Using Heart Rate Fluctuations for Stress Monitoring in Post-Traumatic Stress Disorder (PTSD) Patients: A Scoping Literature Review

This study focuses on use of heart rate (HR) measurements and aspects of HR that affect measurement to be a reliable indicator for assessment of anxiety disorders like Posttraumatic stress disorder (PTSD). PTSD is a concern for combat veterans. The motivation for the paper is the lack of continuous monitoring of PTSD individuals like combat veterans to assess their status in daily life. The goal is to intervene at the correct moment to reduce the ill effects of past traumatic events. Heart rate measures like resting heart rates along with heart rate accelerations are important measures to monitor status of PTSD individuals than heart rate variability (HRV). Heart rate accelerations in response to traumatic reminders are more reliable than resting or basal heart rates. In conclusion, the report of this scoping review can educate key informants about the use of heart rate for assessment and monitoring of certain health conditions to mitigate their ill effects.

INTRODUCTION

Post-traumatic stress disorder (PTSD) is a mental disorder that is estimated to impact up to 17% of combat veterans returning from recent wars (Richardson et al., 2010). Approximately 40% of the general populations will experience at least one traumatic event in their lifetime, of which an estimated 5.6% will develop PTSD (Holliday et al., 2015). In addition, Lustyk et al. (2012) suggests that the early onset of heart disease may be attributed to military service among PTSD positive veterans. Mortality associated with cardiovascular outcomes is linked to elevated resting heart rates (Coote, 2010), a phenomenon likely to be present in individuals with anxiety or panic disorders.

Heart rate variability, resting heart rates are used in past research studies as an indicator to predict PTSD (e.g. Blanchard et al., 1996; Elsesser et al., 2004; Suendermann et al., 2010). In spite of research, there is conflicting evidence in the literature regarding reliability of these measures for accurately predicting PTSD. Resting heart rates in addition to heart rate accelerations can be used in conjunction to reveal trends and track responses to stressful triggers to traumatic reminders for PTSD individuals. Resting heart rates as a measure of cardiovascular health and heart rate accelerations as a measure to track acute stress triggers can be used to intervene at the desired moment to reduce ill effects of anxiety disorders. In this paper, we document the findings from a scoping literature review that investigates links between heart rate, a physiological measure for investigation of anxiety disorders like PTSD.

METHOD

A systematic review was conducted in order to gather an adequate amount of relevant literature on relevance of heart rate as the indicator for predicting anxiety disorders like PTSD. A preliminary search was done to identify PTSD and heart rate associated terms and the relevant databases for use.

Two databases were used in the literature review, "Medline" and "PubMed" databases, 149 and 1,831 articles were identified respectively. All searches included either "heart rate," "stress disorders," "post traumatic" or "post-traumatic stress disorders." Other important terms were "resting heart rate" and "baseline heart rate".

The search started with a scope of the literature relevant to PTSD and heart rate in Medline and PubMed. The search was prioritized to include reviews due to sheer size of the literature on heart rate. In total, 1,980 articles were included for abstract screening and initial review from the databases. Total of 1,901 articles were excluded from the abstract screening. Seventy-nine articles were assessed for full-text eligibility; four articles from the resting heart rate literature were added from Google scholar. Forty-four articles were selected for this review. The review was conducted according to the guidelines of the Preferred Reporting Items for Systematic Review and Meta Analyses (PRISMA), (Moher et al., 2009).

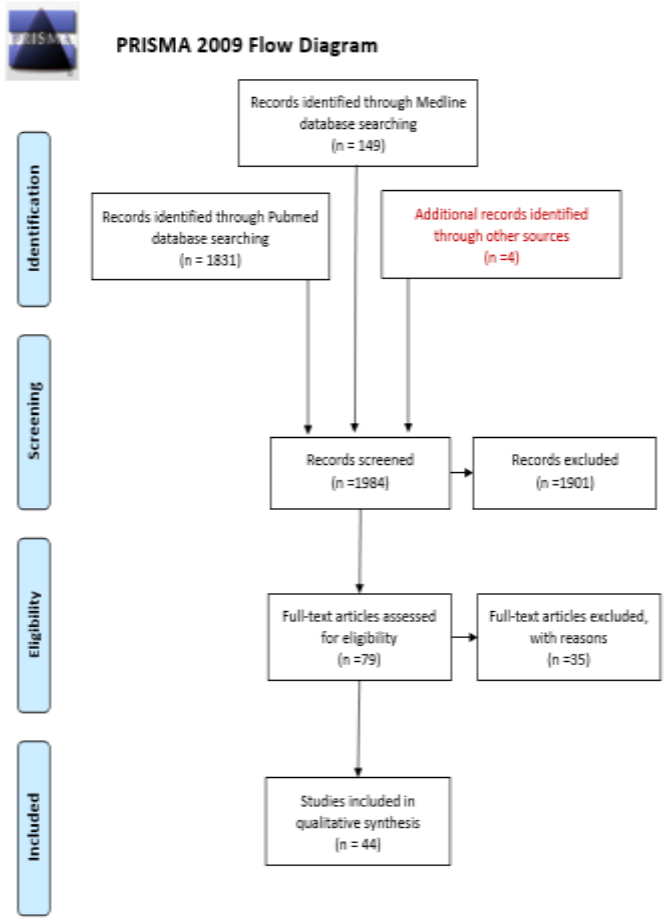

Figure 1. The PRSIMA Flow diagram for this scoping review

Table 1. Inclusion and exclusion criteria for the literature review

Inclusion criteria	Exclusion criteria
Heart rate as an indicator for predicting health outcomes	Reviews explaining neurological aspects of PTSD or heart rate
Post-traumatic stress disorder	Reviews focusing on underlying causes for anxiety disorders.
Anxiety and panic disorder	Explaining heart rate's link to oncology
Heart rate baseline	
Cardiovascular mortality	
Heart rate elevation	
Heart rate variability reviews	

RESULTS

Use of Heart Rate Variability (HRV) as Diagnostic Tool

Research shows that decreased HRV and decreased vagal cardiac modulation is associated with a variety of clinical conditions and can lead to adverse outcomes (Routledge et al., 2010). HRV is the time duration between hearts beats, usually the RR interval corresponding to the peaks of the QRS complex of an electrocardiogram (ECG) wave. Extensive research has shown that heart rate variability is used as an indicator for certain conditions in humans like mortality. Sacha (2014) has stated in the paper on heart rate and heart rate variability that HRV is a strong risk factor for cardiovascular mortality. The predictive power of HRV increases for cardiovascular related deaths and decreases for non-cardiovascular deaths in males though it decreases for all deaths in females. This suggests HRV can be used as a diagnostic indicator for adverse outcomes in males but not in females.

Sex Based Differences in Resting Heart Rates

Baseline or resting heart rates may be a more pronounced factor to predict mortality in humans than HRV. The baseline heart rate can be used to predict risk for females as well as males. Females have a higher heart rate than males because average maximum VO2 is higher in males than females (Magder, 2012). Kannel et al. (1987) stated that as age increases, there is an increase in HR for both sexes, with rates in females exceeding those of males by three bpm. Mortality rates also increase with an increase in resting heart rate for both sexes, with no indication of a safe threshold or critical value. The Framingham study (Kannel et al., 1987) of 5,070 healthy subjects that measured the heart rates of the participants every two years concluded that cardiovascular and coronary mortality rates increased, as there was an increase in resting heart rate for both sexes and for all ages. However, the study also concluded, the increased risk was more pronounced for males than females. The National Health and Nutrition Examination Survey (NHANES) I Epidemiologic Follow-up Study (Gillum et al., 1991) also concluded that elevation in resting heart rate is an autonomous risk for cardiovascular diseases or mortality.

Using Heart Rate as a Predictor for Anxiety Disorders

Heart rate has been used as a predictor for anxiety and panic disorders such as PTSD. With respect to external or internal cues of a reminder of the traumatic event, individuals with PTSD show heightened physiological responses (Suendermann et al., 2010). The paper also states that for trauma survivors, the heart rate responses to standard trauma reminders after six months of the event could be used to differentiate between individuals that have PTSD and without PTSD. People with chronic PTSD and recent trauma survivors who also suffered from acute stress disorders (ASD) at six weeks after the event, presented heart rate accelerations when they were shown personalized trauma related pictures as compared to people who were not traumatized and did not have ASD (Elsesser et al., 2004, in Suendermann et al., 2010). Other research studies have shown that traumatic event reminders along with heart rate responses can be used to predict the severity of PTSD symptoms after two and a half months till a year after the traumatic event (Elsesser et al., 2004; Blanchard et al., 1996; Kleim et al., 2009, in Suendermann et al., 2010).

Resting Heart Rate as a Predictor for PTSD

Resting heart rate immediately after a traumatic event is used as a measure for classification of individuals that later on develop PTSD.

Elevated HRs in the laboratory are observed for PTSD patients as they anticipate traumatic reminders of the event. Beckham et al. (2000), in (Woodward et al., 2009) found a five-bpm increase in basal HR for combat veterans with and without PTSD. Buckley et al. (2004), in (Woodward et al. 2009) with adjusting for covariates such as smoking, body mass index, ethnicity found basal HR was 6.6 bpm higher for PTSD positive compared to non-PTSD participants.

Kleim et al. (2010) state that females that show elevated HR to traumatic event reminders may be at particular risk of developing PTSD. Males do not showcase this pattern.

There are studies in the literature that either support or contradict the use of resting heart rate as a diagnostic measure for PTSD. Studies like (Jovanovic et al., 2009) support using basal heart rates and initial startle magnitude as indicators for PTSD. While other studies like (Hopper et al., 2006) do not support using a resting heart rate as a diagnostic measure for PTSD, their argument is it is necessary to look at both branches of the autonomic nervous system (ANS) as a substantial number of their participants with PTSD did not show elevated basal heart rates.

Physiological Theories for Increase in Heart Rate in Anxiety Disorders

Anxiety disorders are associated with reduced HRV measures (Chalmers et al., 2014). In terms of beats per minute, anxiety disorder patients have higher heart rates as compared to non-anxiety disorder patients. Chalmers et al. (2014) explained low HRV or higher heart rates by two models in theory, the polyvagal theory and the neurovisceral integration theory. Polyvagal theory links low HRV to symptoms of withdrawal behavior. The neurovisceral integration theory suggests pathways between central and peripheral nervous systems that connect autonomic, attentional and affective systems to be

ineffective in anxiety disorders. This results in difficulty in disengagement from inhibiting threat detection resulting in cases of hyper-vigilance, apprehension, heart rate elevation and an associated decrease in HRV.

In case of the autonomic nervous system (ANS), studies have observed reduced parasympathetic reactivity to stressors or emotional challenges for individuals who suffer from PTSD (Cohen et al., 2000; Sahar et al., 2001; Woodward et al., 2008, in Clapp et al., 2015).

Co-Variates that Affect Heart Rate

Important covariates that have an effect and elevate the heart rate need to be accounted for while using resting heart rates as an indicator for anxiety disorders. Alcohol, smoking, diabetes, beta-blocker use, history of heart rate problems, increased age have been associated with decreasing HRV (Tsuji et al., 1996).

The scientific theory behind diabetes and elevation of heart rate is diabetes as a chronic disease is associated with cardiac parasympathetic damage or combined sympathetic and parasympathetic damage. In addition, the sequential heart rate rise may be due to damage to vagus nerve (Ewing et al., 1981). Ewing et al. (1981) state that compared to normal subjects, patients with diabetes had faster resting heart rates taking into account covariates such as age and sex.

Cigarette smoking also increases heart rate as it transiently elevates catecholamines. Catecholamines are the principal neurotransmitters that control central nervous system functions (Kannel et al., 1987).

Increase in alcohol dose can reduce HRV to values lower than those who abstain or drink moderately (Karpyak et al., 2014).

Stress and Physiology

Stress plays an important role in physiological response of the body and affects physiological functions like heart rate. While acute stressors produce potentially beneficial changes in the human immune system based on the human's fight or flight response to potentially threatening situations; as the stressors become more chronic in nature, the immune system is affected in a negative manner (Segerstrom and Miller, 2004). While acute stress may not be able to produce negative effects, chronic psychological stress may produce changes in the system such as slowing down responses to a future stressor (Oken et al., 2015).

Lower levels of vagal modulation can leave the heart vulnerable to sudden cardiac death, as it cannot adequately counterbalance sympathetic stimulation as in case of stressors (Carnevali & Sgoifo, 2007). Studies show evidence that a higher vagal tone or lower heart rate than average at rest tends to aid in being more resilient towards stress and adapting well to a variety of situations (El-Sheikh et al., 2001; Kok & Fredrickson, 2010; Smeets et al., 2010; Souza et al., 2013, in Carnevali & Sgoifo 2007).

Shaffer et al. (2014) provide further evidence of linking stress and cardiovascular system. Based on indirect evidence, reduced HRV may correlate to increased diseases and mortality as it reduces the ability of the body to adapt to various challenges in the form of stressors. Reduced HRV may contribute to other health issues such as inflammation and hypertension.

Stults-Kolehmainen & Sinha. (2014) found out that experience of stress is also a major hindrance for efforts and motivation to be physically active. Physical activity aids in reducing the resting heart rate or increasing vagal tone (Carnevali & Sgoifo, 2007). Jensen et al. (2013) study in Denmark involving 2,798 subjects found that resting heart rate was inversely related to physical fitness. Increasing the resting heart rate (RHR) was associated with mortality in a stepwise manner accounting for covariates like physical fitness and cardiovascular risk.

Mortality Risk and Resting Heart Rate

Clinicians can use lower resting heart rate as a predictor of mortality in patients at risk of cardiac diseases (Coote, 2010). Evidence shows that risk of mortality and resting heart rate are correlated. Lower the heart rate lower the risk of mortality. Risk of mortality increased 16% per 10-bpm rise in RHR when RHR was taken as a continuous variable (Jensen et al., 2013). Jouven et al. (2005); Palatini et al. (HARVEST study) (2006); Aboyens & Criqui. (2006), in Arnold et al. (2008) have shown that for every 20 bpm increase in heart rate at rest there is an increase in risk of mortality of 30-50%. Despite research, elevation in heart rate is not well defined. Arnold et al. (2008) state that heart rate elevation is multidimensional and there is no precise definition for defining elevation in heart rate.

For establishing thresholds for elevation of heart rates, (Spodick, 1996, in Arnold et al 2008) provided new thresholds for bradycardia and tachycardia. The authors provide a new rounded sinus rhythm of 50 bpm to 90 bpm as opposed to the 60-100 bpm commonly used range for bradycardia and tachycardia respectively.

Anxiety Disorders, Heart Rates and Effects on Health

Lower HRV or higher heart rates are linked to anxiety disorders; these higher heart rates also increase cardiovascular disease risk for individuals who are diagnosed with anxiety and panic disorders such PTSD. Sumner et al. (2015) looking at trauma exposure and PTSD symptoms for predicting cardiovascular events in women showed that higher PTSD symptoms were associated with increased risk of cardiovascular disease incidences in their 20-year study on female nurses. Boscarino (2008) looked at PTSD and an earlier than expected heart disease mortality in Vietnam veterans, the findings of the study show that PTSD was associated with heart disease mortality among veterans who were previously free of any heart diseases before positive identification of PTSD. The study also suggests that the early onset of heart disease may be due to military service in the PTSD positive veterans' group.

Suglia et al. (2015) on violence and cardiovascular health had the following findings; child abuse (trauma) was consistently linked to increased cardiovascular disease in

adulthood, despite the fact that there are many definitions of child abuse. Long-term effects of violence exposure have been linked to depression, risk-taking behavior and aggression (Suglia et al., 2015). The effects of children and adults exposed to violence may be adverse mental effects such as emotional disturbances like PTSD and adverse physical effects in case of absence of mental effects. Trickett et al. (2011) study of impact of sexual abuse on female development suggests that females who are sexually abused were more prone to be depressed, suffer from PTSD and dissociative symptoms; they were also changed biologically with lower resting levels of cortisol, asymmetrical stress responses and cognitive ability deficits. Lower Cortisol along with abnormal Adrenocorticotropic hormone (ACTH) levels are seen in anxiety disorder patients (BBC, 2009).

Several studies have shown that individuals with PTSD have higher risk of cardiovascular disease (CVD). It should be noted that (Gallo et al., 2014) in their review of literature stated that health risks occur only when trauma is followed by PTSD.

DISCUSSION

Based on the literature review, in order to study anxiety disorders a majority of studies focus on HRV instead of heart rate. Although, evidence suggests a lower heart rate variability (HRV) for anxiety disorder patients, the analysis and assessment based on HRV is not conducive for continuous monitoring as there are few commercial products that can provide HRV analysis to the extent that it can be used as a diagnostic assessment tool in day-to-day life. In addition to that, since females have higher heart rates compared to males and limitations of HRV to provide values for higher ranges of heart rate, HRV might not provide reliable measurements that are ideal for both sexes. Heart rate compared to HRV is easier to measure. Commercial products are available to measure the heart rate. There is a research gap in the literature comparing these two measures with available technology in daily life of patients.

To date, research to the best of our knowledge has tended to focus on establishing a relationship between anxiety disorders and basal heart rates to provide a trend of the condition rather than establishing thresholds or ranges for heart rates. Studies have shown harmful effects of rising resting heart rates. This is particularly true for individuals with anxiety disorders. These individuals are at risk to develop cardiovascular diseases and higher chances of mortality based on their higher resting heart rates.

Research studies have focused on using heart rate as an indicator; however, few studies track changes in resting heart rates for individuals over a period after they are diagnosed with PTSD. This can address the rise in resting heart rates and recommend changes that could slow down the initiation of cardiovascular diseases.

Evidence from the literature review shows that individuals with anxiety or panic disorders like PTSD have higher heart rate accelerations when exposed to reminders of traumatic events undergone in the time frame of a year. There is a research gap in utilizing the heart rate accelerations with continuous monitoring of individuals with PTSD. Continuous monitoring can allow for intervention at the right moment to help patients with anxiety disorders.

The study was limited to two databases and might have missed articles that were not present in the medical databases. The search terms were limited to heart rate and PTSD, and articles that involve different terms for these two constructs were not included. The search was limited to English language and foreign language papers were not included. This is a scoping review of reviews, and certain original studies might have been missed.

Future studies should inspect closely the link between resting heart rates and heart rate accelerations for designing a monitoring system for anxiety disorders using heart rate sensors.

CONCLUSIONS

The study offers some important insights into using heart rate as a physiological indicator for assessment of anxiety disorders. With an increase in the use of smart wearables that include a biometric sensor to measure heart rate, there is an opportunity to use continuous monitoring of heart rate as an indicator for assessment of heart rate accelerations. The paper focuses on aspects of heart rate measurements that need to be taken into account for effectively using heart rate for objective assessment. This scoping review can educate key informants about the use of heart rate as a type of physiological measure for assessment and monitoring of certain anxiety disorders to mitigate their ill effects.

REFERENCES

Aboyans, V., & Criqui, M. H. (2006). Can we improve cardiovascular risk prediction beyond risk equations in the physician's office? *Journal of clinical epidemiology*, *59*(6), 547-558.

Arnold, J. M., Fitchett, D. H., Howlett, J. G., Lonn, E. M., & Tardif, J. C. (2008). Resting heart rate: a modifiable prognostic indicator of cardiovascular risk and outcomes? Canadian Journal of Cardiology, 24, 3A-15A.

BBC (2009) Asperger's stress hormone 'link'. Available at: http://news.bbc.co.uk/2/hi/health/7976489.stm

Beckham, J. C., Feldman, M. E., Barefoot, J. C., Fairbank, J. A., Helms, M. J., Haney, T. L., ... & Davidson, J. R. (2000). Ambulatory cardiovascular activity in Vietnam combat veterans with and without posttraumatic stress disorder. *Journal of consulting and clinical psychology*, *68*(2), 269.

Boscarino, J. A. (2008). A prospective study of PTSD and early-age heart disease mortality among Vietnam veterans: implications for surveillance and prevention. Psychosomatic medicine, 70(6), 668.

Buckley, T. C., Holohan, D., Greif, J. L., Bedard, M., & Suvak, M. (2004). Twenty-four-hour ambulatory assessment of heart rate and blood pressure in chronic PTSD and non-PTSD veterans. *Journal of Traumatic Stress*, *17*(2), 163-171.

Carnevali, L., & Sgoifo, A. (2007). Vagal modulation of resting heart rate in rats: the role of stress, psychosocial factors, and physical exercise. *The Role of Physical Fitness on Cardiovascular Responses to Stress*, 19.

Chalmers, J. A., Quintana, D. S., Abbott, M. J., & Kemp, A. H. (2014). Anxiety disorders are associated with reduced heart rate variability: a meta-analysis. *Frontiers in psychiatry*, *5*, 80.

Clapp, J. D., Patton, S. C., & Beck, J. G. (2015). Expressive inhibition in response to stress: Implications for emotional processing following trauma. *Journal of anxiety disorders*, *29*, 109-118.

Cohen, H., Benjamin, J., Geva, A. B., Matar, M. A., Kaplan, Z., & Kotler, M. (2000). Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: application of power spectrum analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. *Psychiatry research*, *96*(1), 1-13.

Coote, J. H. (2010). Recovery of heart rate following intense dynamic exercise. Experimental physiology, 95(3), 431-440.

Elsesser, K., Sartory, G., & Tackenberg, A. (2004). Attention, heart rate, and startle response during exposure to trauma-relevant pictures: a comparison of recent trauma victims and patients with posttraumatic stress disorder. *Journal of abnormal psychology*, *113*(2), 289.

El-Sheikh, M., Harger, J., & Whitson, S. M. (2001). Exposure to interparental conflict and children's adjustment and physical health: The moderating role of vagal tone. *Child development*, *72*(6), 1617-1636.

Ewing, D. J., Campbell, I. W., & Clarke, B. F. (1981). Heart rate changes in diabetes mellitus. *The Lancet*, *317*(8213), 183-186.

Gallo, L. C., Roesch, S. C., Fortmann, A. L., Carnethon, M. R., Penedo, F. J., Perreira, K., ... & Sotres-Alvarez, D. (2014). Associations of chronic stress burden, perceived stress, and traumatic stress with cardiovascular disease prevalence and risk factors in the HCHS/SOL Sociocultural Ancillary Study. Psychosomatic medicine, 76(6), 468.

Gillum, R. F., Makuc, D. M., & Feldman, J. J. (1991). Pulse rate, coronary heart disease, and death: the NHANES I Epidemiologic Follow-up Study. *American heart journal*, *121*(1), 172-177.

Holliday, R., Smith, J., North, C., & Surís, A. (2015). Developing the PTSD Checklist-I/F for the DSM-IV (PCL-I/F): assessing PTSD symptom frequency and intensity in a pilot study of male veterans with combat-related PTSD. Behavioral Sciences, 5(1), 59-69.

Hopper, J. W., Spinazzola, J., Simpson, W. B., & van der Kolk, B. A. (2006). Preliminary evidence of parasympathetic influence on basal heart rate in posttraumatic stress disorder. *Journal of Psychosomatic Research*, *60*(1), 83-90.

Jensen, M. T., Suadicani, P., Hein, H. O., & Gyntelberg, F. (2013). Elevated resting heart rate, physical fitness and all-cause mortality: a 16-year follow-up in the Copenhagen Male Study. *Heart*, *99*(12), 882-887.

Jensen, M. T., Suadicani, P., Hein, H. O., & Gyntelberg, F. (2013). Elevated resting heart rate, physical fitness and all-cause mortality: a 16-year follow-up in the Copenhagen Male Study. *Heart*, *99*(12), 882-887.

Jouven, X., Empana, J. P., Schwartz, P. J., Desnos, M., Courbon, D., & Ducimetière, P. (2005). Heart-rate profile during exercise as a predictor of sudden death. *New England Journal of Medicine*, *352*(19), 1951-1958.

Jovanovic, T., Norrholm, S. D., Sakoman, A. J., Esterajher, S., & Kozarić-Kovačić, D. (2009). Altered resting psychophysiology and startle response in Croatian combat veterans with PTSD. International Journal of Psychophysiology, 71(3), 264-268.

Kannel, W. B., Kannel, C., Paffenbarger, R. S., & Cupples, L. A. (1987). Heart rate and cardiovascular mortality: the Framingham Study. *American heart journal*, *113*(6), 1489-1494.

Kannel, W. B., Kannel, C., Paffenbarger, R. S., & Cupples, L. A. (1987). Heart rate and cardiovascular mortality: the Framingham Study. *American heart journal*, *113*(6), 1489-1494.

Karpyak, V. M., Romanowicz, M., Schmidt, J. E., Lewis, K. A., & Bostwick, J. M. (2014). Characteristics of Heart Rate Variability in Alcohol-Dependent Subjects and Nondependent Chronic Alcohol Users. *Alcoholism: Clinical and Experimental Research*, *38*(1), 9-26.

Kleim, B., Wilhelm, F. H., Glucksman, E., & Ehlers, A. (2010). Sex differences in heart rate responses to script-driven imagery soon after trauma and risk of posttraumatic stress disorder. *Psychosomatic medicine*, *72*(9), 917.

Kok, B. E., & Fredrickson, B. L. (2010). Upward spirals of the heart: Autonomic flexibility, as indexed by vagal tone, reciprocally and prospectively predicts positive emotions and social connectedness. *Biological psychology*, *85*(3), 432-436.

Lustyk, M. K. B., Douglas, H. A., Bentley, J. A., & Gerrish, W. G. (2012). Cardiovascular responses to a laboratory stressor in women: Assessing the role of body awareness. Body, Movement and Dance in Psychotherapy, 7(1), 55-70.

Magder, S. A. (2012). The ups and downs of heart rate. *Critical care medicine*, *40*(1), 239-245.

Moher, D., Liberati, A., Tetzlaff, J., Altman, D. G., & Prisma Group. (2009). Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *PLoS med*, *6*(7), e1000097.

Oken, B. S., Chamine, I., & Wakeland, W. (2015). A systems approach to stress, stressors and resilience in humans. Behavioural brain research, 282, 144-154.

Palatini, P., Dorigatti, F., Zaetta, V., Mormino, P., Mazzer, A., Bortolazzi, A., ... & HARVEST Study Group. (2006). Heart rate as a predictor of development of sustained hypertension in subjects screened for stage 1 hypertension: the HARVEST Study. *Journal of hypertension*, *24*(9), 1873-1880.

Richardson, L. K., Frueh, B. C., & Acierno, R. (2010). Prevalence estimates of combat-related post-traumatic stress disorder: critical review. Australian and New Zealand Journal of Psychiatry, 44(1), 4-19.

Routledge, F. S., Campbell, T. S., McFetridge-Durdle, J. A., & Bacon, S. L. (2010). Improvements in heart rate variability with exercise therapy. *Canadian Journal of Cardiology*, *26*(6), 303-312.

Sacha, J. (2014). Interaction between heart rate and heart rate variability. *Annals of Noninvasive Electrocardiology*, *19*(3), 207-216.

Sahar, T., Shalev, A. Y., & Porges, S. W. (2001). Vagal modulation of responses to mental challenge in posttraumatic stress disorder. *Biological psychiatry*, *49*(7), 637-643.

Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the human immune system: a meta-analytic study of 30 years of inquiry. *Psychological bulletin*, *130*(4), 601

Shaffer, F., McCraty, R., & Zerr, C. L. (2014). A healthy heart is not a metronome: an integrative review of the heart's anatomy and heart rate variability. *Frontiers in psychology*, *5*, 1040.

Smeets, T., Giesbrecht, T., Raymaekers, L., Shaw, J., & Merckelbach, H. (2010). Autobiographical integration of trauma memories and repressive coping predict post-traumatic stress symptoms in undergraduate students. *Clinical psychology & psychotherapy*, *17*(3), 211-218.

Souza, G. G. L., Magalhães, L. N., Da Cruz, T. A. R., Mendonça-De-Souza, A. C. F., Duarte, A. F. A., Fischer, N. L., ... & Figueira, I. (2013). Resting vagal control and resilience as predictors of cardiovascular allostasis in peacekeepers. *Stress*, *16*(4), 377-383.

Spodick, D. H. (1996). Normal sinus heart rate: appropriate rate thresholds for sinus tachycardia and bradycardia. Southern medical journal, 89(7), 666-667.

Spodick, D. H., Raju, P., Bishop, R. L., & Rifkin, R. D. (1992). Operational definition of normal sinus heart rate. *The American journal of cardiology*, *69*(14), 1245-1246.

Stults-Kolehmainen, M. A., & Sinha, R. (2014). The effects of stress on physical activity and exercise. *Sports medicine*, *44*(1), 81-121.

Suendermann, O., Ehlers, A., Boellinghaus, I., Gamer, M., & Glucksman, E. (2010). Early heart rate responses to standardized trauma-related pictures predict posttraumatic stress disorder–a prospective study. *Psychosomatic medicine*, *72*(3), 301.

Suglia, S. F., Sapra, K. J., & Koenen, K. C. (2015). Violence and cardiovascular health: a systematic review. American journal of preventive medicine, 48(2), 205-212.

Sumner, J. A., Kubzansky, L. D., Elkind, M. S., Roberts, A. L., Agnew-Blais, J., Chen, Q., ... & Suglia, S. F. (2015). Trauma exposure and posttraumatic stress disorder symptoms predict onset of cardiovascular events in women. Circulation, CIRCULATIONAHA-114.

Trickett, P. K., Noll, J. G., & Putnam, F. W. (2011). The impact of sexual abuse on female development: Lessons from a multigenerational, longitudinal research study. *Development and Psychopathology*, *23*(02), 453-476.

Woodward, S. H., Arsenault, N. J., Voelker, K., Nguyen, T., Lynch, J., Skultety, K., & Sheikh, J. I. (2009). Autonomic activation during sleep in posttraumatic stress disorder and panic: a mattress actigraphic study. *Biological psychiatry*, *66*(1), 41-46.

Woodward, S. H., Kaloupek, D. G., Schaer, M., Martinez, C., & Eliez, S. (2008). Right anterior cingulate cortical volume covaries with respiratory sinus arrhythmia magnitude in combat veterans. *Journal of Rehabilitation Research & Development*, *45*(3), 451-464.