

Original quantitative research

Cardiac function and posttraumatic stress disorder: a review of the literature and case report

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Abstract

Introduction: Posttraumatic stress disorder (PTSD) can induce an elevation in sympathetic tone; however, research pertaining to the cardiac cycle in patients with PTSD is limited.

Methods: A literature review was conducted with PubMed, MEDLINE and Web of Science. Articles discussing changes and associations in echocardiography and PTSD or related symptoms were synthesized for the current review. We have also included data from a case report of a male participant aged 33 years experiencing potentially psychologically traumatic events, who wore a noninvasive cardiac sensor to assess the timing intervals and contractility parameters of the cardiac cycle using seismocardiography. The intervals included systolic time, isovolumic contraction time (IVCT) and isovolumic relaxation time (IVRT). Calculations of systolic (IVCT/systole), diastolic (IVRT/systole) and myocardial [(IVCT + IVRT)/systole] performance indices were completed.

Results: The review identified 55 articles, 14 of which assessed cardiac function using echocardiography in patients with PTSD symptoms. Cardiac dysfunction varied across studies, with diastolic and systolic impairments found in patients with PTSD. Our case study showed that occupational stress elevated cardiac performance indices, suggesting increased ventricular stress and supporting results in the existing literature.

Conclusion: The literature review results suggest that a controlled approach to assessing cardiac function in patients with PTSD is required. The case study results further suggest that acute bouts of stress can alter cardiac function, with potential for sustained occupational stress to induce changes in cardiac function. Cardiac monitoring can be used prospectively to identify changes induced by potentially psychologically traumatic event exposures that can lead to the development of PTSD symptoms.

Keywords: *echocardiography, seismocardiography, PTSD, case study, systole, diastole, myocardial performance indices, autonomic nervous system*

Introduction

Research relating to cardiovascular function associated with posttraumatic stress disorder (PTSD) and other posttraumatic stress injuries remains very limited. Changes in autonomic nervous system function due to PTSD have been documented extensively, and most of the associated meta-analyses have focussed on heart rate variability (HRV).^{1,2} In patients

with PTSD, one meta-analysis evidenced that the root mean square of successive differences, as well as high frequency (HF) and low frequency (LF) parameters, were decreased during baseline rest, with HF also decreased during acute stress.³ The meta-analytic results also showed the LF/HF ratio was increased during rest in people with PTSD, indicating a reduction in parasympathetic tone.³

Highlights

- Research on cardiac cycle timing intervals and posttraumatic stress disorder (PTSD) is very limited.
- Systolic and diastolic dysfunction is documented in PTSD; however, there is much variation in the literature on cardiac function in PTSD.
- Potentially psychologically traumatic events (PPTE) can increase myocardial performance index, in part due to shortened systolic time.
- PPTE can result in elevated cardiac stress, and this can be easily and noninvasively monitored using seismocardiography.

HRV can be a useful indicator of emotional and physical stress;⁴ however, as HRV reflects the regulation of autonomic balance,⁵ the mechanism by which cardiac function is altered cannot fully be answered by HRV alone. Understanding how PTSD interacts with cardiac function (particularly right and left ventricular function and cardiac cycle timing intervals) may provide useful prospective diagnostic tools. Assessing cardiac function is usually done with echocardiography, a commonly used method for assessing cardiac dysfunction.⁶ A study with 28 female participants, 14 of whom had PTSD and 14 of whom did not, demonstrated significantly impaired end-diastolic pressure and left ventricular suction power in the PTSD group.⁷ The suction power of the heart is usually measured as the velocity of propagation in early diastole, and when combined with impaired end-diastolic pressure, the measurements indicate left ventricular

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diastolic dysfunction which can be caused by emotional stress.⁷

Cardiac disease-induced PTSD has been studied, with much of the research focusing on PTSD following an acute cardiac event such as a myocardial infarction.⁸ However, clinical studies utilizing echocardiography for cardiac assessment to document changes in cardiac cycle parameters as a primary measurement in occupational and stressful environments are very limited. Potential research barriers to using cardiac magnetic resonance imaging and echocardiography include the cost, time and access to technicians.⁹ There are alternative methods for analyzing cardiac function and cardiac cycle timing intervals using ballistocardiography (BCG) or seismocardiography (SCG).¹⁰⁻¹⁴ Analyses using BCG or SCG allow for identifying cardiac cycle parameters on every heartbeat during data collection and are relatively cost-effective.¹⁵ SCG has been correlated with stroke volume¹⁶ and total systolic time and pre-ejection period,¹⁷ and has been used to document changes in cardiac function related to long and acute COVID-19,^{15,18} acute sport-related concussion¹⁹ and hypertrophic cardiomyopathy.²⁰ Furthermore, normative data on the cardiac cycle using the SCG have also been published.²¹

The cardiac cycle timing intervals are known to change with age and sex. There is an increase in isovolumic relaxation time (IVRT) with age, which in turn leads to an increase in myocardial performance index (MPI) as well. MPI tends to increase from 0.38 to 0.53, and IVRT from 78 ms to 109 ms, for individuals aged 20 to 39 and 60 or above, respectively.²² There are also differences between males and females for systolic time and MPI as well.²² Cardiac remodelling and the demographics of the individuals can also alter these timing intervals as per the requirements of the sport. Specifically, relative to the normative data for males and females, athletes in their early twenties exhibit different mean values at an IVRT of 82 ms and 97 ms, and an MPI of 0.41 and 0.46, respectively.²¹

This study was designed to present a brief review of the literature on PTSD and cardiac function to provide insight on the importance of monitoring cardiac performance that could be applied to stressful occupational settings, such as those of public safety personnel (e.g. border services

personnel, correctional workers, firefighters, paramedics, police, public safety communicators).²³ We provide a proof-of-concept case report of changes in cardiac function measured by SCG technology following a series of acute occupational stressors.

Methods

Literature review

We conducted a database search using the keywords [(echocardiography) OR (ejection fraction) OR (left ventricular) OR (right ventricular)) AND ((PTSD) OR (post traumatic stress disorder))] in PubMed, MEDLINE and Web of Science. The date range was from database inception to 30 April 2022. Given the limited number of studies, all studies that included echocardiography parameters were included for our review. The review is meant to be narrative in nature and to summarize the available literature. The search was completed by one author (JS), and a reference review of the included articles was also conducted to find articles that fit the criteria.

Case report

Ethics approval

This study was approved by the University of Regina Research Ethics Board (REB# 2020-073) and the participant signed an informed consent form.

Participant and equipment

The participant, a rehabilitation physiotherapist in a hospital environment, completed data collection during a baseline period. The participant continued to collect daily cardiac data using a noninvasive cardiac sensor (LLA Recordis, LLA Technologies, Langley, BC, CA) to record the vibrations of the sternum resulting from the heart contraction.¹⁵ The cardiac sensor was snapped into a single adhesive electrocardiogram (ECG) gel electrode and placed one centimetre above the xiphoid process on the sternum of the chest directly over the skin. The cardiac data were collected for one minute while the participant was in a supine resting position. The participant collected data once per day for one month.

Data analyses

The recorded data were analyzed offline. The data analysis details have been previously reported.¹⁵ Briefly, a first order Butterworth bandpass filter with a low

cut-off frequency of 1 Hz and high cut-off frequency of 30 Hz was applied to the signal. Analysis of fiducial points of the cardiac cycle was completed using an independent, proprietary algorithm (LLA Technologies Inc). The fiducial points included the mitral valve closure (MVC), aortic valve opening (AVO), aortic twist (ATT), aortic systole, rapid ejection period (REP), aortic valve closure (AVC), ventricular untwisting, mitral valve opening (MVO), atrial systole (AS) and contractile twist force (TF). After the extraction of morphological features, temporal features were calculated (milliseconds [ms]) including diastole (MVC – MVO timing), systole (AVO – AVC timing), isovolumic contraction time (IVCT; MVC – AVO), isovolumic relaxation time (IVRT; AVC – MVO) and end of REP. Every heartbeat within the one-minute data collection was analyzed.¹⁵

Common performance indices were calculated following the calculation of the temporal features. Specifically, the systolic performance index (SPI) was calculated as IVCT/systole, the diastolic performance index (DPI) was calculated as IVRT/systole, and the myocardial or heart performance index (or Tei index) was calculated as (IVCT + IVRT)/systole.^{15,24,25}

The participant recorded data on three separate occasions (“incidents”) that included experiencing a prominent occupational stressor, that is, a potentially psychologically traumatic event: (1) Incident A—the participant exhibited psychological work-related distress due to suicidal tendencies of a client; (2) Incident B—the participant exhibited psychological work-related distress again due to a bomb scare/potential terrorism issue; and (3) Incident C—the recording average over an unusually stressful work week. As the participant collected data daily, a two-day average prior to the potentially psychologically traumatic event exposure is presented as the baseline.

Case study rationale

The CARE case report guidelines²⁶ were followed for this case study. The case study presented in this paper provides examples of occupational stress in a participant who experienced three highly stressful events to illustrate the effects of PPTE on cardiac function. However, this participant did not experience PTSD, but repeated bouts of acute stress, which can have a potential long-lasting effect on the heart. In fact, the Royal Canadian Mounted

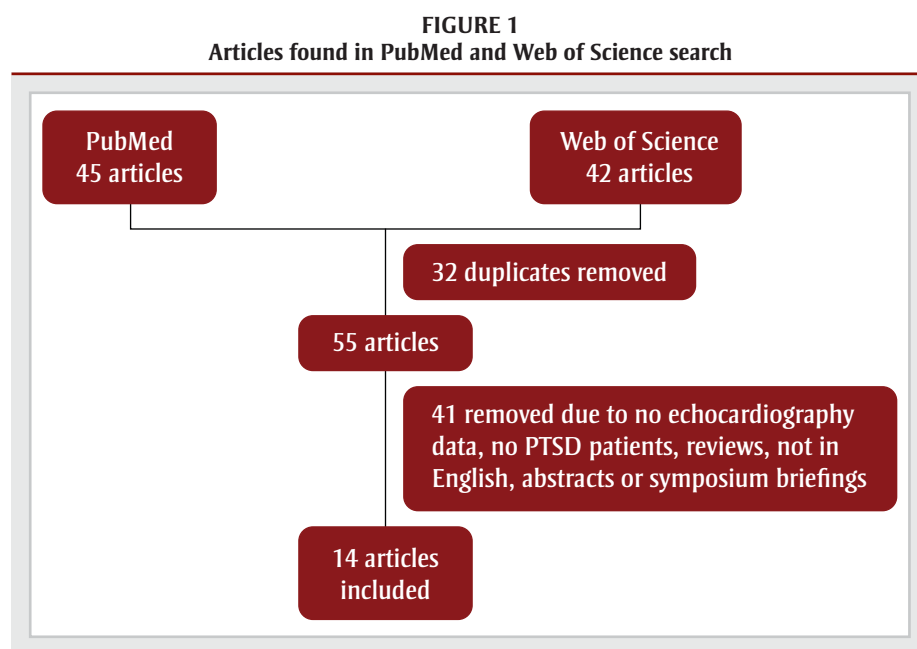
Police (RCMP) has recently developed a study protocol that observes exposure to PPTE, which can cause posttraumatic stress injuries.²⁷ Furthermore, exposure to PPTE can result in PTSD.^{28,29} For example, nurses often experience PPTE, with the exposures being associated with mental disorders such as PTSD.²⁹ Eliminating or even reducing potentially psychologically traumatic event exposures may reduce the prevalence of PTSD among nurses by up to 56.7%.²⁹ The impact of these PPTE on the cardiac cycle are not well understood. The case study presented here serves as a proof of concept that potentially psychologically traumatic event exposures can induce acute changes to cardiac function, which explains altered cardiac function in PTSD following repeated stressors.

Results

Literature review

Figure 1 is a flow diagram of our search, which resulted in 45 studies from PubMed/MEDLINE and 42 studies from Web of Science. Of these 87 studies, 32 were duplicates, resulting in 55 articles found from the literature search. Seven of the studies were case studies, 1 was focussed on cardiac biomarkers, 1 was a symposium briefing, 5 had an emphasis on anatomical changes of the brain, 2 contained electrocardiogram (ECG) data as the primary measure, 2 used animal models and emphasized molecular pathways, 2 were not written in English, 5 were review articles, 1 concentrated on acute stress disorder, and 18 were mainly concerned with PTSD symptom development following a cardiac procedure or observing the progression of PTSD symptoms following surgical procedures or anesthesia. None of the 10 articles that were found in the Web of Science (and not in PubMed) were included in our review, as four studies did not include echocardiography data, 1 did not include a population with PTSD, 1 was a review, 1 was an abstract, and 3 focussed on anatomical changes of the brain. As per Figure 1, 41 of the 55 articles were removed, resulting in 14 articles in this review.

The PubMed search also resulted in one study centred around echocardiographic changes due to PTSD, evidencing left ventricular diastolic function impairment specific to the end-diastolic pressure and left ventricular suction power in participants



Abbreviation: PTSD, posttraumatic stress disorder.

Note: The search of PubMed and Web of Science databases covered the time from database inception to 30 April 2022.

with PTSD.⁷ The article by Hieda and colleagues⁷ was the study most reflective of echocardiographic results due specifically to PTSD in the absence of other cardiac dysfunction. The participants did include three women who were menopausal in the PTSD group and one woman who was menopausal in the control group, which can potentially influence cardiac function.⁷

Takotsubo cardiomyopathy

Many of the case studies identified for this review described Takotsubo cardiomyopathy or reverse Takotsubo cardiomyopathy (Table 1), a condition with myocardial infarction-like symptoms due to stress that leads to apical ballooning (i.e. enlargement of the left ventricle). Most patients (i.e. 3 of 4) developed left ventricular abnormalities following exposure to a motor vehicle accident,³⁰ physical assault,³¹ vigorous arguments³² or serving in Iraq and subsequently being diagnosed with PTSD.³³ A prospective study with Takotsubo patients evidenced that the impairments in left ventricular ejection fraction had returned to normal at three-month follow-up.³⁴

Previous cardiac complications

Studies with participants who have multiple cardiac complications can also provide some indirect insights into the relationship between PTSD and cardiac function (Table 1). For example, patients with mitral regurgitation have reported higher PTSD symptoms, but without statistically significant differences between patients with (n = 44)

and without (n = 144) PTSD;³⁵ however, cardiac disease was a confounding variable because patients with PTSD also reported more somatic symptoms. PTSD was associated with increased mortality when comparing 111 970 veterans who had heart failure and reduced ejection fraction but no PTSD to 11 039 veterans with heart failure and reduced ejection fraction and PTSD.³⁶ No other cardiac data was provided that discriminated between patients with and without PTSD.

In a study of patients (n = 128) with implantable cardioverter-defibrillators, being female and having impaired left ventricular ejection fraction was statistically significantly related to PTSD severity;³⁷ unfortunately, the cardiac data and psychological data were collected within 30 days of each other instead of on the same day. A study of coronary heart disease found statistically significant differences in left ventricular ejection fraction between patients with (n = 95) and without (n = 927) current PTSD, possibly due in part to damaged ventricular function.³⁸ There is evidence that patients with acute myocardial infarction following percutaneous coronary intervention who had left ventricular ejection fraction < 50% are more likely to have PTSD;³⁹ however, studies of post-myocardial infarction patients have also shown no statistically significant differences between patients with (n = 40) and without (n = 19) PTSD.^{40,41}

TABLE 1
Summary of relevant cardiac and PTSD studies in the literature review

Author	Sample size	Population	Age (average \pm SD), in years	Main results	Strengths/limitations
Hieda et al., 2019 ⁷	28	14 women with PTSD and 14 women without PTSD as controls (diagnosed using the CAPS-5 scale); no other cardiac comorbidities	43.9 \pm 11.6	PTSD showed greater E/e' (end-diastolic pressure) and decreased Vp (LV suction power during the early phase of diastole), suggesting impaired LV diastolic function	<ul style="list-style-type: none"> • Direct echocardiographic measurements • Female population only • 3 menopausal women in PTSD group and 1 in control, which can influence results
Sawatari et al., 2016 ³⁷	128	Outpatients with implantable cardioverter-defibrillators; 103 males	59 \pm 16	Being female and impaired LVEF were found to be related to PTSD severity, which was more obvious when LVEF < 35%	<ul style="list-style-type: none"> • Presence of implantable cardioverter-defibrillators makes it difficult to assess and provides an extra influence on cardiac function • Echocardiography and questionnaire data were not collected on the same day • Patients were on many medications
Bayer-Topilsky et al., 2013 ³⁵	266	186 patients (134 males) with moderate to severe mitral regurgitation and 80 controls (38 patients with normal cardiac function and 42 patients with mild mitral valve prolapse; 64% had mild mitral regurgitation)	60 \pm 13	Greater PTSD scores in mitral regurgitation patients and no significant differences in echocardiographic measures	<ul style="list-style-type: none"> • No causal link can be implied as this is a prospective study • Only 42 of the 186 test subjects had PTSD • PTSD group had greater somatic symptoms due to cardiac complications
Ilhan et al., 2013 ⁴⁴	24	12 PTSD patients (11 females) and 12 healthy controls	28.4 \pm 7.5	No significant differences in LV function using transthoracic echocardiography	<ul style="list-style-type: none"> • Small sample size • Primary focus on electrocardiogram parameters • Limited demographic information
Xue et al., 2012 ⁴³	891	91 military veterans with PTSD (89 males)	66 \pm 13	Decreased left ventricular end-systolic diameter and increased ejection fraction for PTSD participants at baseline	<ul style="list-style-type: none"> • Only baseline echocardiograms (no echocardiogram data on follow up) • Focus on cardiac biomarkers of injury • Unequal sample sizes per group (those with compared to without PTSD)
Waldenborg et al., 2011 ³⁴	13	9 patients with Takotsubo cardiomyopathy with cardiac data (all female)	Range from 69–74	Impairments in left ventricular ejection fraction was back to normal during the 3-month follow-up	<ul style="list-style-type: none"> • Small sample size • No other echocardiographic data • No direct comparison to PTSD
Cohen et al., 2009 ³⁸	1022	95 patients with current PTSD (72 males) and 927 without current PTSD	61 \pm 11	No significant differences in left ventricular ejection fraction or ischemia	<ul style="list-style-type: none"> • Unequal sample sizes • PTSD can increase cardiovascular disease risk • No causal implications • Other cardiac complications
Guler et al., 2009 ⁴⁰	394	69 patients completed diagnostic interview and 40 had PTSD	61 \pm 10	Left ventricular ejection fraction was not found to be a predictor of PTSD status	<ul style="list-style-type: none"> • Unequal sample sizes • No causal implications • Post myocardial infarction • Ventriculography technique was used for left ventricular ejection fraction
Lakusic et al., 2007 ⁴²	68	34 male veterans with myocardial infarction history and PTSD, and 34 male veterans with myocardial infarction history and no PTSD	49 \pm 8	No significant differences in echocardiography assessments	<ul style="list-style-type: none"> • Primary focus on ECG monitored heart rate variability • Myocardial infarction can influence results

Continued on the following page

TABLE 1 (continued)
Summary of relevant cardiac and PTSD studies in the literature review

Author	Sample size	Population	Age (average \pm SD), in years	Main results	Strengths/limitations
Pedersen et al., 2003 ⁴¹	227	112 patients (33 females) four to six weeks post myocardial infarction, and 115 healthy controls; 25 patients and 8 controls qualified as PTSD	60 \pm 9.7	Left ventricular ejection fraction not associated with PTSD diagnosis	<ul style="list-style-type: none"> • Self-assessed psychological measures • Myocardial infarction may have affected responses • Unequal sample sizes

Abbreviations: CAPS-5, clinician administered PTSD scale; E/e', ratio of early mitral inflow velocity to mitral annular early diastolic velocity; LV, left ventricular; LVEF, left ventricular ejection fraction; PTSD, posttraumatic stress disorder; SD, standard deviation; Vp, velocity of propagation.

Notes: Only studies with echocardiography measurements in PTSD patients are included. No case studies are included.

Finally, a reference review of the articles identified another study that used echocardiography data and heart rate variability to assess cardiac function in patients with PTSD and a pre-existing history of myocardial infarction.⁴² The results evidenced no statistically significant differences in the end-diastolic diameter of the left ventricle or the left ventricular ejection fraction between patients with (n = 34) and without (n = 34) PTSD.⁴²

The remaining studies with patients who had cardiac complications focussed on questionnaires or did not have echocardiographic data (Table 1).

PTSD only

Baseline echocardiography has also been used to show decreased left ventricular end-systolic diameter and increased ejection fraction for military veterans with PTSD (n = 91) in comparison to military veterans without PTSD (n = 800).⁴³ Other research results indicate no statistically significant differences in echocardiography parameters between patients with PTSD (n = 12) and healthy controls (n = 12),⁴⁴ although the results were likely mediated by the small sample size.

There is nonclinical evidence suggesting that increased sympathetic autonomic tone (as assessed by heart rate variability) is induced during stress;⁴⁵ however, the information provided by heart rate variability is very limited and cannot be used to describe the direct underlying mechanisms or functional morphology. The echocardiography research with PTSD suggests that impaired left ventricular diastolic function is associated with PTSD in the absence of comorbid cardiac concerns. Most studies that provide echocardiographic data involve patients with pre-existing cardiac complications. Prospective studies are required to assess for mechanical cardiac changes among persons with PTSD

who do not have pre-existing cardiac complications, such as the RCMP Study Protocol.²⁷

Case report using seismocardiography

Participant

The participant was a male aged 33 years (BMI = 29 kg/m²) working in both a clinical health rehabilitation setting and an office. The participant had no smoking history, drank socially and was sedentary during stressful work time.

Incident A

Incident A involved the participant learning that one of his clients at the rehabilitation hospital where he worked was suicidal. The participant worked closely with his client to help them deal with and recover from the suicidal experience, and in the process the participant also experienced psychological distress. A seismocardiogram recording was taken the morning following the incident. Heart rate was similar to baseline; therefore, the cardiac timing changes are not influenced by any heart rate variations. Of the abnormal values in cardiac timing, IVCT was prolonged to 57 ms (48%), and systolic time was reduced to 218 ms (-24%). Reductions in AS to MVC (-47%) and REP (-26%) in the absence of an elevated HR were also found and are suggestive of inadequate maintenance of ventricular pressure. The change in systole consequently impacts the performance indices, with the elevations in MPI (60%), SPI (93%) and DPI (47%) all suggesting elevated cardiac stress.¹⁵ The cardiac stress elevation is further exemplified by the increase in twist force (milligravity [mG]).

Incident B

Incident B involved a bomb threat at the participant's workplace, and resulted in psychological distress. The participant was not involved with the clearance of the bomb threat; however, because of the

threat, the workplace was more stressful than expected. The participant was required to take precautions and clear the area quickly. Once the bomb threat was cleared, the participant returned to work. The recording was taken during resting conditions after work. Similar to Incident A, an increase in IVCT to 49 ms (26%) and a decrease in systole to 220 ms (-23%) were seen. As Incident A also suggested, the acute distress implies that there were issues with maintaining adequate ventricular pressure, and this is further supported by the reductions in AS to MVC to 14 ms (-32%) and REP to 53 ms (-36%) in the absence of an elevated heart rate. Given the decreased systolic time period, there was an increase in the performance indices, with MPI at 0.63 (48%), thus suggesting that cardiac stress was elevated. This elevation in cardiac stress is further shown by the increase in TF to 12 mG (68%).

Incident C

Incident C involved distress owing to a week of elevated occupational stress due to unexpected changes to workplace requirements, difficult and highly stressful working conditions and sustained anxiety. During this week, the participant's heart rate was similar to baseline. As the presented data is an average of the data for the week, the increase in IVCT to 46 ms (19%) and decrease in systole to 234 ms (-19%) was not the same magnitude of change compared to Incidents A and B. However, as seen from the decrease in REP to 65 ms (-21%) and the increase in both MPI and TF to 0.59 (38%) and 11 mG (53%), respectively, issues with maintenance of ventricular pressure and elevations in cardiac stress were still prevalent.

All findings from the cardiac cycle parameters for each variable are available in Table 2.

Discussion

Variable results

Research on PTSD patients and cardiac function as the primary outcome is currently limited. The literature review results suggest that cardiac dysfunction varies from study to study. For example, diastolic⁷ and systolic^{37,43} impairments were found in patients diagnosed with PTSD, even though other research results suggest that parameters such as LVEF are not associated with PTSD.^{34,38,41,42,44} Despite the varying results, most studies have not correlated echocardiography parameters with PTSD as a primary research outcome.

Implications from case report using seismocardiography

This case study serves as a proof of concept that impairments in the cardiac cycle timing intervals can occur during acute occupational stressors. Exposures to PPTE and other cumulative stressors can lead to chronic mental health challenges, including but not limited to PTSD,^{28,29} therefore, early detection of cardiac dysfunction can help reinforce proactive health care behaviours. We have observed that sustained complications with maintaining ventricular pressure can lead to complications with LV suction power in women with PTSD.⁷ Our case study results

suggest that the potential diastolic dysfunction present in PTSD can result from elevated cardiac stress accumulated during the acute period.

The changes noted in the cardiac profile for PPTE in our case study are due to elevated acute stress. Elevations in IVCT (19% to 48%) and TF (53% to 73%), and reductions in systole (−19% to −24%), AS to MVC (−14% to −47%) and REP (−21% to −36%), all suggest that acute stress resulted in cardiac complications in trying to maintain adequate ventricular pressure. Increased IVCT implies systolic complications and limitations in building ventricular pressure gradients. Considering the Wiggers model,⁴⁶ acute stress appears to prolong the time required to build up ventricular pressure to overcome the opening of the aortic valve, and once AVO occurs, there is an elevated TF, which is required to sustain stroke volume (and cardiac output) in a reduced systolic time period.

Therefore, as supported by other research in which reduced ejection time was a predictor of cardiac complications, including all-cause mortality and heart failure,⁴⁷ sustained psychological stress can result in prolonged periods of shortened ejection time, thereby heightening the risk for cardiac disease and serving as an indicator

that a posttraumatic stress injury may have occurred. Importantly, the changes in the cardiac cycle intervals all occurred with no increase in HR. Altered cardiac cycle intervals are expected when HR is increased (for example, reduced timing intervals during exercise), and therefore, the case study findings directly imply altered systolic and diastolic function in this participant.

The MPI can reflect cardiac stress.¹⁵ The MPI is directly related to both IVRT and IVCT, and is inversely related to systolic time $[(IVCT + IVRT)/systole]$.^{24,25} Given the changes to systolic time and IVCT in this participant, the acute stress experienced by our case study participant resulted in an elevated MPI. An elevated MPI appears common among people with a history of major adverse cardiac events and is also useful for predicting major adverse cardiac events in the general public.⁴⁸ Pro-inflammatory cytokines are associated with cardiac cycle timing intervals and the MPI.⁴⁹ While our findings are currently limited to our case study, the mechanisms suggest that elevated psychological stress can elevate pro-inflammatory cytokine release, which can impair cardiac function. Psychological stress has been associated with several posttraumatic stress injuries, as evidenced by journalists exposed to PPTE.⁵⁰ Chronic exposures to occupational stressors among public safety personnel and military personnel has been shown to elevate levels of circulating cytokines and impair blood brain barrier integrity.⁵¹ The changes may result from physical complications (e.g. sleep deprivation, exhaustion) alone or in combination with acute exposures to PPTE, which appear to be associated with elevated risk for PTSD.⁵¹ More research is required for a better understanding of the cardiac cycle timing intervals in patients with PTSD.

Strengths and limitations

The literature review was extensive and provided a background of the research available pertaining to echocardiography and PTSD. Furthermore, our case study highlights the novel insights of altered cardiac timing intervals in PPTE, which will impact the performance indices (i.e. MPI, SPI, DPI) reported. These findings, which imply that acute occupational stress can have a direct impact on the cardiac cycle,

TABLE 2
Cardiac cycle timing intervals and magnitude of change from baseline for all three incidents for the case study participant

Cardiac indices	Baseline	Incident A	Incident B	Incident C
Heart rate (bpm)	70	71 (1)	73 (4)	68 (−3)
IVCT (ms)	39	57 (48)	49 (26)	46 (19)
IVRT (ms)	83	91 (10)	91 (10)	90 (9)
Systole (ms)	287	218 (−24)	220 (−23)	234 (−19)
Diastole (ms)	459	454 (−1)	471 (3)	496 (8)
MVO to E (ms)	12	8 (−3)	8 (−17)	10 (−10)
AS to MVC (ms)	20	11 (−47)	14 (−32)	18 (−14)
REP (ms)	82	61 (−26)	53 (−36)	65 (−21)
MPI	0.43	0.68 (60)	0.63 (48)	0.59 (38)
SPI	0.14	0.26 (93)	0.22 (63)	0.20 (47)
DPI	0.29	0.42 (47)	0.41 (44)	0.39 (36)
AS (mG)	6	5 (−4)	5 (−11)	5 (−18)
TF (mG)	7	12 (73)	12 (68)	11 (53)

Abbreviations: AS, atrial systole; AS to MVC, atrial systole to mitral valve closure; bpm, beats per minute; DPI, diastolic performance index; IVCT, isovolumic contraction time; IVRT, isovolumic relaxation time; mG, milligravity; ms, millisecond; MPI, myocardial performance index; MVO to E, mitral valve open to E wave; REP, rapid ejection period; SPI, systolic performance index; TF, twist force.

Note: All data are presented as the mean value for each incident (% change from baseline).

have not been reported previously in the literature.

However, the case study approach does limit the generalizability of the study. The research available in the literature is conflicting and therefore there are difficulties in understanding the mechanism of cardiac dysfunction following PTSD. Furthermore, there is much variation in the methodology, sample population and outcome variables in the known literature. Finally, one of the two articles excluded for language included echocardiography assessment, which may have further enhanced our understanding of cardiac function and PTSD. Therefore, there is a need for more consistent research with respect to similar patient populations and research methodologies that can allow for a more accurate comparison of altered cardiac function in PTSD.

Conclusion

Research about cardiac cycle timing intervals and cardiac function in PTSD is nascent; nevertheless, the available literature suggests cardiac function assessments can provide new insights into posttraumatic stress injuries. We provided an overview of the published research, indicating that variations in relationships between PTSD and cardiac function appear to depend on study design. Our case study report exemplifies how PPTE appear to alter cardiac function. Prolonged stress, without rehabilitative intervention, can further impair systolic and diastolic function, resulting in a heightened risk for cardiac disease, and arguably potentiating posttraumatic stress injuries.

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Conflicts of interest

The authors have no conflicts of interest to declare.

Authors' contributions and statement

JS, JPN—conceptualization, data curation, formal analysis, methodology. JPN—resources,

funding acquisition. JS, JPN, NC—writing—original draft, writing—review and editing.

The content and views expressed in this article are those of the authors and do not necessarily reflect those of the Government of Canada.

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