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Heart Rate Variability Following Treatment for PTSD: Testing the Polyvagal Theory

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Acronyms

Acronym	Full Name
HRV	Heart rate variability
HR	Heart rate
RSA	Respiratory sinus arrhythmia
ECG	Electrocardiogram
F	High frequency
LF	Low frequency
ANS	Autonomic nervous system
SNS	Sympathetic nervous system
PNS	Parasympathetic nervous system
PTSD	Posttraumatic stress disorder
SA	Sino-atrial
DMNX	Dorsal motor nucleus
NA	Nucleus ambiguus
CPT	Cognitive Processing Therapy
ssmCPT	sleep and symptom monitoring plus CPT
hypCPT	Sleep-directed hypnosis plus CPT
SDNN	Standard deviation of normal-to-normal RR intervals
RMSSD	Root mean square of successive RR interval differences
CAPS	Clinician Administered PTSD Scale
DSM	Diagnostic and Statistical Manual of Mental Disorders
SAS-SR	Social Adjustment Scale-Self Report
BDI	Beck Depression Inventory
QOLI	Quality of Life Inventory

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Abstract

Posttraumatic stress disorder (PTSD) has been linked to lower heart rate variability (HRV), including measures of vagal tone. Treatments targeting the autonomic nervous system (ANS) have demonstrated efficacy in improving vagal tone, but it is less clear whether similar effects can also be achieved with cognitive therapies. The polyvagal theory has suggested that symptoms of social dysfunction are linked to vagal tone through a phylogenetically organized response to stress. HRV was collected during rest, reactivity (exposure to personalized trauma scripts), and recovery using a scripted imagery paradigm in female PTSD positive physical and sexual assault survivors (N = 41) prior to and following completion of cognitive processing therapy (CPT). Effects of treatment response and a supplementary sleep-directed hypnosis treatment were also assessed. To test the premise that vagal tone is related to social functioning, regressions predicting scores on Social Adjustment Scale (SAS) with high frequency (HF) HRV were completed. Vagal tone during trauma cue exposure improved following CPT, but only in treatment responders; during all other assessment periods it decreased posttreatment and there was no effect of treatment response. However, including depression symptoms as a covariate rendered all previously significant effects non-significant. The supplementary treatment had no effect on HRV during any of the measurement periods. Findings indicate the potential for cognitive therapies to impact vagal tone, despite not directly targeting the ANS. Minimal support for the polyvagal theory was found in the extended family subscale of the SAS, which was the only domain to demonstrate a significant relationship to vagal tone.

Keywords: heart rate variability, vagal tone, posttraumatic stress disorder, polyvagal theory, cognitive processing therapy, social adjustment scale

Heart Rate Variability Following Treatment for PTSD: Testing the Polyvagal Theory

Traumatic stress is not a new concept and indeed reactions to trauma have been described throughout history (Gersons & Carlier, 1992). However, these reactions were only organized into a formal diagnosis of posttraumatic stress disorder (PTSD) in the 1980s (American Psychiatric Association [APA], 1980). PTSD is a pervasive psychological disorder characterized by symptoms of intrusive thoughts, avoidance of trauma cues, negative alterations in cognitions and mood and arousal and reactivity (APA, 2013). Once thought to be relatively rare and limited to extreme trauma, like the experience of war, epidemiological evidence suggests that 6.8% of adults in the U.S. experience PTSD over their lifetime and 3.6% within the past year (Kessler et al., 2005). PTSD symptoms significantly impact the lives of those with the diagnosis and have been linked to increased physical health issues (Gupta, 2013) and functional impairment (Amaya-Jackson et al., 1999), as well as increased health care costs (Walker et al., 2003). One important aspect of functional impairment faced by those with PTSD is impairment in social functioning (Olatunji et al., 2007), which can significantly impact multiple aspects of daily life, including work and personal relationships.

In addition to psychological symptoms, PTSD is characterized by alterations in the autonomic nervous system (ANS) assessed using psychophysiology techniques (Cohen et al., 2000). Heart rate variability (HRV), or the beat-to beat variation in cardiac activity, is one such technique that measures activity of the nervous system, including sympathetic nervous system (SNS) and parasympathetic nervous system (PNS) activity (Laborde et al., 2017). The polyvagal theory links nervous system activity to social

functioning and posits that the vagal system is divided into two distinct systems, one of which must be active in order to socially engage. It further suggests that this activity can be measured using HRV (Porges, 2001). This theory is gaining in popularity but is still lacking in experimental evidence.

The current study will examine the role of the ANS in recovery from PTSD as well as the relationship between social functioning and PTSD in a treatment-seeking sample of those with a PTSD diagnosis. Understanding the underlying mechanisms in the relationship between PTSD and social impairment is important for research that aims to improve current treatment approaches for PTSD. Just as trauma has existed throughout human history, it will likely always be part of the human experience and understanding how the brain and body respond to trauma and treatment is imperative in improving lives around the world.

Posttraumatic Stress Disorder

PTSD has been described by many names, including “soldier’s heart” and “shell shock”. It became a formal diagnosis in 1980 with the release of the third edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM-III; APA, 1980). Diagnostic criteria were revised in subsequent versions, most recently in 2013 with the release of DSM-5 (APA, 2013). In the fourth edition (DSM-IV-TR; APA, 1994), a PTSD diagnosis required experiencing, witnessing, or being confronted with an event that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others, as well a response of fear, helplessness or horror. The most recent revision in DSM-5 removed the fear, helplessness or horror criterion to better capture trauma responses that may be more varied in presentation. In DSM-IV symptoms were

divided into three clusters: re-experiencing, avoidance and numbing, and hyperarousal. In DSM-5, the avoidance and numbing cluster was split into separate clusters, the numbing cluster was renamed Cognitions and Mood, and additional symptoms were added. PTSD was also moved from the Anxiety Disorder class to a new class labeled Trauma- and Stressor-Related Disorders. A dissociative subtype, requiring persistent depersonalization and derealization symptoms in response to the traumatic event was added in response to an accumulating body of literature that supports a sub-group of those with PTSD presenting with high dissociative symptoms and distinct symptom profiles that differ from those without the sub-type (for review see Hansen et al., 2017).

PTSD Treatment

Traditionally, cognitive-behavioral treatment for PTSD has focused on changing dysfunctional thought patterns associated with a person's trauma. Trauma-Focused Cognitive Behavioral Therapy, Prolonged Exposure and Cognitive Processing Therapy (CPT) are all effective treatments that facilitate re-structuring of cognitive processes around a trauma (Resick et al., 2002). While both psychotherapy and psychopharmacology result in physiological changes, greater effect sizes are typically seen in psychotherapy (Watts et al., 2013). Decreases in heart rate (HR) reactivity to trauma cues and startle reactions have been demonstrated following successful completion of treatment (Blanchard et al., 2002; Griffin et al., 2012).

While cognition focused therapies are effective, a significant proportion of patients either do not improve with these treatments or drop out before completion (Bisson et al., 2013; Resick et al., 2008; Schottenbauer et al., 2008; van Minnen et al., 2002). The possibility that physiological parameters can be ameliorated with

psychological treatment and vice versa, has sparked interest in applying a bottom-up approach to target psychological dysfunction, and research evaluating the efficacy of biofeedback and other breathing retraining treatments (yoga, meditations etc.) has yielded promising results. Meditation and breathing-focused yoga are effective in increasing HRV in non-clinical samples (Bernardi et al., 2001; Krygier et al., 2013). In people with PTSD, similar treatments have been effective in normalizing autonomic regulation as measured by increases in HRV (Tan et al., 2011; Tan et al., 2013) and reducing overall PTSD symptoms (van der Kolk et al., 2014). The precise mechanisms underlying these improvements are not largely understood and require additional empirical support.

PTSD and Social Functioning

Impairment in social functioning is a significant daily problem faced by those with PTSD. The DSM identifies as part of the diagnostic criteria for most disorders, including PTSD, impairment in social, occupational, or other important areas of functioning (APA, 2013). This inability to meaningfully engage with others can pervade nearly all aspects of an individual's life. PTSD has been linked to social impairment in several areas, including work or school (Olatunji et al., 2007; Solomon & Mikulincer, 2007), marriage and relationships (Amaya-Jackson et al., 1999; Schnurr et al., 2009), and social and family relationships (Norman et al., 2007). Social dysfunction has also been identified in those with subthreshold PTSD, suggesting it may be important to understand how social function relates to specific symptoms (Stein et al., 1997). Social support has been identified as a protective factor in developing PTSD (Brewin et al., 2000) and is most effective when the time since the trauma has been longer, suggesting that its effects may be cumulative (Ozer et al., 2003).

Shnaider et al. (2014) investigated the relationship between specific domains of social functioning and PTSD symptom clusters following CPT. They found that improvement in overall PTSD symptoms was related to all domains of social functioning but that the relationship between symptom clusters and specific domains of social functioning differed. Hyperarousal symptoms were related to outcomes in overall, daily living, and household chores but improvements in emotional numbing symptoms were related to outcomes in the nonfamily relationship domain. These findings suggests that PTSD symptoms are differentially related to social functioning. The ability to socially connect with others is critical to the happiness of humans and is key to achieving a substantial quality of life. The underlying causes of these impairments in those with PTSD are still unclear and require further investigation. However, if this area of impairment can be addressed, the quality of life of those living with this disorder could be significantly improved.

Heart Rate Variability

Early History

While HRV is a relatively new measure of cardiac activity, it was borne from the long history of psychophysiology, dating as far back as 300 BC, with written descriptions of pulse by Greek physician and scientist Herophilos; HR was later linked to health by Galen of Pergamon circa 170 AD, setting the stage for scientists to uncover the complex interactions between cardiac function and disease, and later, mental health (Billman, 2011). Across the next two centuries, knowledge and technology pertaining to cardiovascular health advanced, culminating in the first recorded evidence of respiratory sinus arrhythmia (RSA) in 1847 by Carl Ludwig. He observed in dogs that cardiac

activity was related to respiration and that pulse became quicker during inspiration and slowed during expiration (Billman, 2011).

With the development of the electrocardiogram (ECG) in the late 1800s and early 1900s, small beat-to-beat variations of the heart were observed for the first time (Billman, 2011). As HR was traditionally viewed as a consistent, stable measure, these fluctuations were first thought to be experimental artifacts resulting from imperfect collection and analysis methods. However, with further technological and analytical advances, consistent patterns involving the beat-to-beat variation began to emerge, leading to the conclusion that normal HR is not constant but, indeed, fluctuates from beat-to-beat (Porges & Byrne, 1992; Shaffer et al., 2014). This beat-to-beat fluctuation was termed HRV and is described by Quintana et al. (2016) as “the complex modification of the heart rate by the coordination of autonomic, respiratory, circulatory, endocrine and mechanical influences over time" (para. 1).

Recent Advances in HRV

Because advances in technology were needed for accurate collection, research using HRV is relatively young, with most studies taking place within the last forty years (Porges, 2007). Over this time, the body of HRV research has grown quickly and several techniques and measures have been developed. Initially HRV was measured simply, using a ruler and the ECG trace. As the research developed, multiple patterns within the ECG tracing overlaying each other were identified. Techniques were developed to isolate these separate rhythms and in the 1970s power spectrum analysis was developed, separating each frequency component (Billman, 2011). The frequency components were isolated and divided into high frequency (HF), low frequency (LF), very low frequency,

and ultra-low frequency bands. Measures are now broadly categorized into time and frequency domains.

Connecting Cardiac Control to Neurological Circuits

Following acceptance of HRV as an observable phenomenon, researchers set out to identify the controlling mechanisms. Despite an early proposal of a brain-heart connection by Darwin (1872/2002), early physiological researchers treated HR control as a "vegetative" system not connected to higher order brain processes (Darrow et al., 1942). However, it has subsequently become apparent that the ANS, including cardiac activation, responds to direct inputs from the cranial nerves that control parasympathetic activation and that this input is modulated by pathways from cortical and subcortical areas, including brain regions involved in emotional and cognitive processes (Porges & Byrne, 1992).

Some of the first evidence of this brain-heart connection came from observation of the orienting reflex in cognitive psychology, in which HR was seen to decrease during orientation toward a stimulus (Porges, 2011). In the 1960s and 70s, the bi-directionality of the central and peripheral nervous systems was an important element in the Laceys' seminal psychophysiological research on the connection between the brain and heart (Lacey & Lacey, 1978) as well as the Sokolov model of psychophysiology (Sokolov, 1963). The Sokolov model includes discussion of efferent pathways and feedback loops within the ANS as well as interactions between the ANS and psychological processes. It is now accepted that most ANS processes, including HR regulation, are the sum of these neural and peripheral inputs. A growing body of research in neurocardiology has

established that HR has a complex system of inputs including SNS and PNS input which interacts with the heart's own intrinsic nervous system (Figure 1; Shaffer et al., 2014).

Because early psychophysiological research was focused on the stress response in the laboratory, the majority of studies focused on linking the SNS to alterations in stress response within clinical populations with very little attention paid to the role of the PNS (Pole, 2007). This focus on sympathetic activation led researchers to focus on HR, blood pressure, electrodermal skin conductance, and facial electromyography, all measures thought to be related to SNS activation. There is growing evidence, however, that the PNS plays an important role in the stress response as well. The association between HR and PTSD has been shown to be more robust than that of skin conductance and PTSD (Pole, 2007). As skin conductance is regulated exclusively by the SNS (Langley, 1891) while HR is regulated by both the SNS and PNS, this is evidence that the PNS plays an important role in stress response and PTSD.

Further evidence for the role of the PNS in the stress response is seen in HRV research. It has been established that HR patterns are influenced by the PNS via the vagus nerve, also known as cranial nerve X (Jose & Collison, 1970), with evidence for vagal control of the heart found in examination of the sino-atrial (SA) node, which sits above the right atrium and serves as the pacemaker of the heart (Shaffer et al., 2014). The same study found that the rhythm induced by the SA node is intrinsically faster than the rate maintained by the PNS. The vagus nerve, which serves as the primary nerve of the PNS, has been identified as being responsible for the slowing of this rhythm. This control of HR through the vagus nerve has been termed "vagal tone" (See Figure 1; Laborde et al., 2017). This vagal action on the heart is immediate but transient (Shaffer et al., 2014). The

vagus nerve releases acetylcholine which acts on muscarinic acetylcholine receptors. While these effects are near instantaneous, they are brief, as the SA node is rich in acetylcholinesterase and quickly hydrolyzes acetylcholine, terminating its neuroactive effect (Task Force of the European Society of Cardiology the North American Society of Pacing Electrophysiology [Task Force], 1996). Despite the fact that the PNS has the stronger influence on the heart, the SNS also exerts some control of the SA node, mediating longer-lasting changes with the release of epinephrine and norepinephrine (Task Force, 1996).

While the vagal system's influences on HRV are significant, cardiac control is complex and there are additional important factors that contribute to HRV. HRV has been shown to have both central and peripheral influences, including mechanical pulmonary stretch receptors which allow breathing rhythms to correspond with HR (Billman, 2011). Additionally, there is an intrinsic nervous system within the heart that sends information from chemosensory and mechanical afferents to the brain (Shaffer et al., 2014). Researchers have found that afferent nerves play a larger role in vagal regulation of HR than initially believed, with 85-95% of vagal fibers being afferent (Shaffer et al., 2014). These afferents receive information from peripheral organs, including the heart, as well as regulatory brain areas like the amygdala. It is now clear that control of HR and HRV involve a complex feedback system, including the SNS and PNS to adapt and respond to a person's environment.

Measuring HRV

Normal HR is represented on an ECG with a series of waves, each corresponding to a specific cardiac event (Shaffer et al., 2014). Theoretically, time-domain measures of

HRV represent the time between SA-node action potentials. The closest measurable activity to this is atrial depolarization measured from P-wave to P-wave. However, because the identification of this wave is difficult due to its small amplitude and high signal-to-noise ratio, ventricular depolarization is typically used in its place, measured from R-peak to R-peak (Tarvainen et al., 2017); this distance is defined as the heart period (See Figure 2). In early studies, this distance was measured with a ruler and plotted vertically to represent beat-to-beat variation (Billman, 2011). Now, sophisticated algorithms, which automatically detect and plot R-R intervals, can more precisely identify and measure heart period, allowing for more advanced methods of analysis to develop (See Figure 3; Task Force, 1996).

Separating Components of HRV to Reflect PNS Function

Using spectral analysis, several frequency components have been separated and each component linked to its own physiological processes. The HF component has been consistently linked to the PNS through neural efferent vagal activity. The physiological correlates of the LF component are less clear. It was initially thought that the SNS was the driving force for this component but laboratory evidence supports a link between the LF band and baroreceptor activity while at rest, which receives input from a combination of SNS and PNS inputs (See Table 1; Shaffer et al., 2014; Task Force, 1996). RSA has also been identified as an index of vagal tone and is closely related to HF. They are synonymous when respiration falls within HF limits and are often used interchangeably (Billman, 2011). To avoid confusion, recent recommendations suggest using HF-HRV when referring to vagal tone and RSA only when specifically discussing HR changes during respiration (Laborde et al., 2017).

HRV in the Clinical Setting

Lower HRV is associated with worse cognitive and psychological outcomes, including anxiety and affective disorders (Friedman & Thayer, 1998). Higher HRV is related to better attentional and emotional control, and low vagal tone to worse self-regulation and a reduction in behavioral flexibility (Porges & Byrne, 1992; Thayer & Lane, 2000). HR pattern (i.e., HRV) has been shown to be important to health independent of HR (Porges & Byrne, 1992). A healthy heart responds quickly to its environment; HRV provides a good index of the flexibility of this system. While Eppinger and Hess were the first to suggest a connection between HRV and clinical status in 1915 (Billman, 2011), laboratory evidence of this relationship was discovered in the 1960s when fetal stress was found to be related to lower HRV in infants (Hon & Lee, 1965). In the 1970s, reduced HRV was linked to mortality following myocardial infarction (Wolf et al., 1978) and lower HRV was used to identify autonomic neuropathy in diabetic patients (Ewing et al., 1985). Subsequently, higher HRV has been consistently linked to neonatal health as well as many health outcomes in adults (Porges & Byrne, 1992), including diabetes, hypertension, and heart failure (Billman, 2011). While higher HRV during resting states is associated with better health outcomes, HRV is dynamic and should also decrease during challenge to prepare for stress in a healthy individual. However, if a stressor requires higher cognitive function, decreasing HRV may be maladaptive as the person will have less cognitive control (Laborde et al., 2017).

HRV in PTSD

Psychophysiology of PTSD

When introduced as a formal disorder in 1980 in the DSM-III (APA, 1980) PTSD was met with skepticism and confusion from many in the public (Gersons & Carlier, 1992). In an effort to legitimize the disorder, researchers attempted to identify objective physiological measures that could discriminate between those with and without the disorder, such as HR, blood pressure, skin conductance, and facial electromyography (Pitman et al., 1990; Pitman et al., 1987). These measures were assessed at rest as well as in reaction to general and trauma specific cues. Many researchers have found strong links between physiological measures and PTSD (Pole, 2007), correctly classifying the majority of participants as having PTSD or not in some cases with 100% accuracy (Keane et al., 1987). In these studies, however, specificity was higher than sensitivity, sometimes resulting in the misclassification of as many as 40% of participants who did not show the expected hyperreactivity to cues (Pole, 2007). This inconsistency in findings has led researchers away from identifying a definitive physiological diagnostic measure.

While the use of physiological measures to establish a diagnosis has been largely abandoned, there are, nevertheless, established consistent relationships between physiological measures and PTSD (Blanchard et al., 1994; Pole, 2007; Shalev & Rogel-Fuchs, 1993). An important meta-analysis found that elevated HR (whether resting, in response to startle sounds, or in response to standardized and idiographic cues) was one of the most reliable physiological measures linked to PTSD (Pole, 2007). More recent research continues to support the relationship between HR and PTSD (Lee et al., 2020; Morris et al., 2016).

The dissociative subtype included in DSM-5 has been linked to a high PTSD severity, increased incidence of early life trauma, and increased levels of comorbid disorders, as well as a different physiological and neurological profile in response to trauma cues (van Huijstee & Vermetten, 2018). While typical reactions to trauma cues in PTSD include increased HR, lower prefrontal and increased amygdala activation, those with the dissociative subtype exhibit the opposite profile, indicating suppressed autonomic activation (van Huijstee & Vermetten, 2018).

Decreased HRV in PTSD

While early psychophysiological research in PTSD focused on SNS activation, more recent research has shifted focus to the role of the PNS in regulation of psychological and physiological functioning. HRV is a current measure of focus in PTSD as it reflects the activity of the PNS and SNS. Several factors, including genetic, developmental, and experiential contribute to the functionality of the nervous system (Quintana & Heathers, 2014). Any assault on this system, such as experiencing a traumatic event and the subsequent development of PTSD, impacts these systems and may result in dysfunction in the ANS which may be identified using HRV.

Alterations in the nervous system, as measured by HRV, have been documented in those with PTSD. HRV research has indicated that primary modulation of the heart has shifted from the PNS to the SNS in those with PTSD, who show decreased HRV compared to controls both during rest (Cohen et al., 2000; Liddell et al., 2016; Tan et al., 2011) and in response to trauma cues (Keary et al., 2009; Sack et al., 2004) as well as in animals following traumatic stress exposure (Cohen et al., 2007). Interestingly, in this animal study, HR returned to near recovery levels more quickly than HRV, suggesting

that HRV is a more sensitive index for persistent ANS dysfunction. Evidence for autonomic dysfunction is further supported in studies comparing HF and LF ratios (Hauschildt et al., 2011) and relating HRV and HR. The most consistent findings are reduced HF-HRV and RSA in those with PTSD when compared to non-trauma-exposed controls. This hypoactivation of the parasympathetic system may make a person more likely to develop PTSD (Minassian et al., 2015). Decreased HRV has been observed in those with subthreshold PTSD as well (Hauschildt et al., 2011), suggesting the relationship between decreased HRV and PTSD may be related more to PTSD symptoms that the diagnosis itself.

HRV and PTSD Symptoms

PTSD is partially characterized by symptoms related to cognitive inflexibility, including intrusive thoughts and avoidance. Cognitive inflexibility has been linked to lower HRV and diminished neural control of the prefrontal cortex on subcortical structures in healthy individuals (for review see Ottaviani, 2018). Deficits in inhibitory control are the most reliable domain of cognitive deficits in those with PTSD (Gillie & Thayer, 2014). This study also found that relationships between other types of cognitive control (i.e., sustained attention and set shifting) and PTSD are mixed. The inability to inhibit unwanted thoughts and memories leads to chronic avoidance, an inflexible reaction to painful or traumatic reminders judged as unacceptable and unendurable (Kashdan & Rottenberg, 2010). This dysfunctional approach precludes any processing or restructuring of thoughts surrounding the event and may be key in the development and maintenance of the disorder. The decreased HRV seen in PTSD may work through maintenance of re-experiencing symptoms; studies in healthy individuals have

established that those with decreased HRV have more difficulty controlling intrusive thoughts (Gillie & Thayer, 2014). The inability to inhibit unwanted thoughts and memories, as measured by lowered HRV, may serve as a pre-trauma risk factor for later PTSD diagnosis.

The Polyvagal Theory

Theories Linking HRV to Psychological Outcomes

Several theories have been put forth linking HRV and psychopathology. While most of these theories generally focus on vagal tone, each has a different emphasis. The neurovisceral integration model links HRV with attentional and emotional control through the central autonomic network, an interconnected group of central and peripheral nervous system structures that control visceromotor, neuroendocrine, and behavioral responses (Thayer & Lane, 2000). Other models connecting HRV to physiological and emotional outcomes focus on specific aspects of this relationship. The resonance frequency model (Lehrer, 2013) focuses on the relationship between breathing and HRV. It proposes that when an individual achieves a "resonance frequency" maximum efficiency in respiratory gas exchange is achieved, increasing oxygenation of tissues, and allowing for better homeostatic maintenance. The author posits that this frequency can be trained with paced breathing, improving HRV and thus regulation of emotion (Lehrer, 2013). Similarly, the psychophysiological coherence model suggests that HRV acts as an index of physiological coherence, or harmony, of systems working across the body to create emotional, social, and global well-being (McCraty & Childre, 2010) while the biological behavioral model focuses on the vagal system in coordinating respiratory and cardiovascular systems to provide energy resources (Grossman & Taylor, 2007).

One theory that has received much attention recently is the polyvagal theory which seeks to explain the relationship between social engagement and regulation of the ANS by the brain using HRV (Porges, 2001). This theory is appealing because it provides a framework for connecting the neural activation of the vagal system to a multitude of behaviors, including social engagement, during the stress response. While this theory has been gaining in popularity, there are still aspects of the theory that are unclear and lack support. The theory has not been sufficiently tested, especially in clinical populations and if this theory is to serve as the framework for further study, more experimental testing is necessary to validate its premises.

Current Mammalian Vagal System

In humans the most broadly reaching of the cranial nerves is the vagus nerve, reflected in its name which derives from the Latin root for "wandering". The vagus nerve contains both afferent and efferent pathways, although the majority (80%) are afferent. The efferent pathways include motor and parasympathetic branches. The vagus nerve arises in the medulla from two clusters of rootlets: the nucleus ambiguus (NA) and dorsal motor nucleus (DMNX). The dorsomedial medulla houses the DMNX while the NA is found ventral to the DMNX in the ventrolateral ventricular formation. These rootlets merge and exit the cranial fossa at the jugular foramen. Branches then spread to innervate multiple visceral targets. While both the NA and DMNX have projections above and below the diaphragm, the majority of the NA pathways are supra-diaphragmatic and the majority of the DMNX pathways are sub-diaphragmatic. One large branch of the NA innervates the muscles of the larynx and pharynx helping to control phonation and swallowing. It also projects to the heart, bronchi, esophagus and soft palate with only the

rostral portion projecting below the diaphragm (Monkhouse, 2006). This face-heart connection links cardiac activity to social expression.

The majority of pathways from the DMNX project below the diaphragm innervating the muscles and glands of the gut, including the stomach and intestines. The DMNX also has projections above the diaphragm projecting to the heart. Sensory afferents from the abdominal viscera, lungs, heart, larynx, and pharynx all terminate at the nucleus tractus solitarius (NTS) also located in the medulla. The NTS projects to both the NA and DMNX, serving as a feedback loop for the vagal system (Monkhouse, 2006).

While both the NA and DMNX have projections to the heart, the majority of these projections are from the NA. Using a retrograde tracer injected into the AV ganglion, researchers found that the NA contained two-thirds more labeled cells than the DMNX, suggesting a larger influence of the NA on the slowing of the heart via the AV ganglion. Also, the type of fibers projecting to the heart from these two nuclei differ. Neurons from the NA that have cardioinhibitory control are fast B fibers while those projecting from the DMNX contain both B fibers and slower C fibers (Porges, 2011).

Evolution of Separate Vagal Systems

It is thought that the differentiation of the vagal system into two distinct nuclei occurred as a result of the need for behavioral strategies in response to novel stimuli accompanied by the high oxygen requirement of mammals. Later, the development of a neural face-heart connection facilitated social interaction as an adaptive survival strategy (Porges, 2011). Even some, more primitive, reptile species show this early separation of the vagal nuclei. Some reptiles (i.e., lizards and crocodiles) have complete separation of these systems, while others (i.e., turtles) retain some connection between the two nuclei

(Barbas-Henry & Lohman, 1984). Reptiles respond to novel stimuli with freezing of motor activity and orienting to outside stimuli. This wait-and-watch approach is accompanied by bradycardia and is an effective strategy for reptiles because of the lower metabolic demands compared to mammals. By contrast, mammals first orient in response to novel stimuli, but follow with either sustained attention or social engagement. Therefore, when faced with novel stimuli, reptiles maintain high engagement of the more primitive DMNX while mammals may first engage the DMNX but quickly shift to higher activation of the NA and a less DMNX engagement (Porges, 2011).

Origin of the PVT: The Vagal Paradox

The polyvagal theory was born from the so-called vagal paradox, an anomaly first observed in infants. High vagal tone was observed in healthy full-term infants, in contrast to the low vagal tone showed by premature infants at discharge, suggesting that vagal tone could function as an index of health in neonates. It was noted that while high vagal tone was protective when occurring in conjunction with RSA[1], it was lethal when paired with bradycardia (significant slowing of the heart that can lead to death) in infants (Porges, 2011). This apparent inconsistency was the first evidence for the existence of two separate vagal sources, each with distinct effects on the nervous system.

The vagal paradox first observed in infants was further observed in other samples. In adults, increased vagal tone can produce neurogenic bradycardia, while decreased vagal tone decreases RSA. However, bradycardia has occurred during suppression of RSA. This finding led to the first premise of the polyvagal theory; neurogenic bradycardia and RSA are controlled by two separate parts of the brain. The theory

[1] While the current convention is to only use the term RSA when discussing HR changes directly related to respiration, the authors of the polyvagal theory use this term throughout their published works. Therefore, in an effort to accurately reflect their theories, “RSA” is used in background discussion of these works.

postulates that neurogenic bradycardia arises from the DMNX while RSA is controlled by the NA (Porges, 2007).

In addition to the vagal paradox, further evidence for two separate vagal origins comes from research on the relationship between HR and RSA. While HR and RSA sometimes covary together (Billman & Dujardin, 1990), independent responses of HR and RSA are observed when examining responses to inhalant anesthesia such that RSA becomes depressed without any accompanying changes in HR (Donchin et al., 1985). The relationship between RSA and HR also appears to depend on the state of alertness, as the two measures correlate more closely when a person is alert compared to when they are drowsy or sleeping (Porges, 2011).

Nervous System Response to Threat

The polyvagal theory is guided by the principle that humans have retained three separate mechanisms, two of which are vagal, to regulate nervous system response to threat. These systems (from newest to oldest) are the vagal system originating from the NA, the sympathetic adrenal system, and the vagal system originating from the DMNX. These systems follow the Jacksonian principle of dissolution (Jackson, 1958), meaning that the systems are engaged in a phylogenetic hierarchy (beginning with the newest) and that if the first approach is unsuccessful, the organism will engage the second and third until a successful strategy is found. In order to engage the newer systems, the older systems must be inhibited (Porges, 2007). The vagal-NA system is thought to be responsible for social engagement and is the phylogenetically newest system to develop. Thus, if social engagement is not effective against a threat, then the sympathetic adrenal system (the active system that will enable the traditional fight or flight behaviors) will be

engaged. If both of these systems fail to address the threat, then the vagal DMNX (the phylogenetically oldest system) will be employed. Engagement of this system will enable freezing or fainting.

The detection of threat plays a key role in the employment of these systems. This detection and employment of the appropriate brain circuit has been termed "neuroception" (Porges, 2007). A key component of the polyvagal theory, neuroception refers to the process that takes place unconsciously in more primitive parts of the brain to determine the level of risk or threat present in the environment. In order to socially engage with another organism, the risk must be evaluated as low and the brain circuits regulating the defensive behaviors must be inhibited. When the process of neuroception is disrupted, as in psychopathology, the threat may be mislabeled as more or less dangerous than it should be and a maladaptive strategy along with inappropriate systems may be employed leading to behaviors that may be destructive or cause long-term distress (Porges, 2011).

Strengths and Weaknesses of the Polyvagal Theory

Support for the polyvagal theory has been found in multiple disorders. A negative relationship has been identified between HRV and self-reports of depression (Hauschildt et al., 2011). Lower vagal tone, measured using HRV, has been recorded in those with disorders involving symptoms of lack of emotion control and impulsivity, including borderline personality disorder (Austin et al., 2007) and schizophrenia (Clamor et al., 2018) compared to controls. Austin and colleagues' finding that lower vagal tone is found in those with borderline personality disorder, characterized by impulsivity and difficulty controlling anger, is consistent with the polyvagal theory's premise that disengagement of

the social engagement system and activation of the SNS is compatible with fight or flight behaviors.

Although there is support for the polyvagal theory, there are also criticisms. Grossman and Taylor (2007) challenge the premise that RSA always reflects vagal tone as well as some of the evolutionary assertions made by the theory. They counter that there are multiple influences on RSA and that, contrary to the polyvagal theory, other vertebrates besides mammals also demonstrate a connection between breathing and HR. The debate over the utility of this theory is ongoing and more evidence across disciplines is necessary to understand whether the assertions made by the polyvagal theory provide an accurate and complete framework for the nervous system's response to threat and potential disruptions to this system.

Current Study

Primary Aims and Hypotheses

Improvements in HRV have been linked to treatments that target the ANS such as yoga or biofeedback through a substantial body of literature. However, there is less research on cognitive-focused treatments and their potential impacts on vagal tone. The current study's design provides a unique opportunity to evaluate HRV following CPT and will contribute to understanding the role vagal tone may play in the development and recovery from traumatic stress. It will also assess the contributions of a supplementary sleep-directed hypnosis component to treatment outcomes.

While the polyvagal theory has been developing in the psychophysiology literature and is now gaining attention in the field of stress and trauma, the majority of the literature remains theoretical and requires evaluation in an experimental setting. The

current study will assess the theory's premise that there is a relationship between vagal tone and social functioning.

Aim 1: test whether autonomic nervous system function as measured by HRV is improved by CPT. Additionally, the current study will test whether the addition of a sleep-directed hypnosis treatment to CPT has any additional effect on HRV and whether potential posttreatment changes in HRV are related to successful remission of PTSD symptoms. The relationships between HRV and specific PTSD symptom clusters will be assessed, and the role of depressive symptoms in this relationship will also be tested.

Hypothesis 1a. All HRV measures will significantly increase following completion of CPT.

Hypothesis 1b. Participants who completed sleep-directed hypnosis prior to CPT will have a significantly larger increase in HRV compared to those who did not. Additionally, it is hypothesized that those whose PTSD has successfully remitted will have a significantly larger increase in HRV compared to those who did not fully remit.

Aim 2: test whether social functioning changes following CPT, whether potential changes are related to treatment response, and whether social functioning is related to vagal tone, thus testing one of the polyvagal theory’s premises. To do this, the relationship between HF-HRV and overall social functioning as well as separate social functioning domains will be assessed before and after CPT. The relationship between changes in HRV and changes in social functioning following treatment will also be assessed. Follow-up analyses will assess the role that vagal tone may play in the relationship between PTSD symptoms and social functioning.

Hypothesis 2a. Participant's social functioning will improve following CPT and those whose PTSD has remitted will have greater improvements.

Hypothesis 2b. HF-HRV will be significantly related to social functioning at pre- and post-treatment, such that higher HF-HRV will be associated with higher social functioning and

changes in HF-HRV will be significantly related to changes in social functioning, such that as participants' HF-HRV increases, self-reported overall social functioning will improve.

Secondary Aims

To further assess the relationship between HRV and treatment outcomes, secondary aims will assess HRV's relationship to sleep and quality of life.

Aim 3: test whether sleep impairment changes following CPT, whether potential changes are related to treatment response and whether sleep impairment is related to HF-HRV in those with PTSD.

Hypothesis 3. Sleep will significantly improve following CPT and those whose PTSD has remitted will have greater improvements. It is also hypothesized that sleep will be related to HF-HRV such that greater sleep impairment will be associated with lower HF-HRV.

Aim 4: test whether quality of life changes following CPT, whether potential changes are related to treatment response and whether quality of life is related to HF-HRV in those with PTSD.

Hypothesis 4. Quality of life will significantly improve following CPT and those whose PTSD has remitted will have greater improvements. It is also hypothesized that

quality of life and HF-HRV will be significantly related such that higher quality of life will be related to higher HRV.

Method

Participants

Participants were 90 female, PTSD-positive physical and/or sexual assault survivors taken from a larger study examining sleep-targeted treatment for PTSD (Galovski et al., 2016). Inclusion criteria included significant sleep impairment defined by scoring at least 3 on D-1 sleep impairment symptom on the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995), at least three months posttrauma, and being stable on any psychotropic medication for at least one month. Exclusion criteria included psychosis, intellectual disability, active suicidality, parasuicidality, current drug or alcohol dependence, ongoing abuse, or stalking. Previous therapy (excluding CPT) and concurrent therapies (excluding trauma or sleep-focused) were permitted. Throughout treatment, participants were asked to limit alcohol use to 14 servings per week, with no more than five servings per day, and to limit caffeine consumption to 500 mg/day with no caffeine consumption after 6:00 pm. Participants were also asked to maintain consistent bedtimes and rise times within 1 hour. For the current study, an intent-to-treat sample approach was used. Therefore, those that dropped out of treatment but returned for follow-up physiological assessments were included in analyses.

Measures

Demographic Questionnaire and Standardized Trauma Interview

A structured interview created locally and previously used (Resick et al., 2008) was completed, which collected demographic information, information about the assault, prior trauma exposure, and treatment history.

PTSD Assessment: Clinician Administered PTSD Scale

The Clinician Administered PTSD Scale (CAPS; Blake et al., 1995) is a 22-item clinician administered scale used to assess the presence of PTSD according to the symptoms listed in the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition-Text Revision (DSM-IV; APA, 2000). Intensity and frequency for each symptom are rated on a 0 to 4 scale and scores are summed for a total severity score. The CAPS has demonstrated high internal consistency across all three clusters (α = .87-.88) and on the 17 core items (α = .95; Weathers et al., 2001). In the current sample, reliability was fair for the reexperiencing (α = .79), avoidance (α = .74), and numbing (α = .71) clusters and poor in the hyperarousal (α = .53) cluster at pretreatment. At posttreatment reliability was good for the reexperiencing (α = .88), numbing (α = .89), and hyperarousal (α = .81) clusters and fair in the avoidance (α = .79) cluster. The F1, I2 rule was used to determine whether a participant met criteria for each symptom. Because data were collected prior to the release of DSM-5, DSM-IV criteria were used to diagnose PTSD. However, the core criteria across both are the same and results will still be relevant for those diagnosed using the new DSM-5 criteria.

Social Behavior: Social Adjustment Scale-Self Report

The Social Adjustment Scale-Self Report (SAS-SR; Weissman & Bothwell, 1976) is a 54 item self-report scale, modified from the Social Adjustment Interview (Weissman & Paykel, 1974), measuring functioning in the following areas: work, student, housework, social/leisure, extended family, marital, parental, family unit and economic functioning. Items are rated from 1 to 5 with higher scores indicating greater impairment. Items are averaged for an overall score. Good validity for the SAS-SR has been established, with the self-report demonstrating excellent agreement with the interview version (r = .72, p < .001; Weissman & Bothwell, 1976) and normative data across multiple disorders is available (Weissman et al., 1978). In the current sample, reliability for the SAS-SR was excellent at pretreatment (α = .97) and fair at posttreatment (α = .79).

Depression Symptoms: Beck Depression Inventory-II

The Beck Depression Inventory-II (BDI-II; Beck et al., 1996) is a 21-item self-report assessment of depressive symptoms using DSM-IV criteria (APA, 2000). Symptoms are rated on a 4-point scale (0 to 3) and summed for a total severity score. The BDI-II has been shown to have good internal consistency (α = .9) and retest reliability (alpha = .73 - .96; Wang & Gorenstein, 2013). In the current sample, reliability for the BDI was good at pretreatment (α = .89) and excellent at posttreatment (α = .97).

Sleep Quality: Pittsburg Sleep Quality Index

The Pittsburg Sleep Quality Index (PSQI; Buysse et al., 1989) consists of 19 self-rated questions and 5 bed-partner or roommate (if available) rated questions assessing several aspects of sleep. Self-rated questions are rated from 0 (no difficulty) to 3 (severe difficulty) and are combined to create seven sleep component scores: subjective sleep

quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. These sub scores are summed for a global sleep quality score which may range from 0 to 21. A score of > 5 has been shown to indicate presence sleep disturbance. The PSQI has demonstrated high test-retest reliability (r = .87) and good validity (Backhaus et al., 2002). In the current sample, internal consistency was acceptable at pretreatment (α = .60) and good at posttreatment (α = .80).

Life Satisfaction: Quality of Life Inventory

The Quality of Life Inventory QOLI (QOLI; Frisch, 1994) is a 32-item self-report measure which assesses life satisfaction across 16 different domains. For each domain, importance to personal happiness is rated from 0 (*not important*) to 2 (*extremely important*) and current satisfaction from -3 (*very dissatisfied*) to +3 (*very satisfied)*. The QOLI has demonstrated reliability (r = .80 - .91) and internal consistency (α = .77-.89) across multiple samples (Frisch et al., 1992). In the current sample, reliability for the BDI was good at pretreatment (α = .81) and poor at posttreatment (α = .04).

Physiological Acquisition

Scripted Imagery Paradigm

A script-driven imagery paradigm created by Lang et al. (1983) to investigate emotional networks and adapted to study physiological reactivity in PTSD (Pitman et al., 1987) was presented to participants as a re-exposure to trauma cues while HRV was measured. Six 30-second pre-recorded scripts were created, which participants listened to via headphones. Two neutral scripts (sitting in a lawn chair on a porch, looking out a window on an autumn day), one positive script (a trip to the beach), one general fear

script (public speaking), and two personalized trauma scripts generated from a written trauma narrative were administered Pitman et al. (1987). A written trauma narrative, which was completed before physiological data collection, was used to create the personalized trauma scripts. A list of potential physical responses was provided to participants and the recordings included those identified by the participant as experienced during the traumatic event. Audio recordings were created in the second person, present tense and trauma narrative was split into two 30-second segments. Scripts were presented consecutively in the following order: neutral, general fear, first idiographic trauma script, neutral, second idiographic trauma script, positive. A four-minute resting baseline HRV preceded script presentations and each assessment consisted of four measurement periods: baseline, script presentation, imagery (participant was asked to imagine the scene just described), and recovery (participant was told to relax and stop imagining the scene; See Figure 4). All periods except the recovery following trauma scripts were 30 seconds; the recovery periods following trauma scripts were increased to one minute to allow for a more complete recovery.

HRV Assessment Periods and Duration

The current study followed recommendations to assess the "3 R's:" rest, reactivity, and recovery (Laborde et al., 2017). This included the initial 2 minutes of rest prior to any scripts, reactivity to trauma cue exposure, and recovery from trauma cue exposure. For reactivity both script presentation and imagery periods were combined for a total of 2 minutes of trauma cue exposure. Recovery periods following script presentation were combined with rest times preceding the subsequent script and combined across trauma scripts for a total of 3 minutes of recovery (See Table 2). This

meets or exceeds recommendations for recording duration for low and high frequency, respectively (Laborde et al., 2017; Task Force, 1996).

HRV Collection and Analysis

R-R Interval Collection

A Coulbourn Instruments modular system high gain bio amplifier (S75-01) was used to collect HR, sampled at 500 Hz with silver/silver chloride electrodes (SilveReez, Clinical Instruments Inc., Littleton, CO) attached to the left wrist and right ankle, and the ground lead to the right wrist. Calculation of the inter-best interval was achieved by converting the raw ECG signal into beats per minute by detection of the R-wave peak amplitude.

Data Pre-Processing and Cleaning

HRV processing was completed using Kubios software (Tarvainen et al., 2014). Following guidelines set by Task Force (1996), Kubios employs a custom QRS detection algorithm based on the Pan-Tompkins algorithm (Pan & Tompkins, 1985). This algorithm includes preprocessing, in which data are bandpass filtered to reduce noise, squared to emphasize peaks, and moving average filtered to smooth close-by peaks. Decision rules of amplitude threshold and expected time between adjacent R-waves were then applied, adapting with each new R-wave detection. Time resolution is achieved by up-sampling data to 2000 Hz using interpolation. The distance between identified R-peaks was then transformed into an R-R interval time series. To address the inherent non-stationarity of the R-R series, the data were detrended using a smoothness-priors approach (Tarvainen et al., 2002).

Artifact Identification and Correction

Per recommendations (Laborde et al., 2017; Task Force, 1996), artifacts in R-peak detection were first corrected manually, followed by interpolation. The ECG was examined for any irregularities in the QRS component of the ECG waveform to identify abnormal beats not generated by the sinus node. ECG is subject to several types of artifact, including technical and physiological (for a complete list of potential artifact types see Shaffer and Combatalade (2013). Any non-sinus node depolarizations were removed. Care was taken to not over-edit the data as recommended by Porges and Byrne (1992) in order to maintain the integrity of the data and identify potentially critical patterns. Following manual removal of artifact, a cubic spline interpolation was applied to the data. This has been identified as an effective interpolation method (Quintana et al., 2016). If greater than 5% of data were corrected for a period, that participant was removed from analyses involving that period (see Figure 5).

HRV Analysis

Time-domain measures, including the standard deviation of normal-to-normal RR intervals (SDNN), a measure of overall HRV, and the root mean square of successive RR interval differences (RMSSD), a measure of vagal tone, were used in addition to frequency domain measures (HF-HRV and LF-HRV) to assess changes pre to posttreatment (See Table 3). Measures were chosen per recommendations by (Task Force, 1996), for short-term recordings. Measures of both time and frequency domains were included for completeness with emphasis on frequency domains, as the physiological interpretation of these measures is clearer and are preferred for short-term

recordings (Task Force, 1996). Any non-normally distributed parameters were transformed using a natural log transformation.

Per recommendations by Laborde et al. (2017), frequency domain analyses were conducted using autoregressive modeling with the order of the model at $p = 16$ (Boardman et al., 2002). Per terminology recommendations (Laborde et al., 2017), HF-HRV, as opposed to RSA, was used to refer to HR changes occurring from .15 to .40 Hz, as the current study's focus was vagal tone and not changes during respiration. Respiration was not controlled but was examined and those whose respiration was outside 9 and 24 cycles per minute (.15 - .40 Hz) were removed for analyses (see Figure 5).

Procedure

All protocols were approved by the Institutional Review Board at the University of Missouri-St. Louis. Assessment was completed over two days. On day one, participants completed the CAPS, trauma narrative, and self-report measures. Participants returned one-week later for the physiological assessment. Following this initial assessment, participants were randomly assigned to either the sleep and symptom monitoring plus CPT (ssmCPT; $n = 48$) group or the sleep-directed hypnosis plus CPT (hypCPT; $n = 42$) group. Two weeks following completion of treatment, participants completed the same two-day assessment. A 3-month follow-up was also completed, consisting only of clinical assessments; physiological assessment was not included.

Treatment Conditions

Sleep and Symptom Monitoring Plus Cognitive Processing Therapy

Participants randomized into the sleep and symptom monitoring plus Cognitive Processing Therapy (ssmCPT) group completed daily assessments of PTSD, depressive symptoms, and sleep for three weeks. They also completed weekly phone checks to assess symptoms and need for emergency care. Participants were encouraged to contact their therapists as needed. This group allowed for control of potential symptom improvement associated with passage of time and expectations of improvement stemming from contact with a therapist. Following three weeks of symptom monitoring, participants completed 12 weeks of CPT.

Sleep-Directed Hypnosis Plus Cognitive Processing Therapy

Participants assigned to the sleep-directed hypnosis plus Cognitive Processing Therapy (hypCPT) group completed daily monitoring of PTSD, depressive, and sleep symptoms similar to the ssmCPT group. Instead of weekly calls, however, this group completed weekly 60-minute sessions of sleep-directed hypnosis for three weeks. The hypnosis sessions targeted sleep onset and difficulty falling asleep following mid-sleep awakenings. The first session consisted of a brief psychoeducation component about the negative effects of sleep loss followed by scripted guidance into a hypnotic trance through eye fixation and progressive muscle relaxation. Guided imagery and ego-strengthening statements were used to achieve relaxation and confidence around sleep capabilities. The treatment attempted to target arousal and anxiety surrounding sleep through increased relaxation and self-efficacy. Participants were instructed to practice this procedure throughout the week. Sessions two and three were nearly identical to the

first, with time for troubleshooting of challenges faced at home in place of the psychoeducation component. Participants were given an audiotape of the sessions for at-home use. For a complete description of this treatment see Galovski et al. (2016).

Cognitive Processing Therapy

Following three weeks of either symptom monitoring (ssmCPT) or sleep-directed hypnosis training (hypCPT) all participants completed 12 weeks of CPT. Sessions were held once a week for 60-minutes and completed individually. CPT is a cognitive therapy targeting maladaptive thoughts and belief patterns that typically develop following a trauma (Resick et al., 2010). CPT has been found to be an effective treatment for PTSD (Resick et al., 2002).

Data Analytic Plan

Preliminary analyses

SPSS version 26 was used to complete all analyses. First, participants were categorized into treatment response groups. A non-responder was defined as anyone who still retained a diagnosis of PTSD posttreatment, was short one symptom in only one cluster, or had a CAPS total of greater than 45 (Griffin et al., 2012; Orr, 1997). All others were categorized as responders. To test whether PTSD symptoms changed following treatment and the validity of the creation treatment response groups, 2 x 2 mixed ANOVAs were run with time (pre and posttreatment) entered as a within-subject variable and treatment response (responder vs non-responder) entered as a between-subject variable with CAPS total and individual cluster scores entered as outcome variables in separate ANOVAs. Simple main effects analyses were completed for significant interactions.

Primary Analyses

Data Analysis Aim 1. To test whether HRV is improved with treatment, the potential role of the addition of the sleep-directed hypnosis component, and whether there is a relationship between potential changes in HRV and PTSD treatment response, 2 x 2 x 2 mixed factorial analyses of variance (ANOVA) were run with time (pre- and post-CPT treatment), treatment condition (ssmCPT and hypCPT) and PTSD treatment response (responder vs non-responder) entered as independent variables. Separate analyses were completed with each HRV measure (HF, LF, SDNN, and RMSSD) within each measurement period (rest, reactivity, and recovery) as the outcome variable. This analysis design allows for assessment of changes in vagal tone, as well as whether these changes are exclusive to vagal tone or are seen in HRV measures associated with other systems (e.g., SNS and overall HRV).

To examine the role depression may play in HRV, analyses of variance (ANCOVA) tests will also be completed mirroring the tests above with depression added as a covariate. Only significant factors from previous analyses will be included in these follow-up analyses. To further assess the importance of depression on HRV, participants were separatee into depression response groups with those scoring at or above 14 on the BDI categorized as having at least mild depression and those below as no depression. These groups were then used to complete 2 x 2 mixed ANOVAs similar to those with PTSD treatment response groups with time (pre and posttreatment) as a within-subject variable and depressive symptom group (depression vs no depression) as between-subject variables and each HRV measure (HF, LF, SDNN, and RMSSD) within each measurement period (rest, reactivity, and recovery) as the outcome variable.

Follow-up analyses included multiple hierarchical linear regressions with HRV predicting PTSD symptom clusters. Consistent with previous research (Shnaider et al., 2014), symptoms were divided into four clusters to better reflect recent changes in diagnostic criteria for DSM-5 and allow for a more sensitive analysis.

Data Analysis Aim 2. Aim 2 will test the premise of the polyvagal theory that HRV is related to social functioning. First, changes in social functioning posttreatment were assessed using 2 x 2 mixed ANOVAs, with time (pre and posttreatment) entered as a within-subject variable and treatment response (responder vs non-responder) entered as a between-subject variable. Separate ANOVAs were completed for SAS total score and subscale scores. Simple main-effects analyses were completed for significant interactions.

The original study design consisted of cross-panel analyses to assess the relation of pre to post HF-HRV and SAS scores as well as their trajectories simultaneously. While this would have been the most parsimonious option, because panel analysis is a type of structural equation modeling, it would have required more participants than were included in the current study to complete with confidence. Therefore, a more conservative approach of using regressions was adopted, using Shnaider et al. (2014) as a model for change analyses.

First, two separate linear regressions were run with HF-HRV predicting SAS score at pre and posttreatment. Next, a multiple hierarchical linear regression was conducted predicting posttreatment SAS scores with pretreatment SAS score entered at step 1 and residualized HF-HRV change score entered at step 2. These were completed at

rest, reactivity, and recovery periods for SAS variables that significantly changed posttreatment.

Follow-up analyses will mirror those above with only those who completed CPT without the added hypnosis component. These will serve as sensitivity analyses to ensure that results are not confounded by the additional treatment.

To further extend the work of Shnaider et al. (2014) and examine the relationship between PTSD symptoms, vagal tone and social functioning, hierarchical multiple regressions were run to examine the relationship between PTSD symptoms and overall social functioning as well as specific domains of functioning. Multiple hierarchical linear regressions were completed with pretreatment SAS score added at step 1, residualized CAPS change score added at step 2 and HF-HRV added as a third step predicting posttreatment SAS score.

Secondary Analyses

Data Analysis Aim 3. To assess changes in sleep posttreatment, a 2 x 2 mixed ANOVA was run with time (pre and posttreatment) entered as a within-subject variable and treatment response (responder vs non-responder) entered as a between-subject variable and PSQI total score as the outcome variable. Next, a similar approach to Aim 2 was used to explore whether changes in sleep were related to vagal tone. Two linear regressions were run with HF-HRV predicting PSQI score at pre and, separately, posttreatment. Next, a multiple hierarchical linear regression with pretreatment PSQI score entered at step 1 and residualized HF-HRV change score entered at step 2 predicting posttreatment PSQI score. These were completed at rest, reactivity, and recovery periods.

Data Analysis Aim 4. To assess changes in quality of life posttreatment and whether these potential changes are related to vagal tone, aim 3 analyses will be repeated with QOLI scores in place of PSQI scores.

Power

Because of the ability to control for individual differences, (Quintana & Heathers, 2014) identify within-subjects design as ideal for HRV studies. Adequate sample sizes for HRV have been investigated by researchers and are still an ongoing discussion in the literature. Quintana (2017) suggests that for group comparisons, a sample size of 61 to detect a medium effect size at 80% power, for case-control studies but acknowledge that this may not be appropriate for pre-post studies. Pinna et al. (2007) examined necessary sample sizes for within-subjects designs and stressed that the necessary sample size depends on the reliability of the HRV index of interest and whether breathing was spontaneous or paced. Using the convention of a 30% change pre to post-test as the effect size, power of 80%, and alpha of .05, they found evidence that the necessary samples sizes for HRV parameters to achieve good reliability when allowing spontaneous breathing, were between 31 and 57 for the variables of interest in the current study (SDNN = 40, RMSSD = 57, LF-AR = 49, and HF-AR = 31).

Results

Participant Attrition

Of the 90 participants assessed in the parent study for inclusion in the current study, 42 did not complete physiological assessment at posttreatment. This included those who dropped out of treatment (n = 20) and those who completed treatment but chose not to complete the posttreatment physiological assessment (n = 22). For the 48 who did

complete physiological assessment at both pre and posttreatment, HRV data were assessed for artifact and respiration violations (see Method section for details). Outlier assessments were then completed using visual and statistical methods and severe outliers identified. Incidents of artifact, respiration, and outlier violations were removed from the rest (n = 10), reactivity (n = 7), and recovery (n = 8) periods (see Figure 5). The final sample included 37 treatment completers and 4 dropouts. Analyses were completed both with and without dropouts and inclusion of dropouts did not change the results of any analysis; therefore all analyses reported below include dropouts.

Demographic Analysis

Of those with valid HRV data in at least one assessment period, 23 reported being White (56%) and 18 African American (44%). None reported being Hispanic. Age ranged from 19 to 70 (M = 39.56, SD = 12.89) with 78% of participants reporting being single, divorced, separated, or widowed, and 22% married or living with someone. On average, participants reported completing 14.61 (SD = 2.97) years of education and most (63%) reported an income of less than $20,000 per year.

Data Screening

After extreme outliers were removed, HRV variables were assessed for normality. Skewness and kurtosis were examined, with anything beyond ±1.00 considered violating normality, as well as Shapiro-Wilk's test of normality evaluated at alpha < .001. All HRV variables violated normality assumptions and were thus log transformed. After transformation, skewness and kurtosis were all within acceptable limits and normality tests were non-significant. All results are reported in natural log units. For a summary of HRV variables in non-transformed, absolute units see Table 4.

Treatment Response Based upon CAPS Scores

Of the participants with valid pre and posttreatment HRV, 30 were categorized as treatment responders and 11 as non-responders (defined as anyone who still retains a diagnosis of PTSD or is short one symptom in only one cluster or has a CAPS total of greater than 45). Responder groups did not differ on reported sleep medication use at pre ($F(1,38) = .92$, $p = .343$,) or posttreatment ($F(1,39) = .01$, $p = .946$). To verify the validity of response groups and assess PTSD symptom changes pre to posttreatment, 2 x 2 mixed design ANOVAs were completed with time (pre and posttreatment) entered as a within-subject variable and treatment response (responder vs non-responder) entered as a between-subject variable. Total CAPS scores and each individual cluster score were entered in separate ANOVAs. The main effect of time was significant for total CAPS ($F(1, 39) = 166.51$, $p < .001$, partial $\eta^2 = .81$), reexperiencing ($F(1, 39) = 88.15$, $p < .001$, partial $\eta^2 = .69$), avoidance ($F(1, 39) = 120.23$, $p < .001$, partial $\eta^2 = .76$), emotional numbing ($F(1, 39) = 42.85$, $p < .001$, partial $\eta^2 = .52$), and hyperarousal ($F(1, 39) = 46.97$, $p < .001$, partial $\eta^2 = .55$) with all CAPS scores decreasing posttreatment. The main effect of treatment response was also significant for total CAPS ($F(1, 39) =32.68$, $p < .001$, partial $\eta^2 = .46$), reexperiencing ($F(1, 39) =4.42$, $p = .042$, partial $\eta^2 = .10$), avoidance ($F(1, 39) = 17.90$, $p < .001$, partial $\eta^2 = .32$), emotional numbing ($F(1, 39) = 33.04$, $p < .001$, partial $\eta^2 = .46$), and hyperarousal ($F(1, 39) = 21.78$, $p < .001$, partial $\eta^2 = .36$) with non-responders reporting higher scores.

In addition, the Time x Treatment Response interaction was significant for total CAPS ($F(1, 39) = 36.88$, $p < .001$, partial $\eta^2 = .49$), reexperiencing ($F(1, 39) = 12.58$, $p = .001$, partial $\eta^2 = .24$), avoidance ($F(1,39) = 15.13$, $p < .001$, partial $\eta^2 = .28$), emotional

numbing ($F(1, 39) = 16.68$, $p < .001$, partial $\eta^2 = .30$), and hyperarousal ($F(1, 39) = 12.03$, $p = .001$, partial $\eta^2 = .24$), (see Table 5a). To further explore the interactions, simple main effects were examined (see Table 5b). Total CAPS scores significantly decreased posttreatment in both the responder ($F(1, 29) = 335.57$, $p < .001$, partial $\eta^2 = .90$) and non-responder ($F(1, 10) = 15.94$, $p < .001$, partial $\eta^2 = .29$) groups. In the responder group, all individual cluster scores also significantly decreased: reexperiencing ($F(1, 29) = 155.91$, $p < .001$, partial $\eta^2 = .80$), avoidance ($F(1, 29) = 205.60$, $p < .001$, partial $\eta^2 = .84$), numbing ($F(1, 29) = 105.29$, $p < .001$, partial $\eta^2 = .73$), and hyperarousal ($F(1, 29) = 99.28$, $p < .001$, partial $\eta^2 = .72$). In the non-responder group, scores in the reexperiencing ($F(1, 10) = 11.66$, $p = .002$, partial $\eta^2 = .23$) and avoidance ($F(1, 10) = 17.10$, $p < .001$, partial $\eta^2 = .31$) clusters significantly decreased, while numbing ($F(1, 10) = 2.07$, $p = .158$, partial $\eta^2 = .05$), and hyperarousal ($F(1, 10) = 3.92$, $p = .055$, partial $\eta^2 = .09$) scores did not significantly change. While both groups' scores decreased at posttreatment across most CAPS measures (excluding the numbing and hyperarousal clusters), the responder group showed a significantly greater decrease than the non-responder group. In the case of the numbing and hyperarousal clusters, the responder group's score significantly decreased posttreatment while the non-responder group's score did not significantly change.

At pretreatment there were no significant differences between the two groups' total CAPS score or individual cluster scores (p-range:.135-.893). At posttreatment, however, the response groups differed significantly in total CAPS ($F(1, 40) = 90.14$, $p < .001$, partial $\eta^2 = .70$) and all cluster scores: reexperiencing ($F(1, 40) = 23.61$, $p < .001$, partial $\eta^2 = .38$), avoidance ($F(1, 40) = 44.87$, $p < .001$, partial $\eta^2 = .54$), numbing ($F(1,$

40) = 57.83, $p < .001$, partial $\eta^2 = .60$), and hyperarousal ($F(1, 40) = 31.08$, $p < .001$, partial $\eta^2 = .44$). These findings validate the separation of PTSD treatment response groups.

Aim 1: HRV and PTSD Treatment

HRV, Treatment Group, and Treatment Response

To address the question of whether HRV improved with treatment and the potential roles of treatment condition and treatment response, 2 x 2 x 2 mixed design ANOVAs were completed with time (pre and posttreatment), treatment condition (ssmCPT and hypCPT) and PTSD treatment response (responder vs non-responder) entered as independent variables. Separate analyses were completed with each HRV measure (HF, LF, SDNN, and RMSSD) within each measurement period (rest, reactivity, and recovery) as the outcome variable. Box's and Levene's tests were examined for each analysis to assess equality of covariance matrices and variances, respectively. All were non-significant, indicating the data do not violate assumptions of equality of covariance matrices or equality of variances for the dependent variable.

Rest Period

In the rest period, the main effect of time was significant for HF ($F(1, 34) = 7.05$, $p = .012$, partial $\eta^2 = .17$), LF ($F(1, 34) = 4.13$, $p = .050$, partial $\eta^2 = .11$), SDNN ($F(1, 34) = 6.70$, $p = .014$, partial $\eta^2 = .17$), and RMSSD ($F(1, 34) = 6.10$, $p = .019$, partial $\eta^2 = .15$) with all HRV variables decreasing posttreatment. Between-subjects main effects of treatment response (*p*-range: .561 - .900) and treatment condition (*p*-range: .653-.961) were not significant. Additionally, the Time x Response (*p*-range: .098-.358), Time x Condition (*p*-range: .366-.547), Response x Condition (*p*-range: .691-.997), and Time x

Response x Condition (*p*-range: .194-.846) interaction effects were all not significant (see Table 6a). In the rest period, all HRV measures decreased posttreatment and neither treatment condition nor treatment response had any effect on HRV changes.

Reactivity Period

In the reactivity period, the main effect of time was significant only in the LF ($F(1, 37) = 11.20$, $p = .002$, partial $\eta^2 = .23$) analysis, with HRV decreasing posttreatment. The Time x Treatment Response interaction effect was significant for HF ($F(1, 37) = 5.17$, $p = .029$, partial $\eta^2 = .12$), LF ($F(1, 37) = 4.76$, $p = .036$, partial $\eta^2 = .11$), SDNN ($F(1, 37) = 6.15$, $p = .018$, partial $\eta^2 = .14$) and RMSSD ($F(1, 37) = 4.77$, $p = .035$, partial $\eta^2 = .11$). In the HF, SDNN, and RMSSD analyses, the treatment responder group's HRV increased post-treatment (*M*-change range: .01-.42) while the non-responder group decreased (*M*-change range: -.48 to -.09). In the case of LF-HRV, the treatment responder group (*M*-change = -.26) decreased less than the non-responder group (*M*-change = -.85). No other main effects or interactions were significant. See Table 6b for complete results.

Simple main effects were examined for the significant Time x Treatment Response interaction effects in the reactivity period (see Table 6c and Figure 6). Notably, the treatment responder groups were not significantly different at pretreatment (*p*-range: .331-.472) or posttreatment (*p*-range: .375-.644) across any HRV measures. In the HF ($F(1, 29) = 4.70$, $p = .037$, partial $\eta^2 = .02$) and RMSSD ($F(1, 29) = 4.30$, $p = .045$, partial $\eta^2 = .10$) analyses, the responder groups' HRV significantly increased posttrauma while there were no significant changes in the non-responder group in either HF ($F(1, 10) = 1.84$, $p = .183$, partial $\eta^2 = .05$ or RMSSD: ($F(1, 10) = 1.71$, $p = .200$, partial $\eta^2 = .04$.

Conversely, the non-responder group's HRV significantly decreased in the LF ($F(1, 10) = 10.34$, $p = .003$, partial $\eta^2 = .22$) and SDNN ($F(1, 10) = 6.65$, $p = .014$, partial $\eta^2 = .15$) analyses while the responder group's HRV did not significantly change (LF: $F(1, 29) = 1.30$, $p = .261$, partial $\eta^2 = .03$; SDNN: $F(1, 29) = .26$, $p = .612$, partial $\eta^2 = .01$). In the reactivity period, treatment response had a significant effect on HRV changes such that treatment responders had better outcomes compared to non-responders across all HRV variables. The best outcomes were seen in vagal tone measures (HF-HRV and RMSSD) with treatment responders' HRV increasing and non-responders' decreasing. In RMSSD and LF-HRV, treatment responders' HRV did not change while non-responders' HRV decreased. Treatment condition had no effect on posttreatment HRV changes.

Recovery Period

The main effect of time was significant in the LF analysis ($F(1, 36) = 4.83$, $p = .034$, partial $\eta^2 = .12$) such that HRV decreased posttreatment but was not significant for any other HRV variables (*p*-range: .059-.545). The main effects of treatment response (*p*-range: .545-.965) and treatment condition (*p*-range: .453-.892) were not significant for any of the HRV variables. No interaction effects were significant in any of the HRV analyses during the recovery period (See Table 6d). In the recovery period, the only posttreatment changes were that LF-HRV decreased posttreatment. Neither treatment condition nor treatment response had any effect on HRV changes.

Depressive Symptoms and HRV

To test whether depressive symptoms are related to HRV changes following treatment, two types of analyses were run. First, a 2 x 2 mixed ANOVA was run with time (pre and posttreatment) and treatment response (responder and non-responder)

entered as independent variables and posttreatment BDI score entered as a covariate. Separate analyses were run with each HRV measure (HF, LF,SDNN,RMSSD) at each measurement period (rest, reactivity, and recovery) as the dependent variable. Treatment condition was not included since it was not a significant factor in previous analyses. With the BDI scores entered into the model, none of the significant findings noted remained significant. There were no significant correlations between posttreatment BDI and HRV measures (*p*-range: .141-.995).

Second, a two-way mixed design ANOVA was completed with time (pre and posttreatment) as a within-subject variable and depressive symptom group (depression vs no depression) as between-subject variables and each HRV measure (HF, LF, SDNN, and RMSSD) within each measurement period (rest, reactivity, and recovery) as the outcome variable. Depression groups were defined using a cutoff of 14 on the BDI. This cutoff separates those with mild and above depression (*n* = 14-15) from those without depression (*n* = 23-26).

The main effect of time in the rest period was significant for the HF ($F(1, 35) = 6.15$, $p = .018$, partial $\eta^2 = .15$), SDNN ($F(1, 35) = 6.08$, $p = .019$, partial $\eta^2 = .15$), and RMSSD ($F(1, 35) = 5.08$, $p = .031$, partial $\eta^2 = .13$) analyses such that HRV decreased posttreatment. Unlike the PTSD responder analyses, time was not significant in the LF analysis ($F(1, 35) = 3.00$, $p = .092$, partial $\eta^2 = .08$). Neither the main effect of depression status (*p*-range: .515-.896) nor the interaction of time and depression status (*p*-range: .128-.565) were significant in any of the HRV analyses for the rest period.

In the reactivity period, the main effect of time was significant only in the LF analysis ($F(1, 39) = 10.32$, $p = .003$, partial $\eta^2 = .21$), with HRV decreasing posttreatment

and the main effect of depression status was not significant for any analysis (*p*-range: .204-.408). The Time x Depression Status interaction was significant for HF ($F(1, 39) = 7.06$, $p = .011$, partial $\eta^2 = .15$), SDNN ($F(1, 39) = 8.26$, $p = .007$, partial $\eta^2 = .18$), and RMSSD ($F(1, 37) = 5.32$, $p = .027$, partial $\eta^2 = .12$) with the no depression groups' HRV increasing and the depression group decreasing posttreatment. While not meeting criteria for significance in the LF analysis, the interaction was marginally significant ($F(1, 39) = 3.66$, $p = .063$, partial $\eta^2 = .09$) with the no depression group decreasing less than the depression group.

The recovery period was notably different from the PTSD treatment response analyses. The main effect of time was significant only in the LF ($F(1,38) = 7.03$, $p = .012$, partial $\eta^2 = .16$) and SDNN ($F(1,38) = 6.21$, $p = .017$, partial $\eta^2 = .14$) analyses, such that HRV decreased posttreatment. The main effect of depression status was not significant for any of the analyses (*p*-range: .241-.290). While no interactions within the recovery period were significant in the initial PTSD responder analyses, the Time x Depression Group interaction effect was significant for the HF ($F(1, 38) = 5.68$, $p = .022$, partial $\eta^2 = .13$) and RMSSD ($F(1, 38) = 4.30$, $p = .045$, partial $\eta^2 = .10$) analyses with the no depression group's HRV increasing and the depression group decreasing posttreatment. The interaction in the SDNN analysis was marginally significant ($F(1, 38) = 3.82$, $p = .058$, partial $\eta^2 = .09$) with the no depression group decreasing less than the depression group (see Table 7), while in the LF analysis, it was not significant ($F(1, 38) = 1.09$, $p = .302$, partial $\eta^2 = .03$).

Overall, the rest and reactivity periods were similar to PTSD treatment responder analyses, while the recovery period had a noticeably different pattern. Whereas there

were no effects of treatment response in the PTSD analyses in the recovery period, in the depression analyses, those with no depression improved on vagal tone measures (RMSSD and HF-HRV) posttreatment while the depression group did not.

HRV and CAPS Clusters

To further examine the relationship between HRV and PTSD symptoms, multiple hierarchical linear regressions were completed with the respective pretreatment HRV measure entered at step 1 and the four posttreatment CAPS cluster scores entered simultaneously at step 2 to predict posttreatment HRV. Separate regressions were completed for all HRV measures only within the reactivity period, as those demonstrated a significant Time x Treatment Response interaction in previous analyses. Multicollinearity was assessed for each regression by examining tolerance and variance inflation factors (VIF) with .01 and 10 used as cutoffs, respectively. No variables demonstrated a multicollinearity problem.

In the HF analysis, multiple *R* was statistically significant ($F(5, 35) = 8.54$, $p < .001$, $R^2\ adj = .49$) but only pretreatment HRV scores contributed significantly to the prediction model ($\beta = .67$, $p < .001$). None of the cluster scores significantly added to the prediction (|β|-range: .02-.75, *p*-range: .131-.928). Results in the LF analysis mirrored those above with a significant multiple *R* ($F(5, 35) = 9.76$, $p < .001$, $R^2\ adj = .52$) and pretreatment HRV as the only significant contributor to prediction ($\beta = .74$, $p < .001$). Again, cluster scores did not add any predictive value to the model (|β|-range: .05-.77, *p*-range: .296 - .780). The SDNN analysis was similar, with a significant prediction model ($F(5, 35) = 9.60$, $p < .001$, $R^2\ adj = .52$) and pretreatment HRV contributing significantly ($\beta = .71$, $p < .001$) while none of the CAPS cluster scores added significantly (|β|-range:

.06-.77, *p*-range: .182-.739). RMSSD followed the same pattern with a significant prediction model ($F(5, 35) = 8.24$, $p < .001$, R^2 *adj* = .48), pretreatment HRV contributing significantly ($\beta = .69$, $p < .001$), and CAPS cluster scores not significant ($|\beta|$-range: .01-.75, *p*-range: .247-.952). These findings suggest that the individual HRV measures tested do not relate to any specific CAPS cluster over the others.

Aim 2: HRV and Social Functioning

Social Functioning Scores and Treatment Response

To test the polyvagal theory's premise that vagal tone is related to social functioning, first changes in social functioning posttreatment were assessed using 2 x 2 mixed ANOVAs, with time (pre and posttreatment) entered as a within-subject variable and treatment response (responder vs non-responder) entered as a between-subject variable. SAS total score and subscale scores were entered into separate ANOVAs as the outcome variable. Because not all the SAS subscales were applicable to all participants (i.e., not everyone is a student or has a job outside of the home), the sample sizes for some of the measures were quite low (i.e., $n_{work} = 10$). To achieve maximum power, the nine subscales of the SAS were condensed into six: housework (unpaid), occupation (work and student), social/leisure, extended family, family unit (primary relationship, parental, and family unit), and economic. Data screening of SAS scores showed that both pre and post scores' skewness and kurtosis were within limits of ± 1.00 and Shapiro-Wilk's tests of normality were all non-significant.

The main effect of time was significant for overall ($F(1, 39) = 11.16$, $p = .002$, partial $\eta^2 = .22$), housework ($F(1, 35) = 5.80$, $p = .021$, partial $\eta^2 = .14$), and social/leisure ($F(1, 39) = 15.10$, $p < .001$, partial $\eta^2 = .28$) such that all scores decreased posttreatment

indicating an improvement in social functioning. The main effect for treatment response was significant for overall ($F(1, 39) = 11.63$, $p = .002$, partial $\eta^2 = .23$), social/leisure ($F(1, 39) = 25.90$, $p < .001$, partial $\eta^2 = .40$), and extended family ($F(1, 38) = 5.57$, $p = .023$, partial $\eta^2 = .13$) such that responders had lower scores than the non-responders (See Table 8a).

The Time x Treatment response interaction effect was significant for overall ($F(1, 39) = 9.57$, $p = .004$, partial $\eta^2 = .20$) and family unit ($F(1, 31) = 5.26$, $p = .029$, partial $\eta^2 = .15$). Simple main effects were run for overall, housework, social/leisure, extended family, and family unit to better identify areas of difference. Simple main effects revealed that in all cases, responders' SAS scores significantly decreased (p-range: < .001-.013) posttreatment while non-responders did not significantly change (p-range: .145-.886). Responder groups were not significantly different from each other at pretreatment (p-range: .143-.907) with the exception of social/leisure in which non-responders scored significantly higher at pretreatment than responders. At posttreatment, the responder groups were significantly different in the overall ($p < .001$), social/leisure ($p < .001$), and extended family scales ($p = .002$), while housework ($p = .099$) and family unit ($p = .064$) were marginally significant (See Table 8b). While no posttreatment changes were seen in family unit or economic functioning, treatment responders improved on overall, housework, occupation, social/leisure, and extended family functioning compared to non-responders who did not change.

HF-HRV Predicting Social Functioning

To assess the relationship between HF-HRV and social functioning, three regressions were completed for each SAS variable of interest. First, two separate linear

regressions were run with HF-HRV predicting SAS score at pre and posttreatment. Next, a multiple hierarchical linear regression was conducted predicting posttreatment SAS scores with pretreatment SAS score entered at step 1 and residualized HF-HRV change score entered at step 2. These were completed at rest, reactivity, and recovery periods for SAS variables that significantly changed posttreatment.

At the individual pre and posttreatment timepoints, HF-HRV did not significantly predict overall SAS score or any subscale. In all of the multiple regressions, pretreatment SAS score significantly predicted posttreatment SAS score (*p*-range: < .001-.045). In nearly all cases, HRV change did not predict posttreatment SAS scores. The only instance of HF-HRV as a significant addition to the model was in the extended family reactivity analysis. In this analysis, the multiple *R* was significant ($F(2, 37) = 5.22$, $p = .010$, $R^2 adj = .18$) and the addition of HF-HRV increased R^2 for the model by 13%, with both SAS pretreatment score ($\beta = .31$, $p = .039$) and HF-HRV ($\beta = -.36$, $p = .017$) significantly contributing to the prediction model. As SAS scores decreased, HF-HRV increased, suggesting that as one improves, so does the other. The only significant relationship between social functioning and HRV was between HF-HRV change during the reactivity period and the extended family subscale, providing minimal support for the polyvagal theory's premise.

Sensitivity Analyses: ssmCPT Group Only

The above analyses were also completed with only the ssmCPT group to ensure that the hypnosis component was not confounding the role HF-HRV may have on changes in social functioning. It should be noted, however, that because of the low sample size these should be interpreted with some caution. At pretreatment, HF-HRV

predicted family unit scores in the rest ($F(1, 15) = 11.38$, $p = .004$, $R^2 adj = .39$, $\beta = -.66$), reactivity ($F(1, 15) = 11.96$, $p = .004$, $R^2 adj = .41$, $\beta = -.66$), and recovery ($F(1, 15) = 14.12$, $p = .002$, $R^2 adj = .45$, $\beta = -.70$).

At posttreatment HF-HRV predicted extended family scores across all three of the assessment periods (rest: $F(1, 16) = 7.27$, $p = .016$, $R^2 adj = .27$, $\beta = -.57$; reactivity: $F(1, 17) = 5.74$, $p = .028$, $R^2 adj = .21$, $\beta = -.50$; recovery: $F(1, 17) = 6.39$, $p = .022$, $R^2 adj = .23$, $\beta = -.52$). Also, in the extended family subscale change score analyses, pretreatment SAS scores did not significantly predict posttreatment scores, but HF-HRV was a significant addition in all measurement periods. In the rest period, the multiple *R* was significant ($F(2, 15) = 9.89$, $p = .002$, $R^2 adj = .51$). While SAS pretreatment score did not add significantly to the model ($\beta = .29$, $p = .115$), HF-HRV did contribute significantly ($\beta = -.65$, $p = .002$). Results for the reactivity period analysis were similar with a significant model ($F(2, 16) = 6.76$, $p = .007$, $R^2 adj = .39$), SAS pretreatment score not contributing significantly ($\beta = .16$, $p = .407$) and HF-HRV adding significantly to the model ($\beta = -.62$, $p = .005$). The recovery period analysis also mirrored those above ($F(2, 16) = 4.28$, $p = .032$, $R^2 adj = .27$; SAS pretreatment SAS score: $\beta = .17$, $p = .416$, HF-HRV: $\beta = -.52$, $p = .023$). Results suggest that removal of those who completed the adjunct sleep-directed hypnosis treatment resulted in increased support for the polyvagal theory, although sample sizes were low-enough to warrant caution in interpretation.

CAPS Score as a Covariate

Shnaider et al. (2014) used multiple hierarchical regressions and residualized change scores to assess how changes in PTSD symptoms related to changes in social functioning. In order to replicate and extend their work, similar analyses were completed

with HRV added to the model to test whether it would be a significant addition. Multiple hierarchical linear regressions were conducted with pretreatment SAS score added at step 1, residualized CAPS change score added at step 2 and HF-HRV added as a third step predicting posttreatment SAS score. To avoid violation of collinearity assumptions, separate regressions were completed for each HRV assessment period (rest, reactivity, and recovery) for overall SAS scores as well as subscales that changed significantly posttreatment (housework, social/leisure, extended family, and family unit). In the overall SAS analysis, the multiple *R* for the rest period was significant ($F(3, 33) = 24.30$, $p < .001$, $R^2\ adj = .66$). SAS pretreatment score ($\beta = .50$, $p < .001$) and residualized CAPS score ($\beta = .60$, $p < .001$) were both significant predictors of posttreatment SAS scores. HF-HRV, however was not a significant contributor to the prediction model ($\beta = .08$, $p = .455$). Reactivity ($F(3, 37) = 27.68$, $p < .001$, $R^2\ adj = .67$) and recovery $F(3, 36) = 27.11$, $p < .001$, $R^2\ adj = .67$) periods were similar with SAS pretreatment score ($\beta = .51$ and .50, respectively, $p < .001$) and residualized CAPS score ($\beta = .57$ and .58, respectively, $p < .001$) both significantly adding to the prediction of posttreatment SAS score and HF-HRV not significantly adding to prediction ($\beta = .01$, $p = .956$ and $\beta = .05$, $p = .632$, respectively). Results were similar across all SAS subscales with pretreatment SAS (β-range: .28-.71, *p*-range: .002-.042) and total CAPS scores contributing significantly to the prediction of posttreatment SAS scores (β-range: .27-.55, *p*-range: < .001-.033, See Table 9). In all cases, lower CAPS scores predicted lower SAS scores. HF-HRV did not significantly contribute to prediction of any of the SAS subscale scores (|β|-range: .08-.20, *p*-range: .081-.545; See Table 9).

To complete the replication and assess whether changes in social functioning were related to changes in specific symptom clusters, the same regressions were completed with residualized CAPS clusters scores entered simultaneously at step 2 in place of total CAPS score. Because it was not a significant addition in the total CAPS analyses, HF-HRV was not added to the model for individual clusters. The multiple *R* was significant in the overall SAS analysis $F(5, 35) = 19.23$, $p < .001$, $R^2 adj = .70$. Pretreatment SAS score was a significant predictor ($\beta = .61$, $p < .001$), as were avoidance ($\beta = -.157$, $p = .027$), numbing ($\beta = .287$, $p = .017$), and hyperarousal ($\beta = .331$, $p = .011$), while reexperiencing was not a significant addition ($\beta = .42$, $p = .278$). In the subscale analyses, the prediction model was significant in all cases (*p*-range: < .001- .041) and pretreatment SAS scores were significant predictors for their respective subscale (|β|-range: .30-.70, *p*-range: < .001- .049). Numbing contributed significantly to prediction of social/leisure ($\beta = .266$, $p = .037$) and family unit ($\beta = .052$, $p = .009$), while hyperarousal was a marginally significant predictor of social/leisure ($\beta = .293$, $p = .053$). Reexperiencing and avoidance were not significant contributors to prediction of any posttreatment subscale. Overall, improvement in PTSD symptoms was related to improvement in social functioning, and the numbing cluster was most frequently linked to separate domains.

Aim 3: HF-HRV and Sleep Impairment

To assess changes in sleep posttreatment, a 2 x 2 mixed ANOVA was run with time (pre and posttreatment) entered as a within-subject variable and treatment response (responder vs non-responder) entered as a between-subject variable and PSQI total score as the outcome variable. PSQI pre and posttreatment scores were assessed for normality;

skewness and kurtosis were within ± 1.00 and Shapiro-Wilk's normality tests were non-significant, indicating no violation of normality. The main effect of time was the only significant effect, with sleep scores decreasing posttreatment ($F(1, 37) = 20.59$, $p < .001$, partial $\eta^2 = .359$). Neither the main effect of treatment response ($F(1, 37) = 2.57$, $p = .118$, partial $\eta^2 = .065$) nor the Time x Treatment Response effects ($F(1, 37) = 2.04$, $p = .162$, partial $\eta^2 = .052$) were significant.

To assess whether changes in HF-HRV are related to sleep, an approach similar to Aim 2 was employed. Two linear regressions were run with HF-HRV predicting PSQI score at pre and, separately, posttreatment. Next, a multiple hierarchical linear regression with pretreatment PSQI score entered at step 1 and residualized HF-HRV change score entered at step 2 predicting posttreatment PSQI score. These were completed at rest, reactivity, and recovery periods. HF-HRV did not significantly predict PSQI scores at pre or posttreatment for any of the assessment periods (*p*-range: .105-.963, |β|-range: .01-.27). Similarly, while pretreatment PSQI scores significantly predicted posttreatment scores in all cases (*p*-range: .005-.009, β-range: .422-.457), HF-HRV did not significantly contribute to the model in any of the assessment periods (*p*-range: .328-.934, β-range: -.013 to -.152). Overall, vagal tone was not related to sleep functioning.

Aim 4: HF-HRV and Quality of Life

To assess changes in quality-of-life posttreatment, a 2 x 2 mixed ANOVA was computed with time (pre and posttreatment) entered as a within-subject variable and treatment response (responder vs non-responder) entered as a between-subject variable to predict QOLI total score. QOLI pre and posttreatment scores were assessed for normality; skewness and kurtosis were within ± 1.00 and Shapiro-Wilk's normality tests

were non-significant, indicating no violation of normality. The main effect of time was significant with QOLI scores increasing posttreatment ($F(1, 38) = 10.06$, $p = .003$, partial $\eta^2 = .209$) as was the main effect of treatment response ($F(1, 38) = 9.50$, $p = .004$, partial $\eta^2 = .200$) with the responder group increasing significantly more than the non-responder group. The Time x Treatment Response effect was not significant ($F(1, 38) = .55$, $p = .463$, partial $\eta^2 = .014$).

To assess whether changes in HF-HRV are related to quality of life, two separate linear regressions were run with HF-HRV predicting QOLI score at pre and posttreatment. Next, a multiple hierarchical linear regression with pretreatment QOLI score entered at step 1 and residualized HF-HRV change score entered at step 2 predicting posttreatment QOLI score. These were completed at rest, reactivity, and recovery periods. HF-HRV did not significantly predict QOLI scores at pre or posttreatment at any of the assessment periods (p-range: .668-.935, $|\beta|$-range: .01-.07). Similarly, while pretreatment QOLI scores significantly predicted posttreatment scores in all cases ($p < .001$, β-range: .616 -.653), HF-HRV did not significantly contribute to the model in any of the assessment periods (p-range: .635-.994, $|\beta|$-range: .001 to .063). Overall, vagal tone was not related to quality of life.

Discussion

The current study assessed the potential changes in vagal tone during a scripted imagery paradigm following CPT and an adjunct relaxation treatment and tested the polyvagal theory's premise that vagal tone is related to social functioning. HRV did improve following treatment but was conditional on assessment period and treatment

response. Support for the polyvagal theory was minimal but there were some significant relationships between vagal tone and social functioning.

Aim 1: HRV and PTSD Treatment

The hypothesis that HRV would increase following CPT was partially supported: HRV did significantly increase after CPT completion, but only for vagal measures in the treatment responder group during the reactivity period. The dominant trend during rest and recovery was for participants' HRV to decrease posttreatment which is congruent with previous findings indicating a shift toward SNS cardiac control in PTSD (Liddell et al., 2016). During the reactivity period, during which participants were exposed to their personal trauma cues, changes in vagal tone (measured with HF and RMSSD) were apparent in those who responded to CPT while those whose PTSD symptoms did not remit saw no significant change. The other measures of overall HRV (SDNN) and dual PNS/SNS input (LF-HRV) still demonstrated differences between response groups but with a different pattern from the vagal tone measures: non-responders significantly decreased while responders did not significantly change (see Figure 6). While responders' overall and dual input measures did not increase with treatment, they did not decline like their non-responder counterparts. Because SDNN and LF include sympathetic inputs, which decreases HRV, this pattern still supports increased PNS activation in the responders. Taken together, these results suggest that the shift toward SNS cardiac control typically seen in PTSD was slowed or reversed for those whom CPT successfully decreased PTSD symptoms.

When faced with a physical or mental stressor that does not require executive function, a reduction in HRV (high level of vagal withdrawal) may be adaptive to

mobilize resources to prepare for a physical response. However, when executive function is required, it is beneficial to have high vagal tone allowing for cognitive flexibility as needed to respond to the environment (Laborde et al., 2017; Park & Thayer, 2014). CPT, based on Lang's information processing theory, targets the neural fear networks created during a trauma by identifying areas of challenge or "stuck points" and restructuring beliefs and thought patterns around the traumatic experience and trauma-related cues (Resick & Schnicke, 1992). This restructuring requires engagement with the trauma cue, and complex, higher-order processing of beliefs and how they fit into established schemas of belief and then processing evidence to potentially change those schemas. Thus, during CPT sessions, the process of engaging and modifying those neural networks introduces the skillset for increased cognitive flexibility moving forward. Thus, it is not surprising that those who were successful in treatment showed evidence of increased cognitive flexibility as measured by vagal tone during exposure to trauma cues. It is interesting to note that while CPT is a cognitively focused treatment, it was still able to improve vagal tone in response to trauma cues, a physiologically based process.

The fact that HRV did not change at rest or recovery periods, regardless of treatment response, may be indicative of the targeting of CPT, or the timing of assessment and the persistence of nervous system alterations in PTSD. Because participants were assessed directly following treatment, it is likely that while participants can increase vagal tone during trauma cue exposure in a deliberate but transient way, the nervous system may not have yet shifted back to a healthy sympathetic and parasympathetic balance. Because rest and recovery periods involve more autonomic

processing and less deliberate, executive functioning, improvements may not yet be evident during these periods.

Treatment Condition and HRV

The hypothesis that participants who completed sleep-directed hypnosis prior to CPT will have a significantly larger increase in HRV was not supported by any measure during any assessment period. While the finding that treatment condition did not have any effect on HRV was surprising, both treatment response and the small sample size may have obscured any real effect of hypnosis treatment. Despite the fact that previous researchers have linked meditation-like treatments to improvements in HRV (Krygier et al., 2013), the parent paper for this study found that the group that did not receive the additional hypnosis treatment caught up to the other group in most symptoms post-CPT (Galovski et al., 2016), so this may have been the case in the current study as well. Since we do not have a physiological assessment between the hypnosis treatment and CPT, the current study could not test whether the hypnosis component alone had any effect on HRV separate from CPT. It may be that HRV was improved by both modes of treatment, but any gains seen after the hypnosis component were matched with CPT and not measurably greater.

Depressive Symptoms and HRV

The inclusion of depression symptoms as a covariate rendered all previously significant effects non-existent in the reactivity period. Because depression symptoms overlap with PTSD symptoms, once depression is accounted for, there may not be enough variance within the groups left to detect differences. Using depression response as group definition, however, uncovered a notably different pattern of HRV response.

With this new grouping, reactivity findings were similar in that improvements in HRV during the reactivity period were related to response group. Depression status, however, appeared to be more closely related to vagal tone during the recovery period than PTSD response group. Vagal tone measures (HF and RMSSD) across the groups were significantly different in the recovery period, such that those with no depression increased while those with at least mild depression decreased. Although depression has been linked to overactivation of the SNS (Carney et al., 2005), it may be that depressive symptoms are not as driven by the SNS as PTSD symptoms (i.e., hyperarousal), and therefore, during recovery, changes in vagal tone are not obscured by SNS activation in depressive groups as they may be in PTSD groups. Since the changes in HRV appeared to be driven by increased vagal input and recovery is even more dependent on the PNS than during the reactivity period, the fact that depressive symptoms are more closely linked to the PNS may explain why there were differences between the depression groups but not the PTSD response groups.

HRV and CAPS Clusters

In examining relationships of specific CAPS clusters to HRV, no individual cluster was better than others at predicting any HRV measure. At first glance, this finding appears surprising, as vagal tone in particular has been linked to the inhibition of intrusive thoughts and prefrontal cortical processing (Gillie & Thayer, 2014). However, this previous research was done using resting HRV and the current study did not identify any changes at rest; thus, it is possible that the differential relationships to individual clusters may not be detectable. Additionally, much of this work compares HRV in those with PTSD to non-trauma exposed individuals as a control group while the current

sample consisted of individuals all with relatively severe PTSD. It is possible that comparison between individuals whom all exhibit relatively severe PTSD symptoms, does not present enough variability in either HRV or cluster scores to detect these differences.

Aim 2: HRV and Social Functioning

Differences at Pretreatment

Overall social functioning as well as functioning in the domains of housework, social/leisure, extended family, and family unit improved for treatment responders but not for non-responders, demonstrating that improvement in PTSD symptoms is related to improvements in day-to-day life. Notably, occupation and economic functioning did not improve, with only about 50% of the sample reported having a job outside the home or being a student at both pre and posttreatment, highlighting the difficulties of maintaining employment or continued education while managing PTSD symptoms. Additionally, both occupation and economic domains potentially represent longer term outcomes and differences may not be apparent directly following treatment.

Social Functioning and HRV

The hypothesis that HF-HRV would be related to social functioning at each timepoint was not supported in any social functioning measure. At pretreatment, this may be explained by the current sample reporting relatively severe PTSD symptoms and relatively low social functioning, limiting the variance in both measures. At posttreatment, it is less clear why there was no relationship between the two. It may be that again, the timing of the assessment prevents seeing more permanent changes in the nervous system that occur later in posttreatment. The fact that HF-HRV was only

different during the reactivity phase may indicate that differences were only evident during cue re-exposure and that more permanent changes were not yet apparent.

Changes in vagal tone were not found to be related to overall social functioning or any subscales posttreatment, with the exception of extended family which was even more robust when the hypnosis subgroup was removed for sensitivity analyses. While vagal tone was expected to be related to all domains of social functioning, closer examination of the subscales may help explain this finding. Within the subscales tested, the degree to which direct social interaction is required varies. Housework, for example does not necessarily require direct social interaction and while social/leisure functioning may include direct interaction with people, these interactions are likely more casual than with a family member and includes activities that require no direct social interaction at all. In the current sample, vagal tone improved during trauma cue exposure but not during rest or recovery; therefore, the domains of social functioning in which the effects of increased vagal tone would be most evident are those that require the cognitive flexibility to inhibit intrusive traumatic information in order to facilitate a meaningful interaction. Thus, because they require direct contact with other individuals and are likely more emotionally intense compared to casual social interactions, extended family and family unit domains may best reflect the effects of improved vagal tone.

That there was no relationship between vagal tone and family unit may reflect this particular sample. Because participants were physical and sexual assault survivors, it is likely that many of their partners were their perpetrators therefore not a safe place to seek support. Unfortunately, participants' proximity to their perpetrator was not available but considering the high rates of intimate partner violence (Smith et al., 2018) it is

statistically likely that this was the case for many participants. It is possible then, that extended family members are a primary source of comfort and support and improvement in vagal tone allowed participants to seek out this support more comfortably and have more positive interactions.

While taken together, findings only weakly support the polyvagal theory, it is important to note that research typically cited as support links vagal tone to psychopathology involving severe social impairment, such as borderline personality disorder (Austin et al., 2007) and schizophrenia (Clamor et al., 2018) and not typical every-day social functioning. It is possible that the connection between vagal tone and social functioning is more evident when social dysfunction is more extreme. Additionally, the link is often seen at rest (Beffara et al., 2016; Geisler et al., 2013); thus because improvement in vagal tone was not detected during the rest period it is possible that this relationship may only be evident further removed from treatment as longer-term changes in the nervous system occur. Because these data were collected immediately following treatment, links between more slowly recovering systems may not be detectable, especially considering the small sample size.

Social Functioning, CAPS and HRV: Replicating and Extending Shnaider et al. (2014)

Posttreatment improvements in social functioning were similar to that of Shnaider et al. (2014), although there were some variations from their findings. Because different measures were used to assess social functioning, the subscales do not match perfectly but are comparable. In both studies, analyses examining overall PTSD symptoms and social functioning found that overall functioning, housework, social/leisure, extended family, and family relationships were all significantly predicted by improvements in PTSD

symptoms, indicating that improvement in PTSD symptoms predicts improvements in social functioning. That Shnaider et al. (2014) found improvements in work, while the current study did not may be due to differences in the samples as the current study selected participants with significant sleep impairments, which may affect work performance or maintenance, supported by the fact that only 50% of the current sample reported having a job or being a student. In the current study's attempts to extend Shnaider et al. (2014), the addition of HF-HRV to the model was not a significant contributor to any aspect of functioning. It is likely that the previous significant relationship between vagal tone and extended family disappeared due to the variance being subsumed under PTSD symptoms.

In analyses examining whether CAPS symptom clusters were associated with particular aspects of social functioning, replication was mixed. The current study was consistent with Shnaider et al. (2014) in that hyperarousal predicted overall functioning but identified numbing as a better predictor of a wider range of social functioning compared to Shnaider et al. (2014). Other differences can be seen in the housework and family unit subscales. In the current study, housework was not predicted by any cluster while Shnaider et al. (2014) found hyperarousal a significant predictor. While the findings of the current study differ somewhat from Shnaider et al. (2014), they are consistent with other previous research that has found numbing to be closely associated with interpersonal relationships and hyperarousal with overall psychosocial functioning (Kuhn et al., 2003; Shea et al., 2010).

Secondary Aims: Vagal Tone, Sleep Impairment, and Quality of Life

While sleep quality improved with treatment, there were no differences between treatment responder and non-responder groups. Additionally, HF-HRV was not a significant predictor of sleep quality at pre or posttreatment and was not a significant addition to the model predicting posttreatment sleep quality once pretreatment sleep scores were considered. Sleep impairment remains one of the most reported symptoms of PTSD (Germain et al., 2013) but also one of the most resistant to treatment (Belleville et al., 2011). While sleep did improve posttreatment, PSQI scores remained beyond the cutoff for presence of sleep impairment (M = 8.77), indicating that sleep quality remained an issue for participants. Comparing findings regarding HRV to previous research is difficult; while higher HRV has been linked to better sleep, most studies assess HRV during sleep as opposed to exposure to trauma related cues (Stoakley et al., 2019). More research is needed to understand how HRV assessed in different paradigms (i.e., during exposure to trauma cues or during recovery) is related to sleep quality and whether particular domains of sleep are more closely related to these different periods of assessment.

Findings regarding quality of life were similar to those of sleep, with posttreatment improvement that was independent of any HRV changes. There was one notable difference, however, such that quality of life differences between the responder groups were seen at pretreatment, with the treatment responder group reporting significantly higher scores. It may be that this higher quality of life contributed to the success of those in the responder group, or there may have been another untested variable responsible for both higher quality of life and greater treatment response.

Limitations and Conclusions

HRV is an accessible measure that captures dynamic and informative physiological processes important in processing traumatic information. Evidence strongly supports CPT as an effective treatment for PTSD and current findings indicate that it can improve HRV in response to trauma cues indicating remediation of dysfunction that may develop in the course of PTSD. Improvements in HRV likely reflect increases in vagal tone that allow for successful engagement of higher-order processing of trauma information as well as thoughts and beliefs surrounding the trauma, highlighting the connection between physiological mechanisms and psychological processes.

Since its introduction in 1994, enthusiasm for the polyvagal theory's elegant framework has grown and is frequently embraced by clinicians as an accessible way to communicate the underlying neuroscience of psychopathology to patients (Wagner, 2016). The theory has grown so popular that a polyvagal institute has been established to promote and provide training for the theory (https://www.polyvagalinstitute.org/). However, there has been significant pushback from researchers questioning its premises (Berntson et al., 2007; Grossman & Taylor, 2007; Monteiro et al., 2018). More recently, an intense debate has grown up around the proliferation of this theory in the public sphere and whether the evidence is strong enough to warrant its widespread use, with some claiming that it should be thrown out altogether (Grossman, 2016; Tang, 2021), and defenders claiming the premises are being misinterpreted (Porges, 2021). The current study found some support for the theory, although limited, with the significant relationship between vagal tone and the extended family domain of social functioning. Much more research is needed before it is abandoned all together and it may be that HRV

is related to social functioning, perhaps just through different mechanisms than those proposed in the polyvagal theory.

Because this population is difficult to recruit and retain, many participants were lost to attrition. This limited power and prevented the use of complex statistical analyses that may have provided a more nuanced view of the relationships assessed. Additionally, some details of participants trauma (i.e., proximity to the perpetrator) were not available for analysis and limited interpretation of findings regarding support for the polyvagal theory. Information regarding previous therapies and psychotropic medications was also unavailable. While participants were required to be stable on medications for a month, there may have been medication effects on HRV that were unable to be tested. While HRV was able to be assessed during multiple periods, data had to be collapsed across multiple instances to meet duration standards HRV measurement. It is possible that this obscured some relationships as participants may have had different cardiac responses to the first exposure vs the second. Also, participants were all female and limited to specific traumatic experiences and thus current results may not generalize to other populations. Because the data were collected before the release of DSM-5, participants were assessed using DSM-IV criteria. Replication needs to be completed using updated DSM-5 criteria before firm conclusions can be drawn from findings.

Despite these limitations, the current study had many strengths that allow for a more comprehensive picture of the effects of trauma on an individual. It was unique in that HRV was collected across multiple assessment periods, including during trauma cue exposure, both pre and posttreatment in PTSD positive physical and sexual assault survivors. Because of its connection to important health outcomes, understanding the role

vagal tone plays in nervous system response to traumatic stress and how treatment can remediate those changes is an important piece of the puzzle in improving the lives of those living with PTSD.

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Table 1

Summary of the Main HRV Measures and Their Physiological Origins

	Variable	Description	Physiological origin
Time-domain	SDNN	Standard deviation of all R–R intervals	Cyclic components responsible for heart rate variability
	RMSSD	Root mean square of successive differences	Vagal tone
	pNN50	Percentage of successive normal sinus RR intervals more than 50 ms	Vagal tone
	Peak-valley	Time-domain filter dynamically centered at the exact ongoing respiratory frequency	Vagal tone
Frequency-domain	ULF	Ultra-low frequencies	Circadian oscillations, core body temperature, metabolism and the renin-angiotensin system
	VLF	Very-low frequencies	Long-term regulation mechanisms, thermoregulation and hormonal mechanisms
	LF	Low frequencies	Mix of sympathetic and vagal activity, baroreflex activity
	HF	High frequencies	Vagal tone
	LF/HF	Low frequencies/high-frequencies ratio	Mix of sympathetic and vagal activity
Non-linear indices	SD1	Standard deviation – Poincaré plot Crosswise	Unclear, depicts quick and high frequent changes in heart rate variability
	SD2	Standard deviation – Poincaré plot Lengthwise	Unclear, depicts long-term changes in heart rate variability

Note. From “Heart rate variability and cardiac vagal tone in psychophysiological research - Recommendations for experiment planning, data analysis, and data reporting” by S. Laborde, E. Mosley, and J. F. Thayer, 2017, Frontiers in Psychology, 8, p. 4 (10.3389/fpsyg.2017.00213. Copyright 2017 by Frontiers Media.

Table 2

HRV Measurement Period Description and Length

Measure	Description	Total Approximate Time (minutes)
Rest	Baseline prior to presentation of any stimuli	4
Reactivity	Combined script reading and imagery periods for both trauma scripts (SCPT+IMAG)	2
Recovery	Combined recovery and subsequent rest time following the trauma scripts before the next script begins.	3

Table 3

HRV Measures for Analysis in Current Study

Measure	Origin of Input	Frequency (Hz)
Time-Domain Measures		
SDNN	Overall HRV	
RMSSD	Vagal Tone	
Frequency-Domain Measures		
High Frequency	Vagal Tone	.15-.40
Low Frequency	Parasympathetic and sympathetic activation	.04-.15

Note. SDNN = standard deviation of all R-R intervals; RMSSD = root mean square of successive differences; Hz = hertz.

Table 4

Means and Standard Deviations HRV Variables in Non-transformed Absolute Units

		Pretreatment		Posttreatment	
	n	*M*	*SD*	*M*	*SD*
Rest	39				
HF[a]		584.20	790.27	409.85	551.52
LF[a]		615.06	648.25	526.62	573.37
SDNN[b]		33.53	17.47	29.48	15.70
RMSSD[b]		31.46	19.28	26.77	17.54
Reactivity	41				
HF[a]		333.00	388.53	436.35	547.45
LF[a]		577.57	652.21	384.42	462.41
SDNN[b]		29.54	15.38	27.85	14.99
RMSSD[b]		24.86	14.66	27.41	17.60
Recovery	40				
HF[a]		518.13	655.31	471.30	672.86
LF[a]		761.68	901.81	546.53	566.19
SDNN[b]		34.07	19.07	30.95	16.69
RMSSD[b]		28.92	19.07	27.82	18.32

Note. HF = high frequency heart rate variability; LF = low frequency heart rate variability; SDNN = standard deviation of all R-R intervals; RMSSD = root mean square of successive differences.

[a] units = ms^2; [b] units = ms

Table 5a

Means, Standard Deviations and Difference Effects in CAPS Scores: Time x Treatment Response

	PRE		POST						
Participant Group	M	SD	M	SD	M_{Diff} POST-PRE[a]	Effect	F(1, 39)	p	Partial η^2
CAPS Total									
Total Sample[a]	74.69	15.89	28.22	22.05	-46.47	**Time**	166.51	< .001	0.81
NR[b]	78.47	14.58	58.27	13.77		**Treatment Response**	32.68	< .001	0.46
R[c]	73.30	16.35	17.20	11.71		**Time x Treatment Response**	36.88	< .001	0.49
Reexperiencing Cluster									
Total Sample[a]	20.39	8.25	5.61	6.65	-14.78	**Time**	88.15	< .001	0.69
NR[b]	20.10	8.64	12.27	8.20		**Treatment Response**	4.42	0.042	0.10
R[c]	20.50	8.25	3.17	3.85		**Time x Treatment Response**	12.58	0.001	0.24
Avoidance Cluster									
Total Sample[a]	10.48	3.31	2.17	3.67	-8.31	**Time**	120.23	< .001	0.76
NR[b]	11.15	2.85	6.55	4.52		**Treatment Response**	17.90	< .001	0.32
R[c]	10.23	3.48	0.57	1.25		**Time x Treatment Response**	15.13	< .001	0.28
Emotional Numbing Cluster									
Total Sample[a]	19.17	6.45	7.95	8.48	-11.22	**Time**	42.85	< .001	0.52
NR[b]	21.91	5.36	18.64	6.20		**Treatment Response**	33.04	< .001	0.46
R[c]	18.17	6.60	4.03	5.16		**Time x Treatment Response**	16.68	< .001	0.30
Hyperarousal Cluster									
Total Sample[a]	24.76	5.45	12.49	7.67	-12.27	**Time**	46.97	< .001	0.55
NR[b]	25.73	4.47	20.82	3.68		**Treatment Response**	21.78	< .001	0.36
R[c]	24.40	5.80	9.43	6.36		**Time x Treatment Response**	12.03	0.001	0.24

Note. Statistically significant effects ($p < .05$) are in bold type. CAPS = Clinician Administered PTSD Scale; POST = posttreatment; PRE = pretreatment; NR = non-responders; R = responders; M_{Diff} = mean difference.

[a] $N = 41$. [b] $n = 11$. [c] $n = 30$.

Table 5b

Simple Main Effects of Time and Treatment Response in CAPS Scores ANOVA

Effect	M_{Diff} POST-PRE	M_{Diff} NR-R	*F*	*p*	Partial η^2
CAPS Total					
Time in NR	-20.19		15.94	< .001	0.29
Time in R	-56.10		335.57	< .001	0.90
Treatment Response at PRE		5.17	0.85	0.363	0.02
Treatment Response at POST		41.07	90.14	< .001	0.70
Reexperiencing Cluster					
Time in NR	-7.83		11.66	0.002	0.23
Time in R	-17.33		155.91	< .001	0.80
Treatment Response at PRE		-0.40	0.02	0.893	0.00
Treatment Response at POST		9.11	23.61	< .001	0.38
Avoidance Cluster					
Time in NR	-4.60		17.10	< .001	0.31
Time in R	-9.67		205.60	< .001	0.84
Treatment Response at PRE		0.92	0.61	0.440	0.02
Treatment Response at POST		5.98	44.87	< .001	0.54
Emotional Numbing Cluster					
Time in NR	-3.27		2.07	0.158	0.05
Time in R	-14.13		105.29	< .001	0.73
Treatment Response at PRE		3.74	2.83	0.135	0.07
Treatment Response at POST		14.60	57.83	< .001	0.60
Hyperarousal Cluster					
Time in NR	-4.91		3.91	0.055	0.09
Time in R	-14.97		99.27	< .001	0.72
Treatment Response at PRE		1.32	0.47	0.497	0.01
Treatment Response at POST		11.38	31.08	< .001	0.44

Note. Statistically significant effects ($p < .05$) are in bold type. Total sample $N = 41$; NR $n = 11$; R $n = 30$. ANOVA = analysis of variance; CAPS = Clinician Administered PTSD Scale; M_{Diff} = mean difference; NR = non-responders; R = responders; POST = posttreatment; PRE = pretreatment.

Table 6a

Means, Standard Deviations and Difference Effects in HRV During the Rest Period: Time x Treatment Response x Treatment Condition

		PRE		POST						
Study Groups	*n*	*M*	*SD*	*M*	*SD*	M_{Diff} POST - PRE	Effect	*F*	*p*	Partial η^2
Rest Period (1, 34)										
						HF				
NR	12	6.12	1.53	5.19	1.49	-0.93	**Time**	7.05	0.012	0.17
ssmCPT	7	6.13	0.96	5.13	1.61	-1.00	Response	0.23	0.632	0.01
hypCPT	5	6.09	2.25	5.26	1.49	-0.83	Condition	0.00	0.961	0.00
R	26	5.54	1.59	5.27	1.60	-0.27	Time x Response	2.90	0.098	0.08
ssmCPT	11	5.26	1.46	5.53	1.57	0.27	Time x Condition	0.84	0.366	0.02
hypCPT	15	5.74	1.69	5.07	1.64	-0.67	Response x Condition	0.00	0.969	0.00
Total ssmCPT	18	5.60	1.33	5.38	1.55	-0.22	Time x Response x Condition	1.75	0.194	0.05
Total hypCPT	20	5.83	1.79	5.12	1.56	-0.71				
Total Sample	38	5.72	1.57	5.24	1.54	-0.48				
						LF				
NR	12	6.19	1.26	5.62	0.82	-0.57	**Time**	4.13	0.050	0.11
ssmCPT	7	6.13	1.08	5.64	1.00	-0.49	Response	0.35	0.561	0.01
hypCPT	5	6.28	1.60	5.59	0.59	-0.69	Condition	0.21	0.653	0.01
R	26	5.82	1.32	5.58	1.31	-0.25	Time x Response	0.87	0.358	0.03
ssmCPT	11	5.54	1.48	5.50	1.42	-0.04	Time x Condition	0.48	0.494	0.01
hypCPT	15	6.03	1.20	5.63	1.27	-0.40	Response x Condition	0.11	0.747	0.00
Total ssmCPT	18	5.77	1.34	5.55	1.24	-0.22	Time x Response x Condition	0.04	0.846	0.00
Total hypCPT	20	6.09	1.27	5.62	1.13	-0.47				
Total Sample	38	5.94	1.30	5.59	1.17	-0.35				
						SDNN				
NR	12	1.54	0.29	1.39	0.22	-0.14	**Time**	6.70	0.014	0.17
ssmCPT	7	1.53	0.21	1.38	0.25	-0.14	Response	0.15	0.706	0.00
hypCPT	5	1.55	0.41	1.41	0.19	-0.14	Condition	0.11	0.744	0.00
R	26	1.47	0.26	1.40	0.28	-0.07	Time x Response	0.98	0.330	0.03
ssmCPT	11	1.43	0.26	1.41	0.31	-0.02	Time x Condition	0.37	0.547	0.01
hypCPT	15	1.51	0.26	1.39	0.26	-0.11	Response x Condition	0.00	0.997	0.00
Total ssmCPT	18	1.47	0.24	1.40	0.28	-0.07	Time x Response x Condition	0.37	0.545	0.01
Total hypCPT	20	1.52	0.30	1.40	0.24	-0.12				
Total Sample	38	1.49	0.27	1.40	0.26	-0.10				
						RMSSD				
NR	12	1.49	0.35	1.31	0.31	-0.18	**Time**	6.10	0.019	0.15
ssmCPT	7	1.46	0.26	1.28	0.35	-0.17	Response	0.02	0.900	0.00
hypCPT	5	1.54	0.48	1.34	0.27	-0.20	Condition	0.06	0.810	0.00
R	26	1.43	0.31	1.35	0.33	-0.08	Time x Response	1.26	0.270	0.04
ssmCPT	11	1.39	0.30	1.40	0.34	0.01	Time x Condition	0.81	0.376	0.02
hypCPT	15	1.46	0.33	1.31	0.33	-0.15	Response x Condition	0.16	0.691	0.01
Total ssmCPT	18	1.42	0.28	1.36	0.34	-0.06	Time x Response x Condition	0.41	0.527	0.01
Total hypCPT	20	1.48	0.36	1.32	0.31	-0.16				
Total Sample	38	1.45	0.32	1.34	0.32	-0.11				

Note. Statistically significant effects ($p < .05$) are in bold type. Degrees of freedom are in parentheses following the assessment period. HF = high frequency heart rate variability; LF = low frequency heart rate variability; SDNN = standard deviation of all R-R intervals; RMSSD = root mean square of successive differences; NR = non-responders; R = responders; POST = posttreatment; PRE = pretreatment; ssmCPT = sleep and symptom monitoring plus cognitive

processing therapy; hypCPT = hypnosis plus cognitive processing therapy; M_{Diff} = mean difference.

Table 6b

Means, Standard Deviations and Difference Effects in HRV During the Reactivity Period: Time x Treatment Response x Treatment Condition

		PRE		POST						
Study Groups	*n*	*M*	*SD*	*M*	*SD*	M_{Diff} POST - PRE	Effect	*F*	*p*	Partial η^2
Reactivity Period (1, 37)										
						HF				
NR	11	5.46	1.22	4.98	1.62	-0.48	Time	0.00	0.953	0.00
ssmCPT	7	5.74	1.33	5.34	1.64	-0.40	Response	0.00	0.982	0.00
hypCPT	4	4.96	0.93	4.34	1.60	-0.62	Condition	0.79	0.380	0.02
R	30	4.87	1.58	5.29	1.56	0.42	**Time x Response**	5.17	0.029	0.12
ssmCPT	12	4.71	1.39	5.50	1.59	0.79	Time x Condition	0.93	0.341	0.02
hypCPT	18	4.98	1.72	5.15	1.57	0.17	Response x Condition	0.67	0.419	0.02
Total ssmCPT	19	5.09	1.43	5.44	1.56	0.35	Time x Response x Condition	0.21	0.646	0.01
Total hypCPT	22	4.98	1.59	5.00	1.57	0.03				
Total Sample	41	5.03	1.50	5.21	1.56	0.18				
						LF				
NR	11	6.00	1.04	5.15	1.43	-0.85	**Time**	11.20	0.002	0.23
ssmCPT	7	6.01	1.25	5.41	1.69	-0.60	Response	0.07	0.791	0.00
hypCPT	4	6.00	0.70	4.70	0.84	-1.29	Condition	0.13	0.716	0.00
R	30	5.53	1.48	5.27	1.41	-0.26	**Time x Response**	4.76	0.036	0.11
ssmCPT	12	5.34	1.67	5.46	1.33	0.12	Time x Condition	3.73	0.061	0.09
hypCPT	18	5.66	1.37	5.14	1.49	-0.52	Response x Condition	0.14	0.715	0.00
Total ssmCPT	19	5.59	1.52	5.44	1.43	-0.15	Time x Response x Condition	0.01	0.940	0.00
Total hypCPT	22	5.72	1.27	5.06	1.39	-0.66				
Total Sample	41	5.66	1.38	5.23	1.40	-0.42				
						SDNN				
NR	11	1.46	0.22	1.32	0.30	-0.14	Time	3.83	0.058	0.09
ssmCPT	7	1.49	0.26	1.37	0.35	-0.11	Response	0.00	0.962	0.00
hypCPT	4	1.43	0.15	1.24	0.20	-0.19	Condition	0.21	0.650	0.01
R	30	1.38	0.27	1.39	0.26	0.01	**Time x Response**	6.15	0.018	0.14
ssmCPT	12	1.34	0.27	1.41	0.28	0.07	Time x Condition	1.80	0.188	0.05
hypCPT	18	1.41	0.26	1.37	0.26	-0.04	Response x Condition	0.36	0.555	0.01
Total ssmCPT	19	1.40	0.27	1.40	0.30	0.00	Time x Response x Condition	0.05	0.827	0.00
Total hypCPT	22	1.41	0.24	1.35	0.25	-0.06				
Total Sample	41	1.40	0.25	1.37	0.27	-0.03				
						RMSSD				
NR	11	1.37	0.28	1.28	0.34	-0.09	Time	0.00	0.951	0.00
ssmCPT	7	1.39	0.35	1.32	0.40	-0.07	Response	0.01	0.924	0.00
hypCPT	4	1.33	0.10	1.21	0.22	-0.12	Condition	0.16	0.691	0.00
R	30	1.28	0.32	1.36	0.31	0.08	**Time x Response**	4.77	0.035	0.11
ssmCPT	12	1.25	0.32	1.40	0.31	0.15	Time x Condition	0.88	0.355	0.02
hypCPT	18	1.30	0.34	1.34	0.31	0.04	Response x Condition	0.13	0.725	0.00
Total ssmCPT	19	1.30	0.33	1.37	0.34	0.07	Time x Response x Condition	0.13	0.721	0.00
Total hypCPT	22	1.31	0.31	1.32	0.30	0.01				
Total Sample	41	1.31	0.31	1.34	0.31	0.03				

Note. Statistically significant effects ($p < .05$) are in bold type. Degrees of freedom are in parentheses following the assessment period. HF = high frequency heart rate variability; LF = low frequency heart rate variability; SDNN = standard deviation of all R-R intervals; RMSSD = root mean square of successive differences; NR = non-responders; R

= responders; POST = posttreatment; PRE = pretreatment; ssmCPT = sleep and symptom monitoring plus cognitive processing therapy; hypCPT = hypnosis plus cognitive processing therapy; M_{Diff} = mean difference.

Table 6c

Simple Main Effects of Time and Treatment Response in HRV ANOVA

Effect	M_{Diff} POST-PRE[a]	M_{Diff} NR-R[a]	*F*	*p*	Partial η^2
		HF			
Time in NR	-0.48		1.84	0.183	0.05
Time in R	0.42		4.70	0.037	0.11
Treatment Response at PRE		0.59	0.84	0.366	0.02
Treatment Response at POST		-0.31	0.69	0.410	0.02
		LF			
Time in NR	-0.85		10.34	0.003	0.22
Time in R	-0.26		1.30	0.261	0.03
Treatment Response at PRE		0.47	0.97	0.331	0.03
Treatment Response at POST		-0.12	0.22	0.644	0.01
		SDNN			
Time in NR	-0.14		6.65	0.014	0.15
Time in R	0.01		0.26	0.612	0.01
Treatment Response at PRE		0.08	0.73	0.399	0.02
Treatment Response at POST		-0.07	0.78	0.382	0.02
		RMSSD			
Time in NR	-0.09		1.71	0.200	0.04
Time in R	0.08		4.30	0.045	0.10
Treatment Response at PRE		0.09	0.53	0.472	0.01
Treatment Response at POST		-0.08	0.81	0.375	0.02

Note. Statistically significant effects ($p < .05$) are in bold type. Total sample $N = 41$; NR $n = 11$; R $n = 30$. HRV = heart rate variability; HF = high frequency; LF = low frequency; SDNN = standard deviation of all R-R intervals; RMSSD = root mean square of successive differences; NR = Non-Responders; R = Responders; POST = posttreatment; PRE = pretreatment; M_{Diff} = mean difference.

[a] All mean differences are in natural log units.

Table 6d

Means, Standard Deviations and Difference Effects in HRV During the Recovery Period: Time x Treatment Response x Treatment Condition

		PRE		POST						
Study Groups	*n*	*M*	*SD*	*M*	*SD*	M_{Diff} POST - PRE	Effect	*F*	*p*	Partial η^2
Recovery Period (1, 36)										
						HF				
NR	11	5.64	1.02	5.28	1.41	-0.37	Time	0.47	0.498	0.01
ssmCPT	7	5.91	1.03	5.51	1.57	-0.40	Response	0.00	0.965	0.00
hypCPT	4	5.17	0.93	4.87	1.16	-0.30	Condition	0.58	0.453	0.02
R	29	5.36	1.66	5.40	1.44	0.05	Time x Response	1.41	0.243	0.04
ssmCPT	12	5.26	1.48	5.61	1.42	0.36	Time x Condition	0.31	0.579	0.01
hypCPT	17	5.42	1.81	5.26	1.48	-0.17	Response x Condition	0.33	0.570	0.01
Total ssmCPT	19	5.50	1.34	5.57	1.43	0.08	Time x Response x Condition	0.71	0.405	0.02
Total hypCPT	21	5.38	1.66	5.18	1.40	-0.19				
Total Sample	40	5.43	1.50	5.37	1.41	-0.07				
						LF				
NR	11	6.19	0.88	5.81	1.24	-0.38	**Time**	4.83	0.034	0.12
ssmCPT	7	6.17	0.96	5.95	1.31	-0.22	Response	0.37	0.549	0.01
hypCPT	4	6.22	0.87	5.57	1.26	-0.65	Condition	0.09	0.764	0.00
R	29	5.93	1.41	5.56	1.37	-0.37	Time x Response	0.04	0.846	0.00
ssmCPT	12	5.63	1.42	5.33	1.34	-0.30	Time x Condition	0.59	0.446	0.02
hypCPT	17	6.14	1.41	5.71	1.40	-0.43	Response x Condition	0.45	0.505	0.01
Total ssmCPT	19	5.83	1.27	5.56	1.33	-0.27	Time x Response x Condition	0.18	0.678	0.01
Total hypCPT	21	6.15	1.31	5.68	1.35	-0.47				
Total Sample	40	6.00	1.28	5.63	1.32	-0.37				
						SDNN				
NR	11	1.51	0.17	1.43	0.25	-0.08	Time	3.79	0.059	0.10
ssmCPT	7	1.52	0.18	1.45	0.28	-0.07	Response	0.10	0.752	0.00
hypCPT	4	1.49	0.17	1.39	0.21	-0.10	Condition	0.00	0.968	0.00
R	29	1.46	0.29	1.41	0.28	-0.04	Time x Response	0.61	0.440	0.02
ssmCPT	12	1.41	0.28	1.41	0.28	0.00	Time x Condition	0.59	0.449	0.02
hypCPT	17	1.49	0.31	1.42	0.29	-0.07	Response x Condition	0.23	0.632	0.01
Total ssmCPT	19	1.45	0.25	1.42	0.28	-0.03	Time x Response x Condition	0.10	0.760	0.00
Total hypCPT	21	1.49	0.28	1.41	0.27	-0.07				
Total Sample	40	1.47	0.26	1.42	0.27	-0.05				
						RMSSD				
NR	11	1.39	0.24	1.33	0.29	-0.06	Time	0.37	0.545	0.01
ssmCPT	7	1.42	0.27	1.34	0.35	-0.08	Response	0.02	0.892	0.00
hypCPT	4	1.33	0.17	1.29	0.20	-0.04	Condition	0.20	0.660	0.01
R	29	1.36	0.35	1.36	0.32	0.00	Time x Response	0.95	0.336	0.03
ssmCPT	12	1.34	0.31	1.42	0.31	0.08	Time x Condition	0.42	0.521	0.01
hypCPT	17	1.38	0.38	1.32	0.32	-0.05	Response x Condition	0.04	0.840	0.00
Total ssmCPT	19	1.37	0.29	1.39	0.32	0.02	Time x Response x Condition	1.44	0.238	0.04
Total hypCPT	21	1.37	0.35	1.32	0.30	-0.05				
Total Sample	40	1.37	0.32	1.35	0.31	-0.02				

Note. Statistically significant effects ($p < .05$) are in bold type. Degrees of freedom are in parentheses following the assessment period. HF = high frequency heart rate variability; LF = low frequency heart rate variability; SDNN = standard deviation of all R-R intervals; RMSSD = root mean square of successive differences; NR = non-responders; R = responders; POST = posttreatment; PRE = pretreatment; ssmCPT = sleep and symptom monitoring plus cognitive

processing therapy; hypCPT = hypnosis plus cognitive processing therapy; M_{Diff} = mean difference.

Table 7

Differences in HRV across Time and Depression Response

		PRE		POST						
Depression Group	*n*	*M*	*SD*	*M*	*SD*	M_{Diff} POST-PRE	Effect	*F*	*p*	Partial η^2
Rest Period (1, 35)										
					HF					
No Depression	23	5.37	1.68	5.19	1.66	-0.18	**Time**	6.15	0.018	0.15
Depression	14	5.99	0.79	5.18	1.35	-0.81	DpR	0.43	0.515	0.01
Total Sample	37	5.61	1.43	5.19	1.53	-0.42	Time x DpR	2.43	0.128	0.07
					LF					
No Depression	23	5.76	1.34	5.56	1.37	-0.20	Time	3.00	0.092	0.08
Depression	14	6.01	0.95	5.61	0.81	-0.40	DpR	0.17	0.687	0.01
Total Sample	37	5.86	1.20	5.58	1.18	-0.28	Time x DpR	0.34	0.565	0.01
					SDNN					
No Depression	23	1.45	0.28	1.39	0.29	-0.06	**Time**	6.08	0.019	0.15
Depression	14	1.50	0.17	1.38	0.20	-0.12	DpR	0.07	0.794	0.00
Total Sample	37	1.47	0.24	1.39	0.26	-0.08	Time x DpR	0.72	0.401	0.02
					RMSSD					
No Depression	23	1.41	0.33	1.33	0.34	-0.07	**Time**	5.08	0.031	0.13
Depression	14	1.45	0.20	1.31	0.29	-0.14	DpR	0.02	0.896	0.00
Total Sample	37	1.42	0.29	1.32	0.32	-0.10	Time x DpR	0.56	0.459	0.02
Reactivity Period (1, 39)										
					HF					
No Depression	26	4.64	1.59	5.19	1.60	0.54	Time	0.06	0.813	0.00
Depression	15	5.70	1.07	5.24	1.55	-0.45	DpR	1.53	0.224	0.04
Total Sample	41	5.03	1.50	5.21	1.56	0.18	**Time x DpR**	7.06	0.011	0.15
					LF					
No Depression	26	5.35	1.47	5.15	1.47	-0.20	**Time**	10.32	0.003	0.21
Depression	15	6.19	1.04	5.39	1.30	-0.80	DpR	1.67	0.204	0.04
Total Sample	41	5.66	1.38	5.23	1.40	-0.42	Time x DpR	3.66	0.063	0.09
					SDNN					
No Depression	26	1.34	0.26	1.37	0.27	0.03	Time	3.50	0.069	0.08
Depression	15	1.52	0.20	1.37	0.28	-0.14	DpR	1.29	0.263	0.03
Total Sample	41	1.40	0.25	1.37	0.27	-0.03	**Time x DpR**	8.26	0.007	0.18
					RMSSD					
No Depression	26	1.25	0.33	1.34	0.32	0.10	Time	0.09	0.768	0.00
Depression	15	1.41	0.25	1.33	0.32	-0.08	DpR	0.70	0.408	0.02
Total Sample	41	1.31	0.31	1.34	0.31	0.03	**Time x DpR**	5.32	0.027	0.12
Recovery Period (1, 38)										
					HF					
No Depression	25	5.09	1.69	5.31	1.50	0.22	Time	1.01	0.321	0.03
Depression	15	6.01	0.91	5.47	1.30	-0.54	DpR	1.50	0.229	0.04
Total Sample	40	5.43	1.50	5.37	1.41	-0.07	**Time x DpR**	5.68	0.022	0.13
					LF					
No Depression	25	5.77	1.38	5.52	1.45	-0.25	**Time**	7.03	0.012	0.16
Depression	15	6.38	1.03	5.80	1.11	-0.58	DpR	1.28	0.265	0.03
Total Sample	40	6.00	1.28	5.63	1.32	-0.37	Time x DpR	1.09	0.302	0.03
					SDNN					
No Depression	25	1.41	0.29	1.40	0.30	-0.01	**Time**	6.21	0.017	0.14
Depression	15	1.56	0.18	1.45	0.22	-0.11	DpR	1.42	0.241	0.04
Total Sample	40	1.47	0.26	1.42	0.27	-0.05	Time x DpR	3.82	0.058	0.09
					RMSSD					
No Depression	25	1.30	0.35	1.34	0.33	0.03	Time	1.06	0.310	0.03
Depression	15	1.47	0.22	1.37	0.27	-0.10	DpR	1.13	0.294	0.03
Total Sample	40	1.37	0.32	1.35	0.31	-0.02	**Time x DpR**	4.30	0.045	0.10

Note. Statistically significant effects ($p < .05$) are in bold type. Degrees of freedom are in parentheses following the assessment period. HF = high frequency; LF = low frequency; SDNN = standard deviation of all R-R intervals; RMSSD = root mean square of successive differences; POST = posttreatment; PRE = pretreatment; DpR = depression response; M_{Diff} = mean difference.

Table 8a

Means, Standard Deviations and Difference Effects in SAS Total and Subscale Score: Time x Treatment Response

		PRE		POST						
Study Group	*n*	*M*[a]	*SD*	*M*[a]	*SD*	M_{Diff} POST-PRE[a]	Effect	*F*	*p*	Partial η^2
Overall (1, 39)										
Total Sample	41	2.57	0.49	2.24	0.49	-0.33	**Response**	11.63	0.002	0.23
NR	11	2.76	0.29	2.74	0.41	-0.02	**Time**	11.16	0.002	0.22
R	30	2.50	0.54	2.06	0.37	-0.44	**Time x Response**	9.57	0.004	0.20
Housework (1, 35)										
Total Sample	37	2.59	0.82	2.25	0.75	-0.34	Response	0.85	0.363	0.02
NR	11	2.61	0.63	2.56	0.64	-0.05	**Time**	5.80	0.021	0.14
R	26	2.58	0.90	2.12	0.76	-0.46	Time x Response	3.70	0.063	0.10
Occupation (1, 18)										
Total Sample	20	2.14	0.79	2.09	1.16	-0.05	Response	0.00	0.970	0.00
NR	6	2.15	0.73	2.06	0.72	-0.10	Time	0.04	0.842	0.00
R	14	2.13	0.84	2.11	1.33	-0.03	Time x Response	0.01	0.908	0.00
Social/Leisure (1, 39)										
Total Sample	41	2.97	0.57	2.55	0.62	-0.42	**Response**	25.90	0.000	0.40
NR	11	3.43	0.61	3.19	0.56	-0.24	**Time**	15.10	0.000	0.28
R	30	2.80	0.45	2.31	0.46	-0.49	Time x Response	1.80	0.188	0.04
Extended Family (1, 38)										
Total Sample	40	2.40	0.67	2.13	0.59	-0.27	**Response**	5.57	0.023	0.13
NR	11	2.53	0.66	2.58	0.48	0.05	Time	1.74	0.194	0.04
R	29	2.35	0.67	1.96	0.54	-0.39	Time x Response	2.93	0.095	0.07
Family Unit (1, 31)										
Total Sample	33	2.25	0.61	2.05	0.76	-0.20	Response	1.16	0.291	0.04
NR	9	2.21	0.67	2.45	0.83	0.24	Time	0.22	0.642	0.01
R	24	2.27	0.60	1.90	0.69	-0.37	**Time x Response**	5.26	0.029	0.15
Financial (1, 33)										
Total Sample	35	3.26	1.62	3.11	1.49	-0.14	Response	3.24	0.081	0.09
NR	10	3.90	1.66	3.70	1.34	-0.20	Time	0.26	0.612	0.01
R	25	3.00	1.55	2.88	1.51	-0.12	Time x Response	0.02	0.899	0.00

Note. Statistically significant effects ($p < .05$) are in bold type. SAS = Social Adjustment Scale; NR = Non-Responders; R = Responders; POST = posttreatment; PRE = pretreatment; M_{Diff} = mean difference.

[a] Lower scores indicate better social functioning.

Table 8b

Simple Main Effects of Time and Treatment Response in SAS Scores

Simple Main Effect	M_{Diff} POST-PRE[a]	M_{Diff} NR-R[a]	*F*	*p*	Partial η^2
Overall (1, 39)					
Time in NR	-0.02		0.02	0.886	0.00
Time in R	-0.44		38.58	0.000	0.50
Treatment Response at PRE		0.26	2.24	0.143	0.05
Treatment Response at POST		0.68	25.85	0.000	0.40
Housework (1, 35)					
Time in NR	-0.05		0.08	0.775	0.00
Time in R	-0.46		15.77	0.000	0.31
Treatment Response at PRE		0.04	0.01	0.907	0.00
Treatment Response at POST		0.45	2.87	0.099	0.08
Social/Leisure (1, 39)					
Time in NR	-0.24		2.22	0.145	0.05
Time in R	-0.49		25.45	0.000	0.40
Treatment Response at PRE		0.62	12.50	0.001	0.24
Treatment Response at POST		0.87	25.64	0.000	0.40
Extended Family (1, 38)					
Time in NR	0.05		0.05	0.820	0.00
Time in R	-0.39		8.35	0.006	0.18
Treatment Response at PRE		0.18	0.58	0.452	0.02
Treatment Response at POST		0.62	11.11	0.002	0.23
Family Unit (1, 31)					
Time in NR	0.242		1.15	0.293	0.04
Time in R	-0.3665		7.01	0.013	0.18
Treatment Response at PRE		-0.06	0.06	0.813	0.00
Treatment Response at POST		0.55	3.70	0.064	0.11

Note. Simple main effects were only completed for scales with a significant effect.

Statistically significant effects ($p < .05$) are in bold type. SAS = Social Adjustment Scale;

NR = Non-Responders; R = Responders; POST = posttreatment; PRE = pretreatment;

M_{Diff} = mean difference.

Table 9

Final Model Regression Results Predicting POST Overall and SAS Subscale Social Functioning

Variable	R^2 *Adj*	ΔR^{2}_{NS}	*B*	*SE*	*β*	*p*
Rest						
Overall						
Final model	0.66	0.01				< .001
PRE SAS Total			0.50	0.10	0.50	< .001
CAPS Total			0.01	0.00	0.60	< .001
HF-HRV			0.00	0.00	0.08	0.455
Housework						
Final model	0.50	0.01				< .001
PRE SAS Total			0.62	0.11	0.69	< .001
CAPS Total			0.01	0.00	0.29	0.029
HF-HRV			0.00	0.00	0.08	0.545
Social/Leisure						
Final model	0.54	0.02				< .001
PRE SAS Total			0.40	0.14	0.37	0.007
CAPS Total			0.02	0.00	0.55	< .001
HF-HRV			0.00	0.00	0.15	0.228
Extended Family						
Final model	0.40	0.01				< .001
PRE SAS Total			0.30	0.12	0.33	0.019
CAPS Total			0.02	0.00	0.53	< .001
HF-HRV			0.00	0.00	-0.12	0.395
Family Unit						
Final model	0.32	0.03				0.004
PRE SAS Total			0.65	0.19	0.53	0.002
CAPS Total			0.01	0.01	0.37	0.023
HF-HRV			0.00	0.00	0.19	0.232
Reactivity						

Overall						
Final model	0.67	0.00				< .001
PRE SAS						
Total			0.50	0.09	0.51	< .001
CAPS Total			0.01	0.00	0.57	< .001
HF-HRV			0.00	0.00	0.01	0.956
Housework						
Final model	0.52	0.01				< .001
PRE SAS						
Total			0.64	0.11	0.70	< .001
CAPS Total			0.01	0.00	0.29	0.024
HF-HRV			0.00	0.00	0.08	0.512
Social/Leisure						
Final model	0.54	0.01				< .001
PRE SAS						
Total			0.43	0.13	0.39	0.003
CAPS Total			0.02	0.00	0.54	< .001
HF-HRV			0.00	0.00	0.10	0.390
Extended Family						
Final model	0.38	0.03				< .001
PRE SAS						
Total			0.24	0.11	0.27	0.042
CAPS Total			0.01	0.00	0.48	0.001
HF-HRV			0.00	0.00	-0.20	0.154
Family Unit						
Final model	0.27	0.01				0.007
PRE SAS						
Total			0.61	0.19	0.49	0.003
CAPS Total			0.01	0.01	0.36	0.029
HF-HRV			0.00	0.00	0.11	0.478
Recovery						
Overall						
Final model	0.67	0.00				< .001
PRE SAS						
Total			0.50	0.09	0.50	< .001
CAPS Total			0.01	0.00	0.58	< .001
HF-HRV			0.00	0.00	0.05	0.632
Housework						
Final model	0.52	0.01				< .001

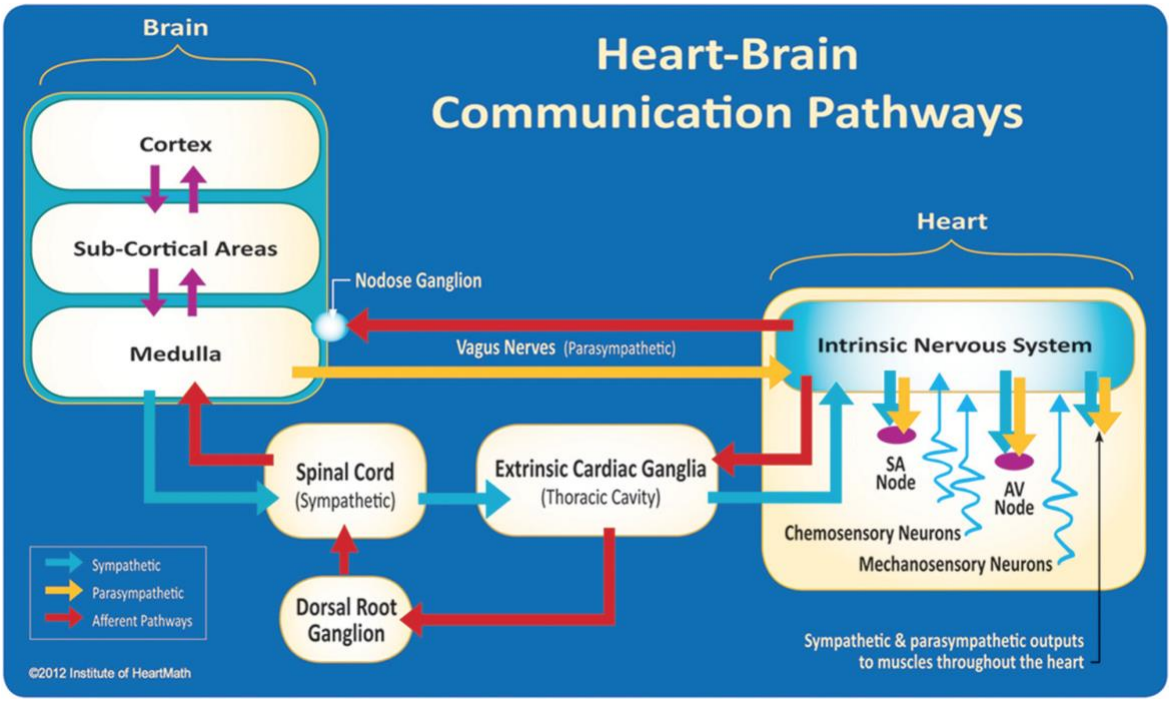
PRE SAS Total			0.65	0.11	0.71	< .001
CAPS Total			0.01	0.00	0.27	0.025
HF-HRV			0.00	0.00	0.08	0.515
Social/Leisure						
Final model	0.57	0.04				< .001
PRE SAS Total			0.43	0.13	0.39	0.002
CAPS Total			0.02	0.00	0.53	< .001
HF-HRV			0.00	0.00	0.19	0.081
Extended Family						
Final model	0.38	0.03				< .001
PRE SAS Total			**0.24**	**0.11**	**0.28**	0.041
CAPS Total			**0.02**	**0.00**	**0.54**	< .001
HF-HRV			**0.00**	**0.00**	**-0.18**	0.180
Family Unit						
Final model	0.27	0.01				0.007
PRE SAS Total			0.62	0.19	0.49	0.003
CAPS Total			0.01	0.01	0.34	0.033
HF-HRV			0.00	0.00	0.11	0.497

Note. Statistically significant effects ($p < .05$) are in bold type. SAS = Social Adjustment Scale; POST = posttreatment; PRE = pretreatment; $\mathbf{\Delta}R^2$ = change in R^2 when HRV was added to the model**.**

[NS] The addition HRV did not significantly change the $\mathbf{\Delta}R^2$ in any analysis.

Figure 1

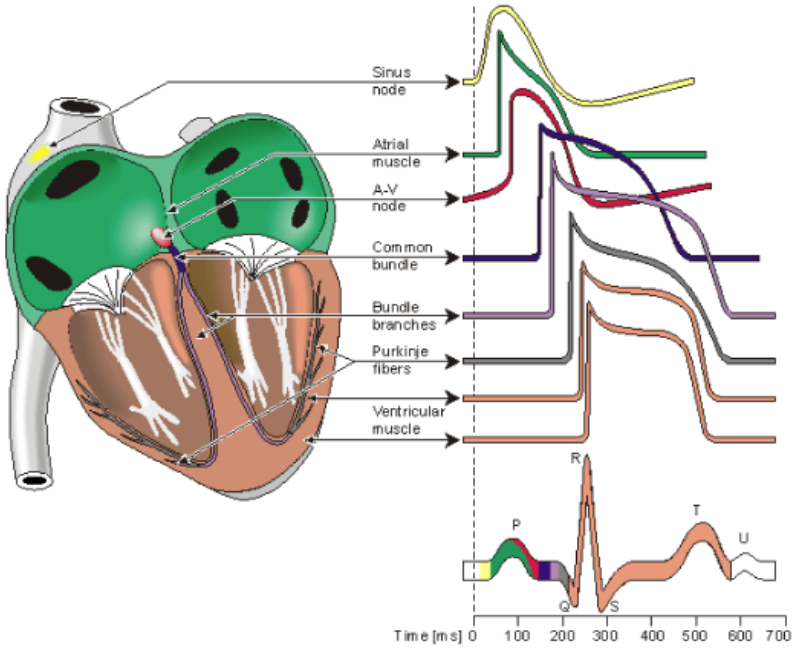
Communication Pathways Between the Heart and Brain



Note. From Science of the heart: Exploring the role of the heart in human performance (Vol. 2) by R. McCraty, 2015. Copyright 2015 HeartMath Institute.

Figure 2

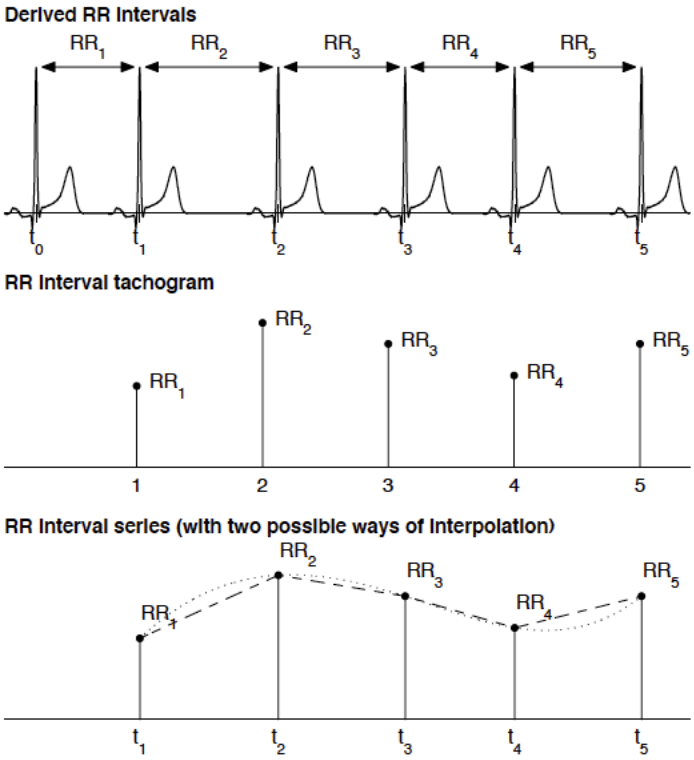
Electrical Output of a Healthy Heart



Note. The waveforms and latency shown approximate those of a healthy heart. From Kubios HRV User's Guide (ver 3.1, p. 11) by M.P Tarvainen, J. Lipponen, J.P. Niskanen and P.O. Ranta-aho, 2018, Kubios Oy. Copyright 2018 by Kubios Oy (limited company).

Figure 3

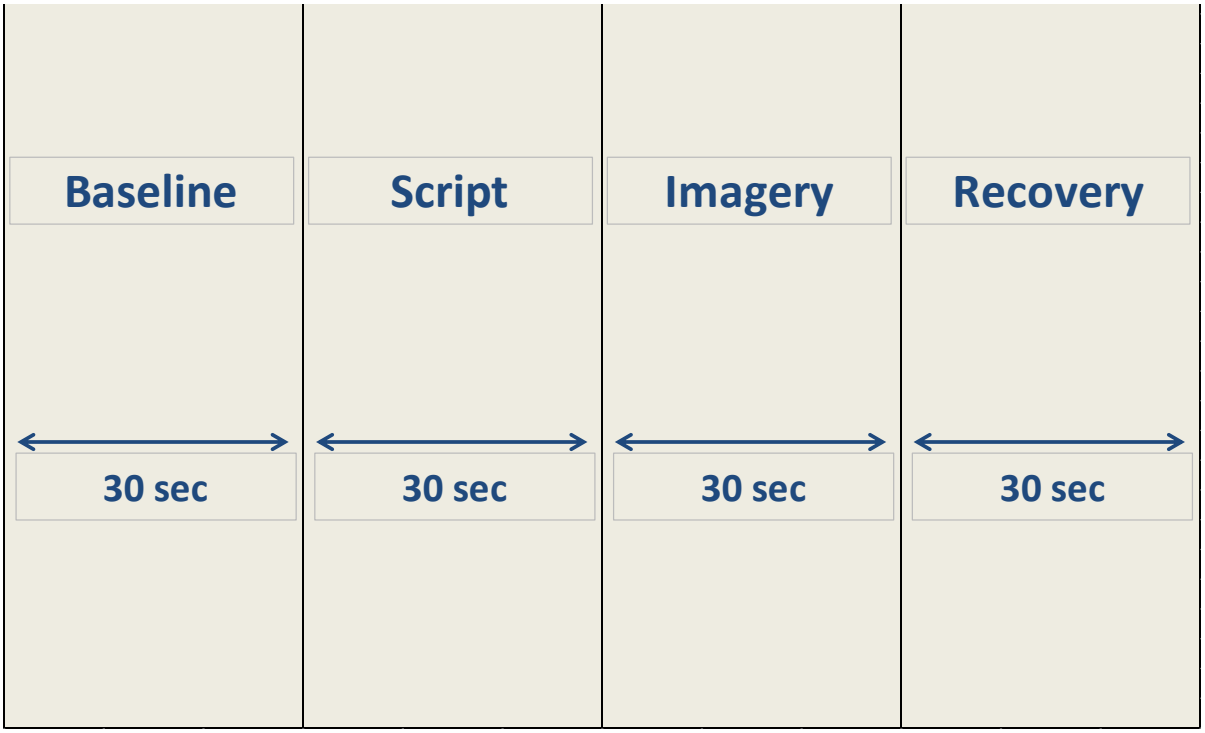
Measuring R-R Intervals from an ECG



Note. From Kubios HRV User's Guide (ver 3.1, p. 13) by M.P Tarvainen, J. Lipponen, J.P. Niskanen and P.O. Ranta-aho, 2018, Kubios Oy. Copyright 2018 by Kubios Oy (limited company).

Figure 4

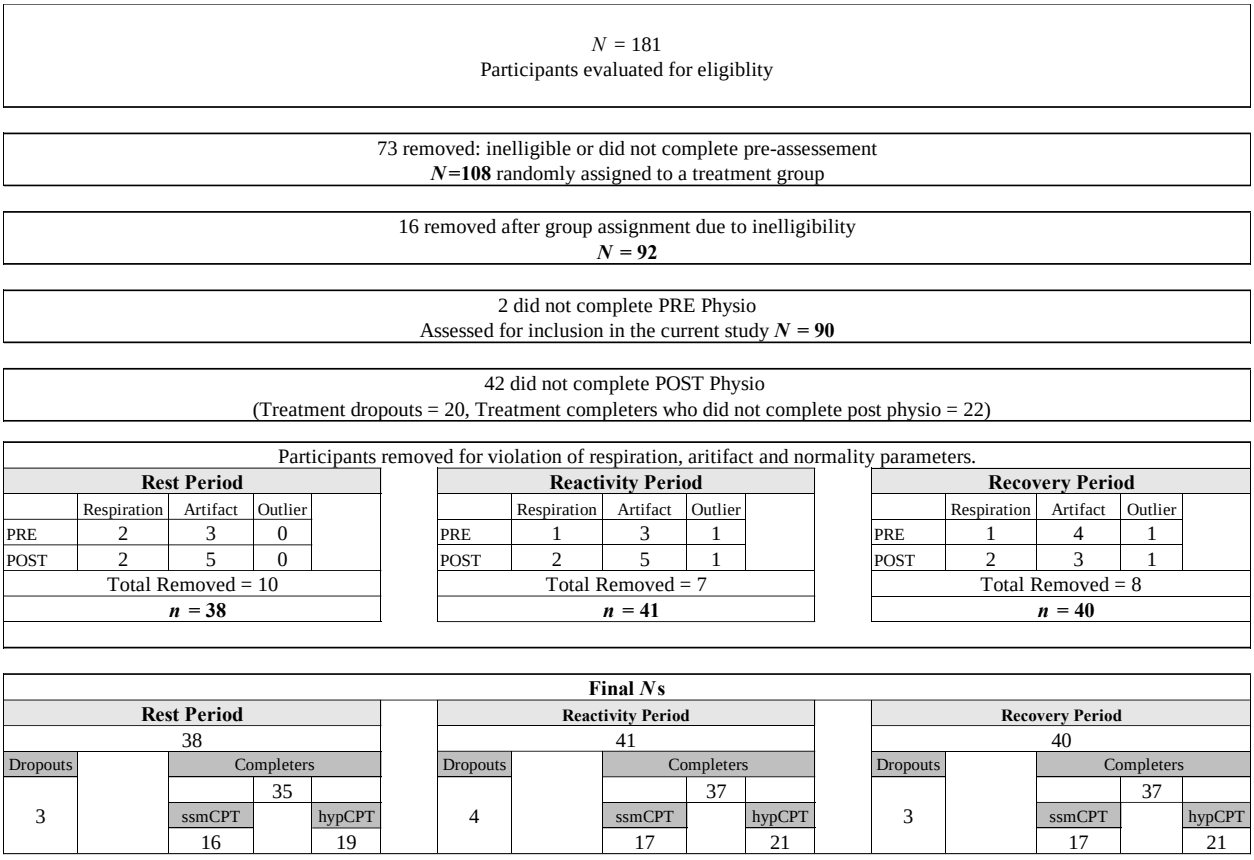
Script-Driven Imagery Procedure



Note. The recovery period following the trauma scripts was increased to one minute to allow for a more complete recovery. All other recovery periods remained 30 seconds long.

Figure 5

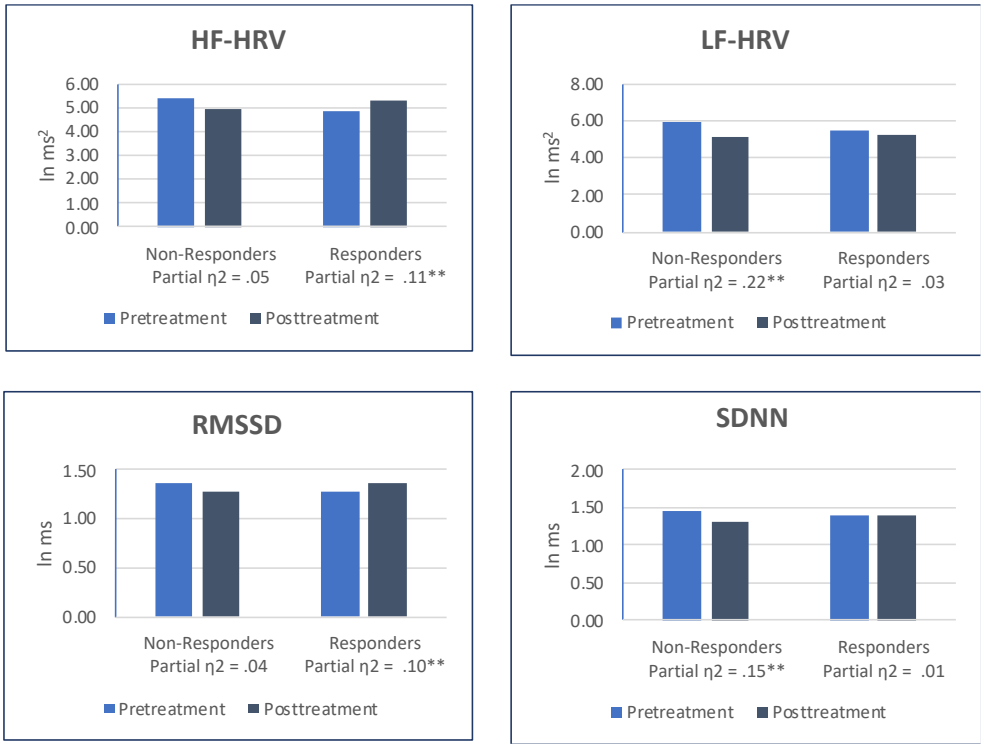
Participant Flow



Note. POST = posttreatment; PRE = pretreatment; ssmCPT = sleep and symptom monitoring plus cognitive processing therapy; hypCPT = hypnosis plus cognitive processing therapy.

Figure 6

Comparing PTSD Treatment Response Groups on HRV Changes Posttreatment During the Reactivity Period



Note. Due to normality violations, all variables were natural log transformed and therefore, HRV results are in log units. HF-HRV = high frequency heart rate variability; LF-HRV = low frequency heart rate variability; SDNN = standard deviation of all R-R intervals; RMSSD = root mean square of successive differences; ln ms = milliseconds in log units.

**p < .01