency bands of 0.04–0.15 Hz and 0.15–0.45 Hz, respectively, and the amplitude time series of LF and HF components were averaged over every 1-minute segment. The LF component is controlled by both branches of the autonomic nervous system and reflects baroreflex function (Rahman, 2011), while the HF component is associated exclusively with parasympathetic responses and is closely linked to respiratory influences. To evaluate the association between EMA-based PTSD symptom report and HRV, we modeled LF and HF recorded within a 60-second interval following each EMA entry.

Analyses

To determine whether momentary PTSD symptoms was associated with HRV recorded during the corresponding minute of the diary entry, multilevel modeling was employed. Two separate models were specified for LF and HF amplitude. Grand-mean standardized PTSD symptom-severity scores were generated by calculating each individual's mean PTSD symptom level across the observation period and *z*-scoring these in relation to other participants. Individual-mean standardized scores were then calculated by using each individual's mean PTSD symptom level and corresponding SD to *z*-score PTSD symptom levels recorded at each reading. Both the grand-mean and individual-mean standardized scores were entered as predictors of HRV. Analyses controlled for age, smoking status, sleep disturbance, and activity level.

Results

Participants underwent EMA and HRV monitoring ($M=18.11$ hours; $SD = 4.84$) and recorded diary entries ($M=6.87$; range: 1–16) with a random alarm compliance rate of 83%. Characteristics and intercorrelations between variables and individual means are reported in Table 1.

Analyses of null models indicated that 49% of the variance in LF amplitude and 58% of the variance in HF amplitude was attributable to inter-individual differences as opposed to intra-individual variability in momentary PTSD symptoms. Results involving the full models are shown in Table 2. Within-person changes in PTSD symptom level were associated with LF amplitude but not HF amplitude. Between-person differences in PTSD symptom level were not associated with either measure of HRV.

Discussion

This study examined the relation between momentary PTSD symptoms and HRV under ambulatory conditions. As hypothesized, momentary PTSD symptom severity was found to be negatively associated with minute-to-minute HRV, albeit just LF amplitude. This constitutes the first real time examination of the effect of PTSD symptoms on HRV. Findings highlight the effect of PTSD symptoms on heart rate, even during young adulthood when few cardiovascular problems are present.

The finding that greater PTSD symptom severity is related to lower LF amplitude is consistent with previous observations that negative affect and perceived stress are temporally associated with a reduction in the LF component of HRV during experimental stress in young healthy individuals (Swenne, Bootsma and vanBolhuis, 1995) together with ambulatory monitoring in coronary heart disease patients (Bacon et al, 2004). This direction of change supports previous findings that the LF component of HRV is not simply a marker of SNS modulation but rather is under dual control by the sympathetic and parasympathetic nervous systems (Goldstein, et al, 2011; Houle et al, 1999). Under resting conditions, studies have demonstrated that the oscillations in HRV across the LF band are the result of baroreflex-mediated adjustments in heart rate (Cerutti et al, 1994; Moak et al, 2007). Acute stress is associated with withdrawal of parasympathetic cardiac control which manifested as

a reduction in LF amplitude at the time of PTSD symptoms. Although this reduction in parasympathetic control during stress is typically also observed at the HF band (Wahbeh & Oken, 2013; Minassian et al., 2014), we failed to observe a significant association between PTSD symptoms and reduced HF amplitude. We attribute this possibly to research methodology differences (e.g., short-term HRV assessment). Although Shah and colleagues (2013) assessed 24-hour HRV in their study of combat male veteran twins (n=459) on autonomic modulation, PTSD symptom severity was only assessed at a single time point.

The sample was comprised of only PTSD individuals; thus results extend previous findings comparing PTSD and non-PTSD individuals (Dennis et al., 2014) to within group HRV variability on PTSD symptom severity. The momentary assessment method and the within-group severity analysis provides further evidence that PTSD symptoms are significantly associated with HRV. There are study limitations. Given the small sample size, results should be replicated. Furthermore, findings may not generalize beyond the age range of the sample.

In summary, PTSD is a deleterious psychiatric condition associated with increased morbidity and mortality (Schnurr & Jankowski, 1999; Schnurr et al., 2000). This study increases the evidence (through ambulatory and momentary monitoring with a younger sample) that one mechanism possibly contributing to cardiovascular illness among those with PTSD is that PTSD is associated with the disruption of the autonomic processes that maintain heartbeat regulation. These findings raise the possibility that early intervention among younger PTSD individuals may reduce the risk for adverse cardiovascular outcomes. Future studies should explore interventions that target the reduction of physiological arousal in PTSD.

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Highlights

- PTSD and HRV were assessed in a young adult sample under everyday conditions.
- Multilevel modeling demonstrated a relation between PTSD and low frequency HRV.
- Heart rate regulation is a possible mechanism of poorer cardiovascular function in PTSD

Table 1

Participant Characteristics and Correlations (N = 83)

Variables	Mean (SD)/Freq (%)	1	2	3	4	5	6	7	8	9
Age (1)	30.19 (5.44)	-								
Male (2)	36 (43%)	.05	-							
Minority (3)	48 (58%)	.15	.17	-						
Current/Former Smoker (4)	51 (61%)	.03	-.17	.12	-					
Sleep Disturbance (5)	9.45 (3.48)	.02	-.27*	-.14	.27*	-				
DTS Total (6)	69.38 (31.76)	.09	-.08	.08	.40***	.44***	-			
Mean EMA PTSD Sx (7)	0.93 (0.73)	.09	-.14	.19 [†]	.28***	.43***	.60***	-		
Mean EMA LF Amp (8)	35.12 (15.13)	-.40**	-.18	-.28*	-.09	-.16	-.25*	-.25*	-	
Mean EMA HF Amp (9)	22.33 (15.13)	-.40**	.04	-.07	-.14	-.15	-.21 [†]	-.17	.78**	-
Mean EMA Heart Rate	86.10 (12.04)	.10	.02	.14	.15	.17	.27*	.11	-.67***	-.72***

Note. Mean EMA PTSD symptoms, HRV, and heart rate refer to individual means. Intercorrelations between sex (male), minority status, and smoking status (current/former smoker) are tetrachoric. DTS = Davidson Trauma Scale; Sx = Symptoms; EMA = ecological momentary assessment; LF Amp = low-frequency amplitude; HF Amp = high-frequency amplitude.

[†] $p < .10$,

* $p < .05$,

*** $p < .01$

Table 2

Multilevel models of LF and HF Amplitude

Parameter	LF Amplitude	HF Amplitude
Within-Person		
Intercept	64.77** (10.25)	51.82** (8.91)
Sitting ^a	5.48 (3.54)	1.71 (2.75)
Light activity (standing up) ^a	5.57 (3.63)	4.07 (2.68)
Heavy activity (standing up) ^a	10.99** (3.76)	8.46** (2.84)
EMA PTSD symptoms	-1.23* (0.61)	-0.40 (0.46)
Between-Person		
Age	-1.05** (0.27)	-0.93** (0.24)
Current/Former Smoker	-0.29 (3.23)	-2.26 (2.84)
Sleep Disturbance	-0.30 (0.48)	-0.35 (0.43)
EMA PTSD symptoms	-2.73 (1.67)	-0.94 (1.47)

Note. Model coefficients and standard errors (in parentheses).

^aProstrate position used as reference value.

*
 $p < .05$,

**
 $p < .01$