HEART RHYTHM VARIABILITY IN PERSONS WITH CHRONIC PAIN

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The present study evaluated the utility of heart rhythm coherence (HRC) feedback to reduce the reported pain intensity of patients enrolled in a multimodal pain management program. Participants were recruited and assigned to a usual treatment group (UT) or a heart rhythm coherence feedback group (UT+HRC). It was hypothesized that UT+HRC participants who achieved heart rhythm coherence would report a reduction of pain intensity, as measured by the McGill Pain Inventory. For those whose pain intensity decreased, it was also expected that their self reported levels of depression as measured by the Beck Depression Inventory-Second Edition and state anger as measured by the State Trait Anger Inventory would decrease. It is also hypothesized that with a reduction in pain levels, anger, and depression, blood pressure would also decrease among those who had high blood pressure prior to the intervention.

Multivariate analyses of variance (MANOVA) were used to investigate the relationship between treatment condition, coherence status and pain levels. A series of independent *t*-tests were utilized to investigate the change in pain, depression, and state anger from baseline to posttest, followed by Pearson product moment correlation coefficients on difference scores to understand the relationship between the outcome variables for Hypothesis 2. Standard multiple regression analyses were computed using difference scores to determine if the outcome measures were significant predictors of systolic blood pressure and diastolic blood pressure. Results indicated a failure to reject

the null with regard to hypothesis one. No relationship between treatment assignment, coherence status or pain levels were found. Hypothesis 2 was partially supported. Although there was a positive significant relationship between depression and anger when utilizing difference scores, these affective measures were not related to difference scores on either pain measure. In regard to Hypothesis 3, there was also a failure to reject the null. None of the outcome measures utilized in this study emerged as being significantly related to changes in systolic or diastolic blood pressure. Limitations of the study and implications for future research are offered.

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CHAPTER 1

INTRODUCTION

It is estimated that 50 million Americans live with chronic pain (Weiner, 2005) Chronic pain is defined as pain that may have "slow or sudden onset of any intensity from mild to severe, constant or recurring without anticipated or predictable end and a duration of greater than 6 months" (Thomas, 1997, p. 1389). Chronic pain may present as the central feature of disorders such as recurrent tension and migraine headache, fibromyalgia, reflex sympathetic dystrophy (RSD) and tempromandibular joint syndrome (TMJ). Further, it can occur in conjunction with disease process (e.g., cancer), follow damage to the central nervous system (e.g., stroke) or damage to other areas of the body, such as that frequently seen with low back pain. Chronic pain may continue even after the injury site has presumably healed. Turk and Monarch (2002) point out that beyond the physical effects of chronic pain, is its impact on a person's activities of daily living (e.g., grooming, chores), work abilities, and social activities. A person may experience a variety of emotions in response to chronic pain and its impact on his or her life, including anxiety, depression and anger.

Models of Pain

Gate Control Theory of Pain

In the 1970s, the gate control theory of pain (Melzack & Wall, 1965) gained widespread acceptance. The gate control theory included physiological mechanisms as a basis for pain, but also held that psychological factors could influence the experience of pain. Thus, this theory provided a way to account for the variation from person to

person in the experience of pain; that is, why one person may perceive particular noxious stimuli to be much more painful than another person would perceive it to be. According to the gate control theory, a “gate” mechanism is located in the dorsal horn of the spinal cord. Peripherally, the activity of fibers that transmit pain signals (nociceptors) can open the gate, whereas, activity of nonnociceptive fibers act to close the gate. Additionally, it is believed that central pathways descending from the brain may also act to open the gate, prolonging the perception of pain, or close the gate decreasing the perception of pain (Gatchel, 2005). This opening and closing of the neural gate is influenced by a person’s thoughts, feelings and behaviors. For example, positive thoughts and coping behaviors are believed to close the gate, while stress, feelings of sadness, anger and hopelessness, and behaviors such as sleeplessness, smoking, and inactivity are believed to open the gate (Gatchel, 2005).

Neuromatrix Theory

Melzack (1999) has extended the gate control theory into the neuromatrix theory. This theory builds upon the underpinnings of the gate control theory, but adds the component of stress. The neuromatrix theory holds the neurons of the body form a matrix whose initial form is influenced by genetics and later shaped by sensory experiences. Within this matrix is a cyclical processing and synthesis of nerve impulses, which Melzack (1999) refers to as a neurosignature. The experience of pain, according to this theory, is related to the combination of a person’s physiological matrix, sensory input, and cognitive processes, including the emotions of sadness or anger. In this manner, the neuromatrix model is largely consistent with the gate control theory.

However, as previously mentioned, this model differs from the gate control theory in that stress takes a central role in this model. Gatchel (2005) conceptualizes the interplay of stress and pain as a diathesis-stress model, whereby individual predispositional factors interact with acute pain (the stressor). As the pain is prolonged, this chronic pain becomes a stressor itself, resulting in sympathetic nervous system arousal.

Stress

Stress can result in activation of the sympathetic nervous system and secretion of glucocorticoids, including cortisol, from the adrenal cortex (Carlson, 2004; Maier, Watkins, & Fleshner, 1994). In the short-term, the release of cortisol benefits the organism during the fight or flight response by aiding in the breakdown of protein and its conversion to glucose, increasing blood flow to needed areas, and inhibiting temporarily unnecessary functions such as digestion. Although beneficial in the short-term, the prolonged release of cortisol can have adverse effects, including high blood pressure, infertility, immunosuppression, stunted growth, and muscle tissue damage (Carlson, 2004), as well as prolonged wound healing (Kiecolt-Glaser, Marucha, Malarkey, Mercado, & Glaser, 1995). According to the neuromatrix theory, the stressor of chronic pain contributes to prolonged cortisol release (Melzack, 1999). This places persons with chronic pain at increased risk for other adverse health conditions such as those mentioned above.

The transactional model of stress and coping (Lazarus & Folkman, 1984) asserts that stress is a manifestation of the interaction between the person and his or her environment. Stress results when an event or situation occurs that the person perceives

as surpassing his or her resources to cope with that situation. Personal injury or illness has long been conceived as a major life stressor (Holmes & Rahe, 1967) and the view of chronic pain as a stressor has been incorporated into the neuromatrix theory (Melzack, 1999). Additional major stressors for a person with chronic pain often include loss of financial stability and changes in family relationships (Hardin, 2000). Further, persons with chronic pain face other major life events (e.g., death of a loved one, sexual difficulties, changes in social activities) as well as minor daily hassles (e.g., being stuck in traffic, misplacing car keys) just as do persons without chronic pain. The more stress a person with chronic pain experiences, the more likely it is that pain will be exacerbated (Gatchel, 2005). In keeping the gate control and neuromatrix theories, Gatchel (2005) conceptualizes the relationship between pain and stress as a cycle whereby stressors increase pain and pain increases stress.

Research has supported the linkage between stress and pain. Flor & Turk (1989) conducted a review of psychophysiological research occurring between 1969 and 1987 and found that stress was associated with increased muscle tension in persons with tension headache and chronic low back pain. Further, in a sample of females with arthritis, increased stress predicted a rise in health complaints and increased negative affect (Skinner, Zautra, & Reich, 2004). Moreover, the increased negative affect was associated with increased pain. Additionally, among persons with sickle-cell disease, daily stressors and negative mood have been associated with increased same-day pain and predicted increased pain two days later, whereas positive mood was associated with decreased same-day pain and predicted decreased pain two days later (Gil, Carson, Porter, Scipio, Bediako, & Orringer, 2004).

Gatchel (2005) advocates for interventions that break the pain-stress cycle and asserts that people can learn to control their stress through learning how to relax. Indeed, relaxation strategies have been effective in reducing stress and pain in persons with spinal cord injuries (Hough & Kleinginna, 2002) and reducing pain intensity associated with tension and migraine headache (Fichtel & Larsson, 2001; Penzien, Rains, & Andrasik, 2002). Biofeedback strategies that incorporate relaxation have also been effective in reducing phantom limb pain as well as upper and lower back pain (Sherman, 2004), arthritis pain (Burke, Hickling, Alfonso, & Blanchard, 1985) and TMJ pain (Flor & Birbaumer, 1993; Gevirtz, Glaros, Hopper, & Schwartz, 1995).

Mood and Chronic Pain

Depression and Pain

Numerous studies have explored the presence of depression in medical patients, including those with chronic pain. Romano and Turner's (1985) review of the literature revealed considerable variability in the rate of depressive symptoms in medical outpatients, which ranged from 12% to 56%. They found even more increased variability in the rate of depressive symptoms among patients with chronic pain (10% - 100%). This variability may be due to the way symptoms are assessed (e.g., clinical interview, self-report questionnaire), type and location of pain, as well as individual differences in affect, cognitions, and behavior. Garnering further support for the link between depression and pain, Fishbain, Cutler, Rosomoff, and Rosomoff (1997) also conducted a review of 83 pain and depression studies occurring between 1980 and 1996. Their findings supported the conclusion that depression is more common in chronic pain

patients than in healthy controls. The numerous articles they reviewed indicated that there is a relationship between the severity of depression and severity and frequency of pain. This finding has been recently supported by the work of Bruehl, Chung, & Burns (2003) who assessed a sample of complex regional pain syndrome (CRPS; n = 34) and non-CRPS chronic pain patients (*n* = 50) enrolled in a multidisciplinary pain treatment program. They found that both groups had elevated scores on the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock & Erbaugh, 1961) at intake (CRPS group *M* = 13.9, *SD* = 1.35; non-CRPS group *M* = 16.6, *SD*= 1.21). Further, they found that scores on the BDI exerted a main effect on pain intensity ratings with greater depression being associated with greater pain intensity for both patient groups. Additionally, Ericsson, Poston, Linder, Taylor, Haddock and Forey (2002) found in a sample of patients with chronic pain, that a *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*, (*DSM-IV*; American Psychiatric Association, 1994) based diagnosis of depression at baseline predicted disability status two and one half years later. Other examples of this depression-pain link is provided by Nelson and Novy (1997), who found in a study of pain patients that the average score on the BDI was within the mildly depressed range (*M* = 15.82, *SD* = 9.77), and the work of Gaskin, Greene, Robinson, & Geisser (1992) who found that depression scores were the single most important predictor of pain intensity ratings in a sample of back, arthritis and fibromyalgia patients with chronic pain.

Although depression is often documented alongside the experience of chronic pain, so is the emotion of anger. Psychodynamic theory has long associated these two emotions. According to psychodynamic theory, depression may result when one is

unable to outwardly release angry feelings. The suppressed anger is turned inward against the self, resulting in problem states such as depression (Arieti & Bemporad, 1980; Bromberger & Matthews, 1996; Johnson, 1990; Alexander & French, 1948, as cited in Spielberger, Johnson, Russell, Crane, Jacobs, & Worden, 1985.) The depression – anger distinction becomes even more difficult when one takes into account gender differences in the expression of emotions. Although men often outwardly display their anger through verbal or physical aggression, women may become tearful when angry (Johnson, 1990; Kemp and Strongman, 1995). This tearfulness is often misclassified as a sign of being emotionally hurt or sad. Therefore, assessment of depression in persons with chronic pain should also investigate any underlying feelings of anger, which may be contributing to emotional and physical distress.

Anger and Pain

Anger has been defined as an unpleasant emotion that ranges in severity from mild annoyance to intense rage (Fernandez & Turk, 1995; Spielberger, Jacobs, Russell, & Crane, 1983). This emotion is accompanied by increased activity of the autonomic nervous system and the endocrine system, as well as increased muscle tension (Carlson, 1994, Johnson, 1990; McCraty, Atkinson, Tiller, Rein, & Watkins, 1995). Hardin (1998) conceptualizes this as a vicious cycle whereby a person experiences increased muscle tension following a period of frustration and anger, which in turn causes increased pain, which is met by even more frustration and anger. The reasons a person with chronic pain might become frustrated and angry are numerous and often include the difficulties faced in obtaining an accurate diagnosis of their medical

condition, an inability to become well despite attempting numerous treatment options, and pressure from family, friends, employers, and health care providers to resume normal activity levels (Witham, 1997). This anger may be directed toward a number of targets. Okifuji, Turk and Curran (1999) found a majority of chronic paint patients were angry with themselves and with health care providers. Other anger targets in their study included significant others, employers, insurance companies, attorneys, and the person who caused the injury. They also found that the intensity of anger was significantly related to pain, depression, and disability.

Anger may also impact persons with chronic pain in that it can lead to maladaptive behaviors such as avoidance of work activities, difficulty in relationships with health care providers, disruption in spousal relationships (Greenwood, Thurston, Tumble, Watters & Keefe, 2003) and contribute to spousal depression (Schwartz, Slater, Birchler, & Atkinson, 1991). Further, anger is believed to impact pain through its actions at the neural gate in the spinal cord. Recall that according to the gate control and neuromatrix theories, negative emotions including anger are believed to open the gate, allowing the passage of pain stimuli to the brain.

Within the literature, anger is often referred to and evaluated as either “state” anger or “trait” anger, terms widely adopted following the work of Spielberger et al. (1983). State anger is used to describe a transient emotion that can fluctuate in intensity, whereas trait anger reflects a personality tendency toward anger. Based on Spielberger and colleagues’ (1983) conceptualization, persons who possess trait anger are likely to perceive a wide variety of situations as anger provoking, and thus respond to these situations with an increased frequency and intensity of anger. Although state

anger has been considered an important predictor of pain ratings (Gaskin et al., 1992), the majority of literature has focused on the relationship between trait anger and chronic pain. While the definition of trait anger is likened to the previously mentioned work of Spielberger and colleagues (1983), the more widely recognized term for this construct is hostility. Hostility is commonly defined as a pervasive form of anger marked by cynicism and mistrust. The lack of a singular term for this pervasive personality pattern makes consumption of research literature difficult.

Despite differing terms for the concept, research has supported a link between a personality tendency toward anger and chronic pain. For example, trait anger was found to have a direct effect on ratings of pain with higher trait anger scores being positively related to higher indices of pain (Conant, 1998). Even among non-medical adults, the presence of high trait anger was related to physiological symptoms including sweating, shakiness, clenching of the jaw, as well as shoulder, back and neck tension (Deffenbacher, Oetting, Thwaites, Lynch, Baker, Stark, et al., 1996). The implications are that among persons with tempromandibular, shoulder, back or neck pain, this propensity toward anger could exacerbate pain both directly through tightening of the muscles as well as indirectly through its action on the neural gate.

The relationship between a personality tendency toward anger and chronic pain, however, may be complicated by other factors. Burns (1997) found that it was not the mere presence of hostility that was related to chronic pain, but rather an interaction between hostility and anger management style that is important. According to Johnson (1990), anger management techniques are often classified in three ways: (1) anger expression, (2) anger suppression, and (3) anger control. Anger expression involves the

outward display of anger through verbal and/or physical means. Anger suppression involves the keeping of angry feelings to one's self as opposed to expressing them verbally or physically. Anger control relates to controlling angry feelings and attempting to solve the underlying problem. As Greenwood et al. (2003) point out, anger control is generally viewed as a positive way of managing anger, however extremely high levels of control may impede appropriate expression of the emotion.

The role of anger expression and anger suppression both have been implicated in chronic pain but the literature is confusing and often conflicting in its findings. For example, greater outward anger expression was related to higher ratings of pain intensity in a sample of CRPS patients but this finding did not hold for non-CRPS patients who were described as predominantly having myofascial pain (Bruehl et al., 2003). In other studies, it is suppression of anger that significantly predicted pain intensity and pain behaviors, accounting for a significant increment in variance above that accounted for by depression measures among a sample of chronic pain patients, the majority of whom had musculoskeletal low back pain (Kerns, Rosenberg, & Jacob, 1993). Further, in a sample of college students, suppressed anger was a significant predictor of somatic complaints as measured by a modified version of the Adult Health Checklist (Forgays, Richards, Forgays, & Sujan, 1999). Additionally, Burns, Johnson, Devine, Mahoney, & Pawl (1998) found that suppressed anger was related to higher depression scores and lower physical activity among men with chronic pain, even after the men completed a 28-day multidisciplinary pain treatment program. This relationship, however, was not found for females in their study.

There are theoretical views regarding differential anger management style based

on gender; however these have seldom been supported by empirical studies. For example, Collier (1982) states that women in our society are taught to hide or suppress their anger, however, in samples of college students, no gender differences on anger suppression were found (Kinney, Smith, & Donzella, 2001; Kopper & Epperson, 1996). Regarding anger expression, several studies found no differences based on gender (Bartz, Blume, & Rose, 1996; Stoner & Spencer 1987). Differentially, Kinney et al. (2001) found that males reported being more verbally aggressive than females. In a factor analytic study of 24 trait anger measures, Martin, Watson, and Wan (2000) found that the overall factor structure of trait anger did not differ for men and women, however, of the three factors that emerged from their study (angry affect, aggression, and cynicism/hostility), mean factor score differences were found, with men having higher behavioral aggression scores and women scoring higher on angry affect.

Few studies have explicitly investigated the interaction of gender and anger among chronic pain patients. In a study previously discussed, Burns et al. (1998) utilized the Anger Expression Scale (AES; Spielberger et al., 1985) and found that men with chronic pain who endorsed a high level of suppressed anger also had higher depression scores and lower physical activity than men with a low level of anger suppression. Additionally, men in their study who expressed a high degree of anger made fewer gains in physical lifting capacity than men who endorsed less outward displays of anger. These effects, however, were not found for females in their study. In a separate study, Burns (1997) also used the AES as well as the Cook Medley Hostility Scale (Cook & Medley, 1954) and found no gender differences among chronic low back pain patients on hostility or anger expression, but males who had higher anger

expression scores experienced increased paraspinal muscle reactivity during an anger recall task. Therefore, among chronic pain patients, anger management style may be a more important consideration in the treatment of male rather than female patients, particularly when extreme suppression or expression tendencies are present.

Due to the various ways in which anger has been measured (state anger, trait anger, hostility, anger expression, anger suppression) results in the literature have varied with regard to the chronic pain literature. Although the data is mixed with regard to a clear relationship between pain and anger suppression or expression, the data is clearer in documenting the presence of anger (state and trait) among persons with chronic pain. Thus is an important consideration for intervention in persons with chronic pain, not only due to its ability to exacerbate pain symptoms, but also its impact on a patient's relationships with others (Greenwood et al., 2003; Schwartz et al., 1991).

Interventions targeted at teaching new coping strategies that can be applied in a variety of circumstances may reduce pain and disability by providing people with alternate ways of responding other than with anger. Such interventions may be helpful by decreasing sympathetic activation, closing the gate within the spinal cord, decreasing maladaptive behavior patterns such as work avoidance, and decreasing conflict in relationships with family, friends, and health care providers.

Heart-Rhythm Coherence

Heart Function and Blood Pressure

The heart is a four-chambered muscular pump. The right side of the heart is involved in receiving deoxygenated blood from the body and pumping that blood into the

lungs where it is oxygenated. The left side of the heart receives oxygenated blood from the lungs and pumps blood through the rest of the body. The top chambers of the heart are known as atria; the bottom chambers are the ventricles. The natural pacemaker of the heart is the sinoatrial node (SA node), which is located on the posterior wall of the right atrium, beneath the superior vena cava (Katz, 1977; Papillo & Shapiro, 1990). The cells of the SA node generate an electrical impulse that spreads across both the left and right atria causing them to contract. This contraction results in the atrial blood being emptied into the ventricular cavities. The impulse from the SA node is also sent to the atrioventricular node (AV node), located on the interatrial septum close to the tricuspid valve (Molson Medical Informatics Project, 1999). The AV node then transmits the impulse to the ventricles causing them to contract. This contraction results in blood being sent either to the lungs (from the right ventricle) or the rest of the body (from the left ventricle).

Papillo and Shapiro (1990) point out that fibers from the sympathetic and parasympathetic branches of the autonomic nervous system terminate on cells in the SA node, and thus act to influence heart rate by increasing or decreasing the firing rate of the SA node. During relaxation, parasympathetic fibers release acetylcholine on SA nodal cells, which alters the depolarization of those cells and results in a reduced heart rate (Papillo & Shapiro). Conversely, sympathetic nervous system fibers release norepinephrine, which also alters the depolarization of SA cells, resulting in an accelerated heart rate (Papillo & Shapiro).

On average, a healthy human heart beats approximately 60-70 times per minute [National Heart, Lung, and Blood Institute (NHLB), 2004]. The contraction of the heart is

known as systole, and is the period when blood pressure is highest; the time between heart contractions when blood pressure dips is known as diastole (NHLB, 2004; Thomas, 1997). Blood pressure is reported as a series of numbers, systolic pressure over diastolic pressure, and is commonly measured with a sphygmomanometer (blood pressure cuff). According to the NHLB, normal blood pressure is lower than 120/80 mmHg. Pre-hypertension occurs when systolic pressure is 120-139 and diastolic pressure is 80-89 and is likely to develop into high blood pressure unless preventative action is taken (NHLB). Stage 1 high blood pressure is diagnosed when systolic levels are 140-159 and diastolic levels are 90-99; Stage 2 high blood pressure occurs when systolic/diastolic blood pressure is greater than 160/100 (NHLB). High blood pressure increases a person's chances of developing kidney problems, or having a heart attack or stroke. The NHLB states that when systolic and diastolic readings fall into different blood pressure categories, the higher category should be used in classifying blood pressure.

Copious amounts of research have indicated a link between anger and blood pressure. Two recent examples are the work of Waldstein et al., (2000) who noted increased blood pressure during transient anger states, and the work of Schum, Jorgensen, Verhaeghen, Sauro, & Thibodeau (2003) who found trait anger to be associated with increased blood pressure. It is also important to note that blood pressure is lowest during sleep and increases when a person arises. Substances such as caffeine and nicotine may increase blood pressure while blood pressure may decrease for several hours following exercise (Thomas, 1997). Additionally, it may rise during emotional states such as when a person is excited or nervous (NHLB). Despite

these potential influences, the NHLB state that blood pressure stays relatively stable during waking hours when the person is sitting or standing still.

The electrocardiogram (ECG; EKG) is a non-invasive procedure used to detect the electrical activity of the heart. The ECG procedure utilizes electrodes placed on the body for recording purposes. Ambulatory ECG monitoring can occur via a Holter monitor, which a person wears for 12- 24 hours, with 5-7 electrodes being attached to the chest. Alternately, stationary monitoring is often conducted in a medical or laboratory setting and usually involves 10-12 electrodes placed on the chest, back, wrists, and ankles (Texas Heart Institute, 2005). The electrical activity of the heart is recorded and analyzed so that the presence of abnormal cardiac rhythm or damage to the heart muscle can be detected. The recordings are mathematically transformed into power spectral density (PSD).

The power spectrum can be divided into various frequency ranges. Akselrod, Gordon, Ubel, Shannon, Barger, and Cohen (1981), divided the spectrum into low frequency (LF; 0-.1 Hz), medium frequency (MF; approximately .1-.3 Hz) and high frequency (HF; .3-.5 Hz) ranges. Others have since adjusted these values based on continuing work with the ECG power spectrum. The American Heart Association (AHA, 1996) now recommends the following values be employed when evaluating frequency domain measures of heart rate variability (HRV): very low frequency (VLF = < .04Hz), LF = .04-.15 and HF = .15-.4. HF has been associated with the influence of respiration on the heart (Appelhans & Luecken, 2006; Cevese, Gulli, Polati, Grottin, & Grasso, 2001; Turjanmaa, Kalli, Sydanmaa, & Uusitalo, 1990) and is mediated by parasympathetic activity (Acharya, Joseph, Kannathal, Lim & Suri, 2006; Akselrod et al.,

1981; Neumann, Waldstein, Sollers, Thayer, & Sorkin, 2004). The LF components associated with the baroreceptor reflex (De Jong, & Randall, 2005) are influenced by sympathetic and parasympathetic activity (Akselrod et al., 1981; Neumann et al., 2004), with research supporting the dominance of parasympathetic over sympathetic activity (Cacioppo, et al., 1994; Taylor, Carr, Myers, & Eckberg, 1998). The VLF components are believed to be largely influenced by circadian rhythm (DeJong, & Randall, 2005). The LF/HF ratio is viewed as a reflection of sympatho/vagal balance (Task Force, 1996). The Task Force points out that values for VLF, LF, and HF may reported as the absolute value of power (ms^2) or LF and HF may be reported as normalized units (n.u.), which represent "the relative value of each power component in proportion to the total power minus the VLF component" (p. 358).

Rationale and Mechanism of Action

HRV refers to variation in the naturally occurring beat-to-beat changes in heart rate. People have greater HRV when they are relaxed and breathing in a regular or slow pattern (Nolan, 2005). Conversely, sympathetic nervous system activity such as that seen during a stress response decreases HRV (Peper, Harvey, Lin, Tylova, & Moss, 2007). Thus, HRV can be used as an indicator of a response to acute stress and over activity of the sympathetic system (MacArthur & MacArthur, n.d.). HRV declines as part of the ageing process (Bonnenmeier et al., 2003; Nolan, 2005). Decreased HRV has also been predictive of future cardiac events and cardiac mortality (see DeJong & Randall, 2005 for a review) and is present after myocardial infarction and heart transplantation (Task Force, 1996). Of note, studies of persons with complete spinal

cord injuries in the cervical region have demonstrated mixed results with regard to the ability to detect LF activity (Task Force, 1996).

The influence of cigarette smoking on HRV has been investigated with mixed results. Kageyama et al. (1997) found smoking status (non smoker/ moderate smoker/ heavy smoker) not to be associated with HRV. However, Barutcu et al. (2005) found baseline LF/HF ratio to be higher in heavy smokers (> 24 cigarettes per day) than non-smokers. Additionally, Stein, Rottman, & Kleiger (1996) found that smoking cessation via nicotine patch increased HRV, with further increases in HRV being found with cessation of nicotine patch use.

The influence of caffeine on HRV has also met with mixed results. For example, Rauh, Burkert, Siepmann, and Mueck-Weymann (2006) found that among habitual caffeine users, ingestion of 100mg or 200mg doses of caffeine did not result in significant HRV differences up to 90 minutes after caffeine ingestion. However, among those who typically consume less than one dose per day of caffeine, administration of 100mg or 200mg caffeine resulted in decreased HRV parameters (Sondermeijer, van Marle, Kamen, & Krum, 2002).

Emotions are also related to HRV. Illustrating this relationship is the study of Neumann et al. (2004) who measured HRV via ECG recording in 80 females to examine the relationship of anger in cardiovascular recovery from an anger recall task. The participants were asked to recall an event that still evoked feelings of anger. Following the recall task, participants were either engaged in a distraction task or standard recovery (no distraction task). Those engaged in a distraction task reported spending significantly less time thinking about the event. Cardiovascular measures

indicated that recall of the angry event was related to increased diastolic and systolic blood pressure, heart rate, and LF power (.04-.15 Hz) with significant decreases in HF power (.15-.4 Hz). During recovery, hostility predicted a slower recovery of systolic blood pressure, but was not significantly related to HRV components. The distraction condition predicted faster heart rate recovery, lower LF power, and higher HF power during recovery. The distraction participants also endorsed lower state anger during recovery than did members of the no distraction group. In numerous studies, anxiety and depression have also been linked to decreased HRV parameters (see Appelhans & Luecken, 2006 for a review). For example, in a sample of non-medicated, young adults with major depressive disorder, decreased HF values were found in comparison to non-depressed controls (Udupa et al., 2007) and in a sample of elderly participants with depression, both LF and HF values were significantly lower than non-depressed controls (van der Kooy, van Hout, van Marwijk, de Haan, Stehouwer, & Beekman, 2006).

Researchers at the Institute of Heart Math (IHM) have begun investigating the interplay between emotion focused interventions and HRV. Two primary techniques utilized by IHM (Boulder Creek, CA) are the Freeze-Frame® and the Heart Lock-in® procedures. The exact Freeze-Frame procedure has varied from study to study (Barrios-Choplin, McCraty, Sundran, & Atkinson, 1999; McCraty, Atkinson, & Lipsenthal, 2000; McCraty & Tomasino, 2004; McCraty, Atkinson, & Tomasino, 2003; Tiller, McCraty, & Atkinson, 1996) but generally incorporates two core activities: 1) a shift of attention away from distressing thoughts/emotions, with the redirection of attention to the area around the heart and 2) the generation of sincere feelings of appreciation, care

and/or experiencing of a fun feeling or time. With practice, the Freeze-Frame technique is meant to be a brief (one minute) intervention to be used to manage day-to-day stressful events by facilitating a shift from unpleasant to pleasant thoughts, which is believed to facilitate a change from primarily sympathetic activity to a balance of the autonomic system (McCraty et al., 2003). The Freeze-Frame is a cognitive technique that does not require the use of a computer or software to learn. The Heart Lock-in procedure is an extended Freeze-Frame, lasting five minutes or more.

Efficacy Information

In order to evaluate the effects of anger and appreciation on HRV following Freeze-Frame technique training, McCraty et al. (1995) analyzed the HRV of 24 healthy adults taken from successive discrete RR interval (time interval between consecutive heartbeats) series from an ECG signal. All 24 subjects were trained in the Freeze-Frame technique. Of those, 12 were asked to recall a situation in their lives that still aroused feelings of anger. The other twelve participants were asked to experience feelings of appreciation. Participants were matched on age and gender and had no history of cardiovascular disease. Each recall session lasted five minutes. This time limit was reportedly imposed for two reasons. First, it was the minimum time required to obtain an accurate power spectrum reading and second, this is considered the maximal amount of time most subjects could sustain emotional focus. McCraty and colleagues reported that both emotions resulted in activation of the autonomic nervous system (as measured by LF + MF + HF), however anger resulted in a significant increase in LF power (.01-.08 Hz) with no change in HF (.15-.5 Hz) power, while appreciation resulted

in a significant increase in LF and a non significant increase in HF power. MF (.08-.15 Hz) significantly increased during anger and appreciation, however, the shift in MF during appreciation was greater than that seen with anger. Of note, McCraty et al. use different LF and HF Hz ranges than that proposed by the AHA (previously discussed). Based on the ranges used in this study, both emotional states resulted in changes in the LF, MF, and HF ranges. Although the study utilized a small number of participants, it provided encouraging results for further investigation of the Freeze-Frame technique in assisting clients to shift from angry emotions that are accompanied by sympathetic activation to feelings of appreciation and caring, associated with parasympathetic activity.

In a different study, Tiller et al. (1995) monitored ECG readings in a sample of adults previously trained in the Freeze-Frame technique. Participants were monitored in a laboratory setting as well as in a work setting. In the laboratory, participants were asked to employ the technique for five minutes while ECG readings were obtained. In the work setting, participants were asked to use the Freeze-Frame technique at least three times during a 24-hour period. In the laboratory setting, HF (.15 - .5 hertz) and MF (.05-.15 hertz) power significantly increased when participants utilized the Freeze-Frame technique, with a non-significant decrease in LF power (.01-.05). In the work setting, significant increases in MF and HF power occurred as well as significant decreases in LF power during the Freeze-Frame task. In this study, Tiller et al. also used differing Hz ranges. In order to review these results in keeping with AHA recommendations, the LF power in the Tiller et al. study would correspond generally

with the VLF components and the MF power range would correspond with the LF power range in the AHA guidelines.

Other research by IHM has implicated the Freeze-Frame technique as a component in successful training programs in a variety of settings and across numerous age groups. For example, when implemented as part of a two-day Power to Change Performance Program for correctional officers with outcome measures were completed prior to the two-day workshop and again 90 days post workshop completion (McCraty, Atkinson, Lipsenthal, & Arguelles, 2003), it was found that participants reported a significant increase in positive outlook and significant reductions in anger as measured by the Personal and Organizational Quality Assessment (POQA; Barios-Choplin & Atkinson, 2000) and hostility, as measured by the Brief Symptom Inventory (BSI; Derogatis, 1993). Additionally, significant reductions in systolic and diastolic blood pressure were found along with significant increases in LF/HF power.

When the Freeze-Frame technique was used as part of a stress prevention program for Motorola employees that consisted of one day of intensive training with encouragement to practice the seminar techniques daily for 6 months, total power declined from pre to posttest, which the authors interpreted as a decline in autonomic resting levels (Barrios-Choplin, McCraty, & Cryer, 1997). Additionally, five participants in this study were classified as hypertensive at the outset of the study and were able to reduce their blood pressure while using the Freeze-Frame technique and continued to demonstrate reduced blood pressure readings at the six-month mark.

When researchers from the Stanford Center for Research in Disease Prevention (Luskin, Reitz, Newell, Quinn, and Haskell, 2002), provided 10 weekly sessions (75

minutes per session) of a stress management program including the Freeze-Frame and Heart Lock-in techniques to a sample of congestive heart failure patients (age = 70+ years), significant improvements were found on measures of depression (Geriatric Depression Scale; Yesavage & Brink, 1983), and stress (Perceived Stress Scale; Cohen, Kamarck, & Mermelstein, 1983). However, no significant changes occurred in HRV.

Additional studies utilizing the Freeze-Frame procedure as part of a stress management/self regulation training program have shown promise. A draw back of these studies is that they did not incorporate HRV as an outcome measure. For example, in a study of hypertensive employees from a global information technology company, Freeze-Frame and Heart Lock-in techniques were used as part of a 16-hour stress management program. Results indicated a significant decreases in systolic blood pressure, a significant increase in positive outlook and significant decreases in stress symptoms as measured by the POQA, and decreased depression and phobic anxiety as measured by the BSI when baseline measures were compared with a three-month follow-up (McCraty et al., 2003). Further, in a sample of patients with both Type I and Type II diabetes, the Freeze-Frame and Heart Lock-in techniques were used as part of a two-day wellness program with two-hour follow-up sessions conducted once per month for three months (McCraty et al., 2000). It was found that when baseline was compared to three-month follow-up data, significant decreases in psychological distress were found, as measured by the following BSI scores: Global Severity Index, Positive Symptoms Distress Index, Positive Symptom Total, Depression, Anxiety, Phobic Anxiety, Somatization, Interpersonal Sensitivity, Paranoid Ideation, and Psychoticism.

Participants also reported significantly improved quality of life, and a significant reduction in the perceived impact of stressful events. In another study, which utilized the Freeze-Frame procedure as part of one six hour self-management training program for information technology employees, with two, three-hour sessions over the next six-weeks, significant reduction in self reports of anger, depression, anxiety, fatigue, sleeplessness were found, as well as significant increases in the self report of peacefulness and vitality (Barrios-Choplin et al., 1999).

Alternate Recording Methods of HRV

An easier to use method of approximating HRV is recording the volume of blood with each pulse (BVP) that passes over a photoplethysmographic optical sensor (PPG) placed at the fingertip or earlobe (McCraty & Tomasino, 2004). Such sensors work by shining an infrared light through body tissue. As Peper et al. (2007, p. 55) explain, "the amount of light that returns to a PPG sensor's photodetector is proportional to the volume of blood in the tissue." They go on to state "because blood volume in the arteries and capillary bed increases with each arterial pulsation, heart rate can be estimated from the BVP." The use of a PPG is a much more convenient procedure than ECG, as multiple recording sites are unnecessary and electrodes are not taped to the skin surface. In this manner, HRV is not meant for the detection of heart damage, but rather to evaluate heart wave patterns and the effects of thoughts and emotions on the body. As Peper et al. (2007) point out, there are several commercially available units that have fostered the transition of HRV out of the laboratory and into the marketplace. A computer assisted training package called the Freeze-Framer® (Quantum Intech,

Inc., http://www.freezeframer.com) utilizes biofeedback to teach regulation of heart rhythm patterns. The BVP is measured by the sensor and then displayed on a computer screen. An erratic wave pattern is shown on the screen during emotional states such as frustration, anger and anxiety, as negative emotions lead to irregular heart rhythms and a lack of balance between the sympathetic and parasympathetic branches of the autonomic nervous system (Heart Math, 2001). The goal is to train participants to achieve a smooth sine wave like pattern to their heart rhythms, as the sine wave pattern is reflective of a more harmonious balance between the sympathetic and parasympathetic systems within the body (McCraty & Tomasino, 2004). Specifically within this system, physiological coherence is depicted "by a smooth, sine wave-like pattern in the heart rhythms and a narrow-band, high-amplitude peak in the low frequency range of the HRV power spectrum, at a frequency of about 0.1 hertz" (McCraty, & Tomasino, 2004, p. 3). The Freeze-Framer software analyzes the heart rhythm patters and computes a coherence ratio (low, medium, high) for each training session. The sum of low, medium, and high ratio is equal to 100 for each training session.

McCraty and Tomasino (2004) report that this system has been useful in many chronic conditions including fibromyalgia, chronic fatigue, and hypertension, in addition to being particularly effective in pain management. During personal communication with R. McCraty (October 10, 2005) it was ascertained that this assertion regarding pain management was based on anecdotal information from burn clinics. To date, the utility of this program in treating persons with chronic pain has not been specifically investigated and is the aim of this study. As research available for review at the time of

this study's proposal was based on ECG data rather than that collected by the Freeze-Framer software system itself, this study is expected to contribute to the literature regarding the utility of this software program.

Hypotheses

1) H_0: There will be no difference in reports of pain intensity between those in the coherence training group (UT+HRC) who achieve a coherent pattern to their heart rhythms (M+H > L) and other participants.

   H_1: UT+HRC participants who achieve a coherent pattern to their heart rhythms (M+H > L) as measured by the Freeze-Framer software will report decreased pain intensity.

2) H_0: There will be no relationship between depression, state anger or pain intensity.

   H_1: Depression and state anger will decrease as pain intensity decreases.

3) H_0: There will be no relationship between blood pressure, pain scores, depression scores or anger scores.

   H_1: As pain intensity, anger, and depression scores decrease, blood pressure levels will also decrease among those who have pressure readings > 120/80 mm Hg at baseline.

CHAPTER 2

METHOD

Participants

Following Institutional Review Board (IRB) approval at the University of North Texas, participants were recruited from High Point Rehabilitation Center (HPRC), a multidisciplinary chronic pain management center in Arlington, Texas. Arlington is located within the greater Dallas-Fort Worth metropolitan area. After many months of data collection, IRB approval was obtained for participant recruitment from another multidisciplinary chronic pain management center, North Texas Pain Recovery (NTPR), in order to increase the pool of available participants. NTPR is also located in Arlington, Texas. Both facilities hold current accreditation by the Commission on Accreditation of Rehabilitation Facilities (CARF). These multi-modal pain management programs incorporate various therapeutic modalities such as individual and group psychotherapy, biofeedback, aquatherapy, and physical rehabilitation. Participants are typically approved for treatment by worker's compensation or private insurance on an incremental basis. Patients often receive 5-10 days of approved treatment at a time, with possible reauthorization that may extend their chronic pain treatment for a period totaling 30 days.

Eligibility criteria for inclusion in the study included the presence of current pain symptoms that persisted for a period of at least six months. Additionally, all participants had worker's compensation or private insurance approval to participate in a chronic pain treatment program for at least 10 days. All participants were experiencing chronic pain that was deemed not primarily due to a malignant condition such as cancer or migraine

or tension headache. Additionally, participants were 18 years of age or older and had sufficient proficiency in English to comprehend and complete questionnaires and participate in therapy.

Measures

Demographic Measure

Participants were asked to complete a demographic questionnaire (see Appendix A) for analysis of demographic information as control for relevant characteristics.

Beck Depression Inventory - Second Edition (BDI-II)

The BDI-II (Beck, Steer, & Brown, 1996) is a self-report measure of symptoms associated with depression. It is a revised version of the original Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock & Erbaugh, 1961), which has been used in assessing patients with chronic pain (e.g., Bruehl et al., 2003; Burns et al., 1996; Nelson & Novy, 1997). The revised version remains a recommended tool for the assessment of persons with chronic pain (Gatchel, 2005). The inventory consists of 21 items answered by the endorsement of one of four ratings, ranging from 0 (*not present*) to 3 (*severe*). Scores of thirteen or less are reflective of minimal depression. Scores from 14-19 are indicative of mild depression, while those in the 20-28 range are considered moderate, and those in the range of 29-63 are indicative of severe depression.

Internal consistency has been reported at .90 within a chronic pain population

comprised of both male and female patients (Nelson & Novy, 1997). Convergent and divergent validity have been established in studies by Beck, Steer, and Brown (1996), Segal, Coolidge, Cahill, and O'Riley (2008), and Steer, Ball, Ranieri, and Beck (1997).

State Trait Anger Expression Inventory (STAXI)

The STAXI (Spielberger, 1988) is a 44-item self- report questionnaire designed to measure the experience, expression and control of anger in adolescents and adults. It is designed to be brief, taking approximately 15 minutes to complete. The questionnaire consists of three parts: "How I feel right now," "How I generally feel," and "How I generally react when angry or furious." Each part uses a 4-point Likert-type rating scale (1 = *not at all or almost never through* 4 = *very much so or almost always*). Numerous possible scores may be derived from the items: State Anger, Trait Anger (includes Angry Temperament and Angry Reaction subscales) as well as Anger Expression-Out, Anger Expression-In, Anger Control Scale and an Anger Expression Index. The State Anger component ("How I feel right now") is to be used in this study.

The STAXI has a history in the chronic pain literature as well as its earlier precursors, the State-Trait Anger Scale (STAS; Spielberger, 1983) and the Anger Expression Scale (Spielberger et al., 1985). Internal reliability estimates for the STAXI range from .70 - .93 with two-week test-retest correlations ranging from .62 to .81, excluding the State anger scales, as these are expected to vary over time (Hollander, Cohen, & Simon, 2000). Spielberger and colleagues (1983) demonstrated small to moderate (.22-.41) significant correlations between state anger and trait anger. This is expected, as in keeping with the theoretical development of the measure, as elevated

trait anger scores reflect a tendency to respond with more frequent and intense episodes of state anger.

The McGill Pain Questionnaire (MPQ)

The MPQ (Melzack, 1975) measures a person's subjective pain experience across sensory-discriminative, affective-motivational, and evaluative-cognitive domains (Burckhardt & Jones, 2003). It is a brief measure, requiring approximately 5-15 minutes to complete and was developed for use with adult chronic pain patients. Respondents are read a list of 78 words reflecting the three aforementioned domains. The words are arranged into 20 subclasses and respondents are instructed to select one word from each subclass if a word in the grouping describes their present pain. Each word in a grouping receives a ranked score, so that the first word in the grouping is a 1, the second word in the grouping is a 2, and so on. If no word from that grouping fits, then no word from that subclass is chosen. The Pain Rating Index (PRI) is based on the rank value of the words and can range from 0-78. The second index obtained is a total of the number of words chosen (NWC), which can range from 0-20. The third index is known as the Present Pain Index (PPI) and is comprised of a one item, five-point scale designed to serve as an indicator of overall pain intensity as the time of questionnaire completion.

The MPQ has been widely used in the assessment of pain, with questionnaires in 12 different languages being developed from its initial format (Melzack & Katz, 1992). Burckhardt & Jones (2003) found that clients tended to choose the same words in the PRI and report the same PPI level across a three to seven day test-retest interval.

According to the Measurement Excellence and Training Resource Information Center (METRIC; 2004) internal consistency reliability estimates range from a low of .46 on the evaluative component (derived from items 1-10), .71 on the affective component (derived from items 11-16), .78 on the sensory component (derived from item 16), and .84 for the total scale. The PRI and PPI are to be used in this study.

Procedure

Patients with chronic pain were recruited for this study from multidisciplinary pain management clinics, from which the client was already seeking services. Clinic staff alerted the researcher to new participants enrolling in the multidisciplinary chronic pain treatment programs with a minimum of 10 days of treatment approved by worker's compensation or private insurance. The researcher then offered each of these participants an opportunity to participate in this research study. The purpose and requirements of the study were explained and written informed consent was obtained. Because participants enter and exit the multidisciplinary pain treatment program at differing points in time, it was not possible to randomly assign participants to groups or match them on various characteristics prior to beginning the study. Thus participants were assigned to groups as they entered the study, one entering the coherence training group (UT+HRC) and the next entering the usual treatment (UT) group in an alternating manner.

Demographic information was collected through participant interview (see Appendix A). Information collected included, age, gender, race/ethnicity, marital status, approximate annual household income, educational attainment in years, work status,

medical diagnosis, medications, approximate number of caffeinated drinks consumed daily, daily nicotine use, and whether the client is currently involved in litigation regarding the basis of their injury/pain. Participants then completed the pain inventory, anger and depression measures. Blood pressure readings and baseline heart rate variability (HRV) measures were then obtained.

Blood pressure was obtained by taking consecutive readings from the non-dominant arm while the participant was seated, utilizing the Reli On Model HEM-780REL automatic blood pressure monitor. After the participant has rested for five minutes, two blood pressure readings were obtained at two-minute intervals. If the difference between the two readings was equal to or less than 5 mm Hg, then the mean of the two readings was considered as the measurement for that session (baseline or post treatment). If the difference between the two readings was greater than 5 mm Hg, a third reading was taken and all three readings averaged. The average served as the blood pressure measurement for that session. This measurement protocol is adopted from the work of Garcia-Vera, Labrador, & Sanz (1999).

A measure of HRV was obtained via a photoplethysmographic optical sensor (PPG) placed at the tip of the index finger of the non-dominant hand. The sensor is designed to be comfortable and its use requires no invasive procedures. The non-dominant hand was used, as it was less likely to be moved during the training session than the dominant hand. HRV readings were then recorded using the Freeze -Framer® software (Quantum Intech, Inc., http://www.freezeframer.com .) The pulse screen of the software was utilized to ensure adequate contact was made between the fingertip and the fingertip sensor at the outset of each HRV recording session.

Following acquisition of baseline measures, participants assigned to the UT group received the usual treatment available to all clients at the treatment center during the next ten days of pain treatment. On the tenth day, the UT group again completed the anger, pain, and depression measures and blood pressure and HRV readings as previously described.

Participants assigned to the UT+HRC group also completed baseline measures as previously described. In addition to the usual treatment provided by the multidisciplinary pain treatment program, participants in this group received six training sessions. Each training session included five minutes of paced breathing training and five minutes of HRV biofeedback. This training occurred over a two-week period. Training sessions were typically provided on a Monday, Wednesday, Friday schedule. However, due to researcher illness, extreme weather conditions, and participant absence, alterations from this schedule were made as necessary and thus occasionally included delivery of an additional training session on a regularly scheduled training day or alternately on a Tuesday or Thursday in order to fulfill the six session training requirement within 10 treatment days.

Deep diaphragmatic breathing has been associated with decreased heart rate, decreased blood pressure, decreased gastrointestinal distress, and increased relaxation (Pepper & Holt, 1993). Thus, prior to recording HRV, participants were first guided through five minutes of paced breathing using an audio compact disc (CD) to enhance relaxation and improve focus during the HRV session. The CD utilized a progression of musical notes up and down a scale to cue inspiration and expiration in a rhythmic manner. Inspiration occurred as five notes progressed up the scale. A chime cued a

brief rest. Expiration then occurred as five notes progressed down the scale. A chime again cued a brief rest. The cycle continued in this manner for the duration of the music track. Rates of breathing on the CD ranged from 40 – 70 breaths per minute. Tracks from the CD were sampled for each participant and participants were asked to choose the pace that was most comfortable for them. This became the track used during the breathing training sessions. Each audio track lasted between 5.14-5.16 minutes.

Following the breathing exercise, each participant was then guided through a training script (see Appendix C) while BVP was recorded by the software package for five minutes. The script was developed from prior published work with the Freeze-Frame® (Institute of Heart Math, Boulder Creek, CA; Childre, 1998; McCraty et al., 2003; Tiller et al., 1996). Participants were instructed shift their attention away from worries or pain to the area around their heart. They were then instructed to then breathe deeply while sustaining the heart focus. Next, participants are encouraged to focus on a positive emotion such as love, appreciation, or care or re-experience a time when they were enjoying themselves. Participants were also encouraged to radiate out sincere thoughts of appreciation, love and caring to themselves and those persons that the participant cares about deeply. Both the breathing and shifting from negative to positive thoughts serve as distraction tasks, whereby attention is shifted from a stressor to a positive emotional state. Such activities are believed to facilitate a shift from the sympathetic activity associated with a stress response to a balance between the sympathetic and parasympathetic systems.

The client's information was displayed on the computer screen so that the client could receive feedback regarding the beat –to –beat changes in heart rate and their

coherence ratios for each session. For this study, participants were determined to have achieved coherence when the medium plus high coherence ratios were greater than the low frequency coherence ratio (M+H>L). This criterion reflects the recommendation of R. McCraty (personal communication, October 10, 2005) in making a quantifiable judgment of whether a participant has achieved a coherent pattern to their heart rhythms. Recall that the coherence ratios in the Freeze-Framer software are a reflection of the amount of time each participant was at low, medium, and high coherence during a given training session. Coherence in this software package based on the amount of time a participant achieved a smooth, sine wave-like pattern to their heart rhythms and achieved a narrow-band, high-amplitude peak at 0.1 hertz during each HRV training/recording session. Following completion of the sixth training session, the anger, pain, and depression measures and blood pressure readings were obtained.

CHAPTER 3

RESULTS

Descriptive Statistics

Participants

A total of 59 participants were recruited from the two multidisciplinary pain management programs. The majority of participants (*n* = 47) were recruited from High Point Rehabilitation Center (HPRC; 79.7%) and the remaining 12 participants (20.3%) were recruited from North Texas Pain Recovery (NTPR). Five participants were excluded from the study due to early discontinuation from their multi-disciplinary treatment programs. Of those removed from treatment, two females from HPRC were excluded due to uncontrolled high blood pressure. One male from HPRC and one male from NTPR were excluded due to non-compliance with program requirements. One participant from HPRC was discontinued at her request, as she verbalized the program was not a good fit for her current rehabilitation needs. Additionally, one participant at HPRC remained in the multidisciplinary treatment program, but was excluded from the research study due erratic attendance, which resulted in his absence on days when the researcher was present. This resulted in retention of 53 participants.

Chi square analysis for discrete data were attempted to determine if significant differences existed between those who completed the study and the six participants that terminated prematurely; however, all attempted pairings yielded fewer than five cases for the expected chi square cells and no significant scores were found. Chi square in this sample may have been unable to detect possible differences that may have existed. Independent samples *t*-tests for continuous data were computed to determine if there

were any significant differences on baseline measures for those who completed the study from those who do not. No significant differences were found for age, education, income, number of days since injury, diastolic blood pressure, BDI-II pre-test scores, STAXI pre-test scores, PPI pretest scores or PRI pretest scores. A significant difference (p = .009) was found for systolic blood pressure, with the excluded group having a higher average score (M= 141.58; SD = 17.22) at pretest compared to those who remained in the study (M = 125.04; SD = 11.43). This was expected, as two participants were excluded from the pain treatment program due to excessively high blood pressure (systolic blood pressure > 160 on baseline measures).

Demographic information for the 53 participants who completed the study can be found in Tables 1-3. Of the 42 participants were recruited from HPRC, 50 % were female. Thirty of the HPRC participants were Caucasian (71.4%). Other ethnicities included eight African Americans (19%), two Latino/as (4.8%) and two people from Africa (non-U.S. origin; 4.8%). HPRC participants ranged in age from 31-68 years, with the average being 50.69 (SD = 8.64). The average educational attainment was 12.86 years (SD = 2.37). The majority of participants were married (27; 64.3%). Three participants had never married (7.1%). Six participants indicated they were divorced (14.3%) and six reporting being widowed (14.3%). Annual household income varied widely among those recruited from HPRC, ranging from $0-200,000 with the median income being $24,000. There was a wide range in the length of time since onset of chronic pain (6.43 - 537.2 months) with the median number of months being 35.48. The majority of participants endorsed having either multi-site pain (28.6%) or chronic low back pain (45.2%), while others endorsed having neck (19%), shoulder/arm (4.8%) or

leg/knee/hip (2.4%) pain. A majority of patients (90.5%) reported taking medications to assist in pain reduction and 14 (33.3%) reported taking medications for high blood pressure. Eighteen participants (42.9%) endorsed smoking on a daily basis and 33 (78.6%) endorsed consuming one or more caffeinated beverages on a daily basis. Only three participants (7.1%) were involved in litigation during the course of their pain treatment program.

Eleven participants from NTPR completed the study (36.4% female). Seven were Caucasian (63.6%). Other ethnicities included three African Americans (27.3%) and one Latino (9.1%). NTPR participants ranged in age from 26-61 years, with the average being 47.45 (*SD* = 10.22). The majority of participants were married (7; 63.6%). Three endorsed being never married (27.3%) and one was divorced (9.1%). Annual household incomes varied widely among those recruited at NTPR, ranging from $0 – 150,000.00, with the median income being $27,000. There was a wide range in the length of time since onset of chronic pain (6.13 –238.5 months) with the median number of months being 39.6. Five participants (45.5%) endorsed having low back pain. All participants (100%) reported taking medications to assist in pain reduction and three participants (27.3%) reported taking medications for high blood pressure. Seven participants (63.3%) endorsed smoking on a daily basis and eight (72.8%) endorsed consuming one or more caffeinated beverages on a daily basis. Two participants (18.2%) were involved in litigation during the course of their pain treatment program.

Chi square analyses for categorical variables were computed to check for treatment group differences on baseline measures. No significant differences emerged (see Table 1.) One-way analyses of variance (ANOVA) were computed for continuous

variables. Significant differences between HRPC and NTPR were found on both posttest measures of pain as well as diastolic blood pressure (see Table 2-3). With regard to PRI, HPRC participants had significantly lower average pain scores at posttest (M = 29.33, SD =13.39) than NTRP participants (M = 46.09, SD = 14.93), suggesting participants at the two sites differentially responded to treatment. Additionally, HPRC participants had significantly higher diastolic blood pressure at posttest (M = 82.15, SD = 9.14) than NTPR participants (M = 74.73, SD = 10.13). Thus, the pooling of participants was deemed inappropriate. Further, due to the small number of participants at NTRP (n = 11), multivariate analyses on this site alone would have resulted in inappropriately low cell sizes. Thus, hypothesis testing preceded utilizing participants from HPRC only (n = 42).

The frequency counts were obtained to determine the number of participants who achieved coherence at pretest and posttest for each group [usual treatment group (UT); coherence training group (UT+HRC)] and may be reviewed in Table 4. Correlation analysis were computed to determine if significant relationships existed among the continuous variables of age, average number of cigarettes smoked per day, average number of caffeinated drinks per day, systolic blood pressure at baseline, diastolic blood pressure at baseline and coherence at baseline. Only systolic blood pressure and diastolic blood pressure were significantly correlated (r = .84, p < .01; see Table 5). Additionally, 14 participants endorsed taking medication for high blood pressure. In order to determine if blood pressure medication status (no medication/medication) were influencing coherence scores at pretest, point biserial correlations were computed; results were not significant (r = .19, p = .23). Blood pressure medication status was not

significantly associated with baseline PRI scores (r = -.03, p = .84) or baseline PPI scores (r = .29, p = .06).

Measures

Descriptive data on the dependent variables (BDI-II, STAXI, PRI, PPI) are shown in Tables 6-9). Data were checked for outliers as well as the assumptions of normality, linearity, homoscedasticity, and multicollinearity. For each continuous variable, the presence of univariate outliers was examined by z scores for each cell. Univariate analysis revealed one outlier for the STAXI posttest score (z = 3.97) and one outlier on the BDI-II difference score (z = 3.32). No multivariate outliers were found when examining data using Mahalanobis distance value χ^2 (4, 42) = 18.47, p = .001. As no cases were multivariate outliers, all cases were retained for analysis.

The STAXI scores were skewed (z = 5.47). Traditionally, such values are transformed prior to analysis. However, transforming this variable and forcing it to be normally disturbed would have resulted in a loss of information valuable to Hypothesis 2, which proposed that as pain decreased, so to would anger and depression. On the contrary, the negative skew of this variable implies that anger scores for some participants increased from baseline to post-test.

Previous research studies found a relationship between heart rate variability (HRV) parameters and depression. Thus, in order to determine if depression was related to coherence values in this study, Pearson product moment correlation coefficients were computed. No significant relationship was found between BDI-II

scores at pretest and coherence scores at pretest or posttest or between BDI-II scores at posttest and coherence scores at posttest.

As McCraty, Atkinson, and Tiller (1995) point out, most individuals will not have a balance between the sympathetic and parasympathetic systems as measured by HRV. However, 15 participants meet this study's predetermined coherence criterion (M+H > L) at baseline. Prior to hypothesis testing, a 2 (Time: PRI pretest/PRI posttest) x 2 (Coherence: coherent at baseline/not coherent at baseline) mixed-model ANOVA was executed to determine if coherence at baseline was related to PRI scores. The results of the ANOVA (see Table 10) revealed that the between subjects main effect for coherence was not significant F (1, 40) = .004, p = .95, partial η^2 < .00. There was no difference in the PRI scores between those not coherent at baseline (M = 32.07) compared to those who were coherent at baseline (M = 35.00). There was a significant main effect for the within subjects variable of Time, F (1, 40) = 6.66, p = .01, partial η^2= .14. PRI pain scores after ten days enrolled in a pain treatment program (M = 28.84) were significantly lower than before treatment (M = 33.54). There was not a significant Time x Coherence interaction F (1, 40) = 3.05, p = .09, partial η^2= .07.

In order to clarify any potential influence of pre-treatment coherence on pain scores, mean differences were examined. This exploration indicated those not coherent at baseline presented with lower PRI scores at baseline (M = 32.07) than those who met the coherence criterion at baseline (M = 35.00) but the difference was not significant F (1, 40) = .44, p = .51). Those coherent at baseline exhibited a decrease in PRI scores from baseline (M = 35.00) to posttest (M = 27.13) and the change was significant F (1, 40) = 7.28, p = .01). However, those who were not coherent at baseline did not exhibit

the same level of decline from baseline (*M* = 32.07) to posttest (*M* = 30.56) and their difference on the PRI score over time was not significant *F* (1, 40) = .49, *p* = .49. Despite the significant decline in PRI scores from pre- to posttest for those who were coherent, the differences between the two groups (coherent/not coherent) at posttest was not significant *F* (1,40) = .62, *p* = .43). Those not coherent at posttest had an average PRI score of 30.56 and those who were coherent had an average PRI posttest score of 27.13.

The previously described ANOVA procedure was repeated for the PPI measure (see Table 11). The 2 (Time: PPI pretest/PPI posttest) x 2 (Coherence: Coherent at baseline/not coherent at baseline) mixed-model ANOVA revealed that the main effect for coherence was not significant *F* (1, 40) = .001, *p* = .99, partial η^2= .00. No significant difference was found between the PPI scores of those not coherent at baseline (*M* = 2.78) compared to those who were coherent at baseline (*M* = 3.07). There was a significant main effect for Time, *F* (1, 40) = 5.69, *p* = .02, partial η^2= .13. PPI pain scores after participants completed ten days of a pain treatment program were lower (*M* = 2.55) than before treatment (*M* = 2.92). The Time x Coherence interaction for PPI scores was not significant, *F* (1, 40) = 3.64, *p* = .06, η^2= .08.

As with the previous analysis, an exploration of the cell mean differences followed. Examination of the cell means indicated that for those not coherent at pretest, PPI scores were relatively stable from baseline (*M* = 2.78) to posttest (*M* = 2.70), with no significant differences being found *F* (1, 40) = .16, p = .69. For those coherent at baseline, PPI scores decreased from baseline (*M* = 3.07) to posttest (*M* = 2.40) and the difference was significant *F* (1, 40) = 7.17, *p* = .01. At baseline, average PPI scores

were slightly lower for noncoherent (*M* =2.78) than coherent (*M* =3.01) participants but the difference between the two groups was not significant *F* (1, 40) = .81, *p* = .37. At posttest, PPI scores were higher for those not coherent at baseline (*M* = 2.70) than those participants who were coherent at baseline (*M* = 2.40) but the difference was also not significant *F* (1, 40) = .91, *p* = .35).

Inferential Statistics

Hypothesis 1 proposed that those in the UT+HRC who achieved coherence at posttest would report decreased levels of pain compared to those who did not achieve coherence at posttest. As a first step in the analysis, participants were divided into groups based on treatment group (UT or UT+HRC) and coherence status at posttest. This resulted in the creation of two groups. HPRC Group A (*n* =15) consisted of those that completed UT+HRC and had coherence at posttest. HPRC Group B (*n* = 27) consisted of those that completed UT+HRC and were not coherent at posttest and those that were assigned to UT only.

In order to determine if age or length of time since injury should be used as a covariate, correlations were computed between age, length of time since injury, PRI difference scores and PPI difference scores. No significant relationships between age, length of time since injury and PRI or PPI difference scores were found (see Table 12). Thus, a multivariate analysis of variance (MANOVA) rather than a multivariate analysis of covariance (MANCOVA) was utilized to investigate differences in the two dependent variables of pain (PRI, PPI) and the independent variable of group assignment (A, B). The Box's Test revealed that equal variances could be assumed, *F* (3, 23581.63) = .43,

p = .73, indicating that the observed covariance matrices of the dependent variables were equal across groups. Therefore, Wilks' Λ criterion was used as the test statistic. MANOVA results indicate that achieving M+H>L coherence at posttest did not significantly affect the PRI difference score or PPI difference score [Wilks' Λ = .89, F (2, 39) = .86, p = .43, partial η^2 = .04]. Univariate ANOVA were computed as post hoc analysis. ANOVA results (Table 13-14) indicate that neither PRI scores differed significantly by group assignment [F (1, 40) =.11, p = .74, partial η^2 = .003], nor did PPI scores [F (1, 40) = 1.14, p = .29, partial η^2 = .03.]

In order to further clarify the potential relationship between coherence status, treatment group and pain levels, the grouping of the participants was reconfigured. Participants were again divided into groups based on treatment group and coherence status at posttest, however, four groups were utilized. HPRC Group 1 (n = 15) consisted of those that completed UT+HRC and had coherence at posttest. HPRC Group 2 (n = 6) consisted of those that received UT+HRC but were not coherent at posttest. HPRC Group 3 (n = 7) contained those that received UT only, but were coherent at posttest while HPRC Group 4 (n = 14) contained those that received UT only and were not coherent at posttest. Another MANOVA was utilized to investigate differences in the two dependent variables of pain (PRI, PPI) based on group assignment (1, 2, 3, 4). The Box's Test revealed that equal variances could be assumed, F (9, 3129.55) = .45, p = .90, indicating that the observed covariance matrices of the dependent variables were equal across groups. Therefore, Wilks' Λ criterion was used as the test statistic. MANOVA results indicate no significant group differences occurred with respect to pain difference scores [Wilks' Λ = .83, F (6, 74) =1.18, p = .32, partial η^2 = .09]. Univariate

ANOVA (Table 15-16) was computed as a follow-up to the MANOVA. Results indicate that neither PRI difference scores differed significantly by group [F (3, 38) =.54, p = .07, partial η^2 = .08], nor did PPI difference scores [F (3, 38) =2.17, p = .11, partial η^2 = .15.] Post hoc results utilizing Tukey's HSD revealed no significant differences between Groups 1, 2, 3, or 4 on PRI or PPI difference scores.

In order to further examine the data, grouping of the participants was reconfigured for a third time. Participants were divided into groups based on coherence status at baseline and posttest. This resulted in the creation of four groups. HPRC Group I (n = 11) consisted of those that had coherence at baseline and posttest. HPRC Group II (n = 16) consisted of those that were not coherent at baseline or posttest. HPRC Group III (n = 4) contained those that were coherent at pretest but not coherent at posttest, while HPRC Group IV (n = 11) contained those that were not coherent at pretest but achieved coherence at posttest. A MANOVA was utilized to investigate differences in the two dependent measures of pain (PRI, PPI) based on coherence status grouping (I, II, III, IV). The Box's Test revealed that equal variances could be assumed, F (9, 373.43) = 1.43, p = 1.73, indicating that the observed covariance matrices of the dependent variables were equal across groups. Therefore, Wilks' Λ criterion was used as the test statistic. MANOVA results again indicated no significant group differences with respect to pain scores [Wilks' Λ = .81, F (6, 74) = .41, p = .22, partial η^2 = .10]. Univariate ANOVA (Table 15-16) was computed as follow up and the results indicate that neither PRI difference scores differ significantly by group [F (3, 38) = 2.04, p = .13, partial η^2 = .14], nor PPI difference scores [F (3, 38) =1.34, p = .27, partial η^2 = .10.] Post hoc results utilizing Tukey's HSD revealed no significant

differences between Groups I, II, III, or IV on the PRI or PPI difference scores. In the case of Hypothesis 1, the null hypothesis is not rejected.

Hypothesis 2 proposed that depression and state anger would decrease as pain intensity decreased. Implicit in the hypothesis is the assumption that BDI-II, STAXI, PRI, and PPI scores would decrease. Thus, as a first step in evaluating the data for this hypothesis, dependent samples *t*-tests (see Table 19) were computed for each outcome variable. Scores on the BDI-II were significantly different from pretest to posttest, with the average pretest score being 23.24 (*SD* = 11.42) and the average posttest score being 18.74 (*SD* = 12.01), *t* (41) = 4.02, *p* < .001. Scores on the PRI measure significantly decreased from pretest (*M* = 33.11, *SD* = 13.61) to posttest (*M* = 29.33, *SD* = 13.39), *t* (41) = 2.12, *p* = .04. Scores on the PPI approached significance (pretest *M* = 2.88, *SD* = .99; posttest *M* = 2.60, *SD* = .99; *t* (41) = 1.86, *p* = .07. State anger scores increased slightly from pretest (*M* = 13.79, *SD* = 4.49) to posttest (*M* = 14.48, *SD* = 6.18) but the change was not significant *t* (41) = -.77, *p* = .45.

Pearson product-moment correlation coefficients were then computed using the difference scores for the sample (*n* =42) and can be reviewed in Table 9. Changes in PRI from pretest to posttest were positively associated with changes in pain levels as measured by the PPI (*r* = .36, *p* = .01; one tailed) but not significantly correlated with changes in BDI-II scores (*r* = .05, *p* =.38), or changes in STAXI scores (*r* = -.03, *p* = .42). The pain level difference score as measured by the PPI was not significantly related to the change in depression scores (*r* = .16, *p* = .15) or the change in STAXI scores from pretest to posttest (*r* = .01, *p* = .47). A moderate relationship was found between the change in BDI-II scores and the change in STAXI scores (*r* = .43, *p* < .01;

one tailed). Therefore, Hypothesis 2 is only partially supported.

To test Hypothesis 3, separate standard multiple regression analyses were conducted to determine if the independent variables (difference in BDI-II scores, difference in STAXI scores, difference in PRI scores, and difference in PPI scores) predict changes in systolic and diastolic blood pressure. Due to the potential influence of blood pressure medication and coherence on blood pressure, these were also used a predictors in the model. Regression results indicate that the overall model did not significantly predict difference in systolic blood pressure from baseline to posttest, $R^{2 =}$ .057, R^2_{adj} = -.104, F (6, 35) = .36, p =.90. This model accounts for 5.7% of the variance in systolic blood pressure difference scores. A summary of the regression coefficients is presented in Table 20. The overall model also did not significantly predict difference in diastolic blood pressure from baseline to posttest, $R^{2 =}$ .072, R^2_{adj} = -.087, F (6, 35) = .45, p =.84. This model accounts for 7.2% of the variance in systolic blood pressure difference scores. A summary of the regression coefficients is presented in Table 21. Pearson product moment correlations between all continuous variables used in this model are presented in Table 22. In the case of Hypothesis 3, the null hypothesis is not rejected.

Additional Analyses

Although there was not significant relationship found between age and coherence at pretest, a significant relationship did emerge between age and coherence at posttest (r = -.42, p = .003, one tailed). In order to better understand the relationship between age and coherence, participants were divided into four groups: Age Group 1

consisted of participants 31-39 years of age (*n* = 3). Age Group 2 consisted of participants age 40-49 years of age (*n* = 15). Age Group 3 consisted of participants age 50-59 years of age (*n* = 16) and Age Group 4 consisted of participants age 60-68 years of age (*n* = 8). Age Group 1 had an average M+H coherence score of 55 (*SD* 16.47) at pretest and an average M+H coherence score of 90.33 (*SD* = 9.07) at posttest. Age Group 2 had an average M+H coherence score at pretest of 42.13 (*SD* = 27.07) at pretest and an average of 60.33 (*SD* = 35.81) at posttest. Age Group 3 had an average M+H coherence score of 33.94 (*SD* = 27.29) at pretest and an average coherence score of 35.19 (*SD* = 28.24) at posttest. Age Group 4 had an average M+H coherence score at pretest of 45.00 (*SD* = 28.14) at pretest and an average coherence score of 44.63 (*SD* = 39.92) at posttest. A mixed model ANOVA (Age Group x Time: Coherence at Pretest/Posttest) revealed that the between subjects main effect for Age Group was not significant F (3, 38) = 2.52, p = .07, partial η^2 = .17. There was no overall significant difference in the coherence scores by age group. There was not a significant main effect for the within subjects variable of time, F (3, 38) = 3.71, p = .06, partial η^2= .09. Coherence scores were not significantly different at posttest (M = 57.62, SD = 6.31) than at pretest (M = 44.02, SD = 5.47). There was not a significant age group X time interaction F (3, 38) = 1.23, p = .31, partial η^2= .09.

In looking for replications of the relationship between the variables used in Hypothesis 2, Pearson product-moment correlations coefficients were computed using the posttest scores rather than difference scores and can be reviewed in Table 8. Posttest PRI scores were significantly related to PPI posttest scores (r = .53, p > .001; one tailed) and BDI posttest scores (r = .29, p = .03; one tailed) but not STAXI posttest

scores (r = .21, p = .10; one tailed). Posttest PPI scores were significantly related to BDI posttest scores (r = .41, p = .003; one tailed) and STAXI posttest scores (r = .36, p = .01; one tailed). Additionally, BDI-II scores were significantly related to STAXI posttest scores (r = .67, p > .001; one tailed). In sum, when evaluating posttest scores, significant relationships were found among the two pain measures (PRI, PPI), between pain and depression (PRI, BDI-II) and pain, depression, and anger (PPI, BDI-II, STAXI).

Further exploratory analysis for Hypothesis 2 was conducted by running additional correlations on difference scores and posttest scores. These were computed for those not coherent at posttest and those coherent at posttest. For those not coherent at posttest, use of the difference scores only yielded significant correlations on the pain measures (r = .39, p = .05; one tailed) and may be reviewed in Table 23. However, when posttest scores were utilized (Table 24), there were significant relationships between the depression and anger measure (r = .50, p = .01; one tailed) and between depression and the PPI (r = .41, p < .04; one tailed). For those coherent at posttest, only the BDI-II difference scores were significantly correlated with the STAXI difference scores (r = .54, p = .004; one tailed) and these scores may be reviewed in Table 25. However, when posttest scores were used (Table 26), anger and depression were significantly correlated (r = .81, p < .000; one tailed), and the pain measures were correlated significantly with one another (r = .66, p < .000; one tailed). Moreover, depression posttest scores were significantly correlated with both the PRI posttest score (r = .43, p = .02; one tailed) and the PPI posttest score (r = .43, p = .02; one tailed). However, a relationship between all four independent variables (depression, anger, and both pain measures) was still not found.

Exploratory analyses regarding Hypothesis 3 involved first computing a repeated measures *t*-test for the 25 participants whose systolic blood pressure decreased from pre to posttest. The average pretest reading was 130.73 (*SD* = 13.66) and the average posttest reading was 119.79 (*SD* =9.54). The change from pre to posttest represented a significant decline, t (24) = 5.84, $p < .000$ in systolic blood pressure. Next, an ANOVA was used to determine if the difference from pre to posttest on systolic blood pressure differed for those who were in the UT and experienced a decline in their systolic blood pressure (n = 14) compared to those in the UT+HRC group who experienced a decline in their systolic blood pressure (n = 11). No significant differences were found [F (1, 23) = .001, p = .97]. Next, correlation analyses (n = 25) were computed and revealed that systolic blood pressure was related to BDI-II scores at pretest only (r = .34, p = .05) but was unrelated to other measures (STAXI, PRI, PPI) and coherence at pre or posttest (see Tables 27-28).

The same series of analyses were completed to investigate diastolic blood pressure changes. For those whose diastolic blood pressure decreased from pre to posttest (n = 25), the average pretest reading was 88.15 (*SD* = 9.77) and the average posttest reading was 79.84 (*SD* = 8.08). The change from pre to posttest represented a significant decline from pre to posttest t (24) = 8.86, $p < .000$ in diastolic blood pressure. Next, an ANOVA was used to determine if the difference from pre to posttest on diastolic blood pressure differed for those who were in the UT (n = 12) compared to those in the UT+HRC group (n = 13). No significant differences were found [F (1, 23) = .77, p = .39]. Next, correlation analyses (n = 25) were computed and revealed that for

these participants, diastolic blood pressure was not significantly related to BDI-II, STAXI, PRI, PPI or coherence scores at pre or posttest (Tables 29-30).

CHAPTER 4

DISCUSSION

Hypothesis 1

The goal of Hypothesis 1 was to investigate the utility of the Freeze- Frame® technique (Institute of Heart Math, Boulder Creek, CA) as a tool for teaching positive coping techniques and assisting in the alleviation of pain. In this particular study, blood volume pulse (BVP) was measured via photoplethysmographic optical sensor (PPG) and the information was displayed on a computer screen. The coherence ratio calculated by the Freeze-Framer® software (Quantum Intech, Inc., http://www.freezeframer.com) was used as a way of quantifying participant coherence, with M+H>L being used as the coherence criterion in this study. It was anticipated that those who participated in the UT+HRC training and achieved coherence would report decreased levels of pain after ten days of multidisciplinary pain treatment more so than those who did not receive this training. The results of this study failed to reject the null hypotheses. That is, there were no significant relationships found between coherence status or treatment group and pain levels as measured by the PRI or PPI at posttest. It is possible that the lack of power in this study increased the likelihood of a Type II error.

Another issue to consider is the study design. The heart rate variability (HRV) training was administered six times over a 10-day period of multidisciplinary pain treatment programming. Each Freeze- Framer session lasted for five minutes and occurred after five minutes of paced breathing. The number of minutes of training was selected based on prior work of McCraty et al. (1995), as they indicated five minutes is often the maximal amount of time people are able to maintain a positive thought focus

at the outset of training. The training schedule was chosen based on what was agreeable to the data collection sites, as time engaged in this treatment protocol would result in taking time away from other programmatic pain treatment activities that were already approved by worker's compensation or private insurance. It is possible, however, that a more intensive treatment, such as daily training, prolonged training sessions, or longer time between pretest and post-test for participants to practice the technique would yield improved outcome results, as prior work by Barrios-Choplin, McCraty, & Cryer, 1997; McCraty Atkinson, Lipsenthal, & Arguelles (2003) incorporated more intensive treatment at the outset of training and longer times between pre and posttest. Since the proposal of this study, an additional study completed by Thurber (2006) supported the Freeze-Framer as a tool for decreasing musical performance anxiety and improving HRV. Although his study does not utilize participants with chronic pain, some important methodological differences between the Thurber study and this one deserve discussion. In Thurber's study, a more intensive treatment was used, in that participants completed four to five training sessions, lasting 30-50 minutes each and participants were provided with equipment to practice the technique at home. An additional difference between Thurber's study and this one was the utilization of a more stringent coherence criterion of 80% in combined medium and high coherence ratios rather than the 51% in combined in medium and high coherence used in this study. Additionally, for his study, the 80% was accumulated over a seven-minute period, as opposed to a five-minute period used in this study.

Due to the limited research utilizing the Freeze-Framer software, perhaps a different way of determining the physiological effects of training would also yield

valuable insight regarding the potential efficacy of thought shifting/appreciation techniques on the alleviation of pain. For example, in their early studies, McCraty, Atkinson, and Tiller (1995) focused on the use of the ECG as a measurement device and evaluated the sine wave pattern and/or focus on the presence/absence of a dominant peak at approximately 0.1 HZ. Additionally, they also utilized the ratio between the LF and HF activity as measured by ECG. In another study, total LF+MF+HF as measured by ECG was also used (McCraty, Atkinson, Tiller, Rein, & Watkins, 1995). Other studies investigating the utility of the thought shifting techniques as part of a stress management program also utilized ECG frequency analysis rather than coherence ratios found in the Freeze-Framer software (e.g. McCraty, Tomasino, Atkinson, Sundram, 1999; McCraty, Atkinson, Lipsenthal, & Arguelles, 2003).

Hypothesis 2

Support for the link between depression and pain has been cited in literature (Fishbain, Cutler, Rosomoff & Rosomoff, 1997; Gaskin, Greene, Robinson, & Geisser, 1992), with depression scores being associated with severity of pain. Additionally, anger has been associated with pain, both in terms of state anger (Gaskin, Greene, Robinson, & Geisser, 1992) and trait anger (Conant, 1998). Moreover, Okifuji, Turk, and Curran (1999) found that pain was significantly related to depression and the intensity of anger in a sample of persons with chronic pain. The goal of Hypothesis 2 was to glean further information regarding the relationship between pain, depression, and state anger, particularly following 10 days of chronic pain treatment.

At baseline, the PRI and PPI measures were significantly correlated with one another, but not with the BDI-II or STAXI scores. This does not replicate the finding of Okifuji, Turk and Curran (1999). Different outcome measures were used in this study as compared to the aforementioned study, which may account for the different findings. At post-test, PRI and PPI scores were positively and significantly correlated with one another. Additionally, the PRI was positively and significantly correlated with BDI-II scores, but not STAXI scores. The PPI posttest scores were positively and significantly correlated with both BDI-II and STAXI posttest scores. The BDI-II and STAXI were also positively and significantly correlated with one another. However, when difference scores (baseline minus posttest score) were used as a reflection of the amount of change from baseline to posttest, neither the PRI or PPI difference scores were significantly correlated with the BDI-II difference score or the STAXI difference score. The PRI and PPI difference scores remained significantly correlated with one another and the BDI-II and STAXI difference scores were significantly correlated with one another. In sum, when difference scores were used for the analysis, the change from pre to posttest did not demonstrate the same strength of relationships among the variables. Although significant relationships among difference scores were found between the two pain measures and between the depression and anger measure, significant relationships among all four dependent variables were not found. Thus, Hypothesis 2 was only partially supported.

The lack of significant relationships among the outcome measures could be related to the measures used, which were different than those used by Okifuji, Turk, and Curran (1999). Additionally, it could be that some other unmeasured factor is acting as a

mediator or moderator between physical sensations and psychological distress. Moreover, through participation in the pain treatment program, perhaps participants were able to distinguish between physiological discomfort and emotional states.

Hypothesis 3

Several studies that incorporated use of the Freeze-Frame as part of a stress reduction program demonstrated significant declines in blood pressure (Barrios-Choplin, McCraty & Cryer, 1997; McCraty, Atkinson, Lipsenthal, & Arguelles, 2003; McCraty, Atkinson, and Tomasino, 2003) as well as declines in anger and hostility, increased positive outlook (McCraty, Atkinson, Lipsenthal, & Arguelles, 2003), and decreased depression scores (Luskin, Reitz, Newell, Quinn, & Haskell, 2002). An inherent difficulty with such outcome studies is that it is difficult to determine if any one component of the program is primarily contributing to outcome results. Because multimodal pain management programs incorporate stress management training and relaxation techniques among their activities, it was hoped that the incorporation of HRV biofeedback into such programs would yield results similar to those found following the multimodal treatment offered by McCraty and colleagues. Specifically for this study, it was proposed that decreased pain, depression, and state anger would be associated with decreased blood pressure for those who had blood pressure greater than 120/80 mm Hg at baseline. Multiple regression equations utilizing difference scores were utilized in an attempt to determine if the outcome measures could predict systolic and diastolic blood pressure. None of the outcome measures were found to be significant, as PRI, PPI, BDI-II, STAXI, and Coherence difference scores entered into standard

regression models only predicted 5.7% of the variance in systolic blood pressure and 7.2% if the variance in diastolic blood pressure. Correlation analyses on difference scores were not significant among the majority of measures. Rather, depression and anger were positively related to each other, the two pain measures were positively related to one another, and systolic and diastolic blood pressure were related with one another. This result is not entirely surprising, given that the relationship between anger, depression, and pain was not fully supported by investigation of Hypothesis 2. In examining information from those who experienced a decline in systolic or diastolic blood pressure during the 10 days of pain treatment, significant differences were not found between those in the UT and the UT+HRC groups. It is possible that more intensive treatment and/or treatment delivered over a longer period of time may be needed in order to see significant changes in blood pressure.

In the McCraty, Atkinson, and Tomasino (2003) study, participants initially completed the stress management program in one full and two half days, participants had access to HRV biofeedback equipment at work and on weekends. Additionally, the posttest follow-up occurred three months after the training seminar. Because posttest measures occurred after only two weeks in the present study, it is possible that this is not sufficient time for HRV training to produce significant changes in blood pressure as that seen by McCraty's group. Additionally, average systolic blood pressure in the 2003 study was higher at baseline (130.4) than for participants in this study (125.78). Of note, HRV outcome measures were not incorporated as part of the aforementioned McCraty study.

Future Directions

The contributions of HRV training for persons with chronic pain needs further investigation. The limitations of this study provide suggestions for future research. For example, providing a more intensive treatment in the beginning of the treatment program such as that provided by the IHM researchers may produce different results than found here. Additionally, providing longer treatment sessions over a shorter time span, coupled with home training equipment, as in the research by Thurber (2006), may yield improved results.

Although not proposed for this study, exploratory examination of cell means following the mixed model ANOVA results revealed a pattern of results that warrant further study. It was unexpected that a number of participants would meet the coherence criterion at baseline. Although these participants had higher pain scores at the outset of the study, they also experienced the most substantial declines in pain scores from pre to posttest when compared to those who were not coherent at baseline. This leads one to suggest that those who are coherent at baseline may benefit the most from this particular type of biofeedback training for pain reduction. Perhaps HVR training prior to entry into a chronic pain program, or HRV training delivered early on in a 30-day pain treatment program could also assist with pain reduction at the 30-day completion date. However, further research will need to be conducted in this area in order to determine if this hypothesis gains support.

Other issues that arose during this study will hopefully provide insight into potential issues for future research. For example, there were significant declines from pretest to posttest on both measures of pain and the measure of depression. However,

there was no significant change in pre-to posttest on the measure of state anger. Although average scores on the state anger were mildly elevated at baseline, they did not decline over the two-week period. With regard to the anger measures, participants were administered the posttest on their tenth treatment day. It is on this day that many participants were informed about extensions to their treatment program. Some participants reported being angry when denied an extension of treatment while others reported being angry at receiving an extension, as this would result in additional time away from home and family. Such news clearly had the potential to influence outcome measure scores. Thus, perhaps a better method of collecting outcome data on anger would have been to administer measures on a daily basis and aggregate this data over time. However, given the length of time given to work with participants for this study, administering the dependent measures on each treatment day was not feasible for this particular study.

Additionally, some participants in this study were staying in a hotel while completing the treatment program, as their homes were geographically too far away from the treatment center to commute daily. Those that stayed in the hotel frequently complained of difficulty sleeping due to uncomfortable surroundings or noises, and on one instance, a participant complained of having his room flooded and having to move in the middle of the night. Such experiences are stressful and may have resulted in less change in scores from pre to posttest than was expected. Moreover, the BDI-II includes questions regarding changes in sleep and appetite, as well as other physical symptoms, which may be influenced by changes in physical activity level and disruptions in routine, such as beginning a new treatment program or staying in uncomfortable surroundings.

Two recent studies regarding HRV and pain have been published since the inception of this study and provide prompting for further investigation. Appelhans and Luecken (2008) completed a study that utilized ECG recordings of HRV among healthy adults presented with an acute pain stressor of cold temperature. The researchers found that higher LF was predictive of persons reporting less pain unpleasantness and having higher thresholds for pain. In another study, Hassett et al. (2007) investigated the utility of HRV biofeedback in persons with fibromyalgia in improving pain, depression, and sleep. Rather than using a thought/emotion focusing technique like the Freeze Frame, their study utilized 10 weekly sessions of resonant frequency breathing, with each session lasting 20 minutes and a follow-up three months after the training. Although there was no statistically significant improvement in pain at the 10-week point, most participants did report significant reductions in pain at the three-month follow-up. Ratings of depression symptoms declined significantly during the intervention and remained significantly lower at three-month follow-up than at baseline. Initial improvements in sleep were made in the first 10-weeks, but these gains were not present at follow-up. While neither of these two studies focused on persons with chronic back/neck pain, the results do provide encouragement for further research to determine if HRV may be of assistance in the treatment of pain through decreasing the perception of pain, or increasing pain tolerance, or by decreasing feelings of depression that often accompany chronic pain.

Table 1

Counts and Percentages of Categorical Demographic Information by Treatment Location

	HPRC *n* = 42		NTPR *n* = 11		Test for distributional differences	
	n	%	*n*	%	X^2	df
Gender					.65	1
Male	21	50	7	63.6		
Female	21	50	4	36.4		
Ethnicity					1.17	3
Caucasian	30	71.4	7	63.6		
African American	8	19.0	23	27.3		
Latino/a	2	4.8	1	9.1		
African	2	4.8	0	0		
Marital Status					4.87	3
Single	3	7.1	3	27.3		
Married	27	64.3	7	63.6		
Divorced / Separated	6	14.3	1	9.1		
Widowed	6	14.3	0	0		
Employment Status					.83	1
Employed	3	7.1	0	0		
Not Employed	39	92.9	11	100		
Litigation Status					1.69	2
Never litigated	37	88.1	9	81.8		
Litigation Ongoing	3	7.1	2	18.2		
Litigation Concluded	2	4.8	0	0		
Pain Location					1.84	4
Low back (Lumbar/Sacral)	19	45.2	5	45.5		
Mid Back (Thoracic)	0	0	0	0		
Neck (Cervical)	8	19.0	1	9.1		
Shoulder / Arm	2	4.8	1	9.1		
Leg /Knee / Hip	1	2.4	1	9.1		
Multi-site	12	28.6	3	27.3		
Pain Medications	38	90.5	11	100	1.13	1
Blood Pressure Medication	14	33.3	3	27.3	.15	1
Antidepressant / Anxiolytic	24	57.1	4	36.4	1.50	1
Daily Nicotine Use	18	42.9	7	63.6	.17	1
Daily Caffeine Use	33	78.6	8	72.8	1.50	1

* $p < .05$, ** $p < .01$

Table 2

One Way ANOVA Mean Differences for Demographics and Paper and Pencil Measures: Information by Site

	HPRC *n* = 42		NTPR *n* = 11		*F*
	M	*SD*	*M*	*SD*	
Demographics					
Age	50.69	8.64	47.45	10.22	1.13
Education	12.86	2.37	12.59	2.18	.13
Income	35764.48	35013.43	38161.45	39709.65	.04
Days since initial injury	2085.31	2667.25	2191.09	2264.620	.02
Paper and Pencil Measures					
Beck Depression Inventory II Pretest	23.24	11.42	26.36	16.40	.54
Beck Depression Inventory II Posttest	18.74	12.01	24.00	14.46	1.54
Beck Depression Inventory II Difference score	4.50	7.26	2.36	6.89	.77
State Anger Pretest	13.79	4.49	13.64	5.54	.01
State Anger Posttest	14.48	6.18	17.55	10.60	1.56
State Anger Difference Score	-.69	5.82	-3.91	8.99	2.10
McGill Pain PRI Pretest	33.12	13.62	38.09	13.12	1.17
McGill Pain PRI Posttest	29.33	13.39	46.09	14.93	13.04**
McGill Pain PRI Difference Score	3.79	11.57	-8.00	13.35	8.49*
McGill Pain PPI Pretest	2.88	.99	3.45	.82	3.10
McGill Pain PPI Posttest	2.60	.99	3.82	1.08	12.85**
McGill Pain PPI Difference Score	.29	.99	-.36	.92	3.82

Table 3

One Way ANOVA Mean Differences for Physiological Measures: Information by Site

	HPRC *n* = 42		NTPR *n* = 11		*F*
	M	*SD*	*M*	*M*	*SD*
Physiological Measures					
Systolic Blood Pressure Pretest	125.78	14.35	122.21	12.45	.57
Systolic Blood Pressure Posttest	122.59	11.41	118.76	16.43	.81
Systolic Blood Pressure Difference score	3.19	12.89	118.76	16.43	.004
Diastolic Blood Pressure Pretest	84.60	9.89	79.06	8.50	2.88
Diastolic Blood Pressure Posttest	82.15	9.14	74.73	10.13	5.50*
Diastolic Blood Pressure Difference score	2.44	8.43	4.33	9.80	.41
Coherence Scores pretest	40.48	28.14	30.73	25.83	.30
Coherence Scores posttest	49.90	35.43	49.00	27.12	.94

* $p < .05$, ** $p < .01$

Table 4

Frequency Counts of Participants for Each Group (UT, UT+HRC) Who Were Coherent at Pretest and Posttest

	Pretest	Posttest
UT Not Coherent	15	14
UT Coherent	6	7
UT+HRC Not Coherent	12	6
UT+HRC Coherent	9	15

Table 5

Correlations Between Selected Demographic Variables and Baseline Coherence (n = *42*).

Scale	1	2	3	4	5	6
Years of Age	1	-.15	-.05	.02	-.12	-.16
Number of Cigarettes Smoked Daily		1	.16	-.18	-.04	.09
Number of Caffeinated Beverages Consumed Daily			1	-.07	-.01	-.07
Systolic Blood Pressure				1	.84**	.05
Diastolic Blood Pressure					1	.04
Baseline Coherence Score						1

***p* < .01

Table 6

Scale Properties

Scale	*M*	*SD*	Range
Beck Depression Inventory-II			
Pretest	23.89	12.50	1-60
HPRC	23.24	11.42	1-40
NTPR	26.36	16.40	3-60
Posttest	19.83	12.59	0-57
HPRC	18.74	12.01	0-57
NTPR	24.00	14.46	4-49
STAXI State Anger			
Pretest	13.75	4.67	10-25
HPRC	13.79	4.49	10-23
NTPR	13.64	5.54	10-25
Posttest	15.11	7.30	10-39
HPRC	14.48	6.18	10-39
NTPR	17.55	10.60	10-39
McGill Pain Questionnaire Pain Rating Index (PRI)			
Pretest	34.15	13.54	0-57
HPRC	33.12	13.62	0-56
NTPR	38.09	13.12	14-57
Posttest	32.81	15.21	4-65
HPRC	29.33	13.39	4-60
NTPR	46.09	14.93	12-65
McGill Pain Questionnaire Pain Present Index (PPI)			
Pretest	3.00	.98	0-5
HPRC	2.88	.99	0-5
NTPR	3.45	.82	2-5
Posttest	2.85	1.12	1-5
HPRC	2.60	.99	1-5
NTPR	3.82	1.08	2-5

Table 7

*Zero Order Correlations of Continuous Variables at Pre-test (*n = *42)*

Scale	1.	2	3	4
1. BDI-II	1	.21	.30	.25
2. STAXI		1	.15	.12
3. PRI			1	.57**
4: PPI				1

***p* < .01 two tailed

Table 8

*Zero Order Correlations of Continuous Variables at Post-test (*n = *42)*

Scale	1.	2	3	4
1. BDI-II	1	.67**	.29*	.41**
2. STAXI		1	.21	.36*
3. PRI			1	.53**
4: PPI				1

**p* < .05 two tailed, ***p* < .01 two tailed

Table 9

*Correlations of Difference Scores for Outcome Measures (*n = *42)*

Scale	1.	2	3	4
1. BDI-II	1	.43**	.05	.17
2. STAXI		1	-.03	.01
3. PRI			1	.36*
4: PPI				1

* *p* < .05 one tailed, ***p* <.01 one tailed

Table 10

Mixed-model 2 (Coherence at Pre-test/Coherence at Posttest) x 2 (Time: PRI Score at Pre-test/PRI Score at Posttest) ANOVA

Source	SS	*df*	*MS*	*F*	*p*	partial η^2
Between Subjects	75049.37	1	75049.38			
Coherence	1.19	1	1.19	.004	.95	.00
Error	12202.02	40	305.05			
Within Subjects						
Time	424.68	1	424.68	6.66	.01	.14
Time X Coherence	194.30	1	194.30	3.05	.09	.07
Error	2550.24	40	63.76			

Table 11

Mixed-model 2 (Coherence at Pre-test/Coherence at Posttest) x 2 (Time: PPI Score at Pre-test/PPI Score at Posttest) ANOVA

Source	SS	*df*	*MS*	*F*	*p*	partial η^2
Between Subjects	577.91	1	577.91			
Coherence	.001	1	.001	.001	.98	.00
Error	60.24	40	1.51			
Within Subjects						
Time	2.64	1	2.64	5.69	.02	.13
Time X Coherence	1.69	1	1.69	3.64	.06	.08
Error	18.59	40	.47			

Table 12

*Correlations Between Age, Length of Time Since Injury, PRI Difference Scores and PPI Difference Scores (*n = *42).*

Scale	1	2	3	4
Age in years	1	.30*	-.002	-.04
Number of days since injury		1	-.02	-.26
PRI Difference Score			1	.36**
PPI Difference Score				1

* $p < .05$ one tailed, ** $p < .01$ one tailed

Table 13

*Groups A and B ANOVA: Coherence at Posttest on PRI and PPI Difference Scores (*n = *42).*

Source	SS	*df*	*MS*	*F*	*p*	partial η^2
Between Treatments						
PRI Difference Score	15.47	1	15.47	.11	.74	.003
Error	5473.60	40	136.84			
Total	6091.00	42				
PPI Difference Score	1.12	1	1.12	1.14	.29	.03
Error	39.45	40	.99			
Total	44.00	40				

Table 14

Groups A and B: Means and Standard Deviations for PRI and PPI Difference Scores by Coherence at Posttest

Score	PRI Difference Score		PPI Difference	
	M	SD	M	SD
Not Coherent At Posttest	3.33	10.81	.41	.97
Coherent at Posttest	4.6	13.18	.07	1.0

Table 15

*Groups 1-4 ANOVA: Coherence at Posttest on PRI and PPI Difference Scores (*n = *42)*

Source	SS	*df*	*MS*	*F*	*p*	partial η^2
Between Treatments						
PRI Difference Score	223.33	3	74.44	.54	.66	.04
Error	5265.74	38	138.57			
Total	6091.00	42				
PPI Difference Score	5.95	3	1.98	2.18	.11	.15
Error	34.64	38	.91			
Total	44.00	42				

Table 16

Groups 1-4: Means and Standard Deviations PRI and PPI Difference Scores by Coherence at Posttest

	PRI Difference Score		PPI Difference Score	
	M	SD	M	SD
Group 1	4.60	13.18	.07	1.03
Group 2	-.50	10.95	-.33	1.03
Group 3	7.43	9.41	.86	1.07
Group 4	2.93	11.44	.50	.76

Table 17

*Groups I-IV ANOVA: Coherence at Posttest on PRI and PPI Difference Scores (*n = *42)*

Source	SS	*df*	*MS*	*F*	*p*	partial η^2
Between Treatments						
PRI Difference Score	760.56	3	253.52	2.04	.13	.14
Error	4728.51	38	124.44			
Total	6091.00	42				
PPI Difference Score	3.89	3	1.30	1.34	.28	.10
Error	36.68	38	.97			
Total	44.00	42				

Table 18

Groups I-IV: Means and Standard Deviations PRI and PPI Difference Scores by Coherence at Posttest

	PRI Difference Score		PPI Difference Score	
	M	SD	M	SD
Group I	10.33	13.69	.67	1.07
Group II	1.94	8.50	.19	.83
Group III	-2.00	23.43	.67	1.53
Group IV	9.09	6.98	-.09	.94

Table 19

*Dependent Samples t-Test (*n = *42)*

Source	M	SD	*df*	*t*	*p*
BDI-II					
Paired Difference	4.50	7.26	41	4.02	<.001**
Pretest	23.24`	11.42			
Posttest	18.74	12.01			
STAXI					
Paired Difference	-.69	5.82	41	-.77	.45
Pretest	13.79	4.49			
Posttest	14.48	6.18			
PRI					
Paired Difference	3.79	11.57	41	2.12	.04*
Pretest	33.12	13.62			
Posttest	29.33	13.39			
PPI					
Paired Difference	.286	.99	41	1.87	.07
Pretest	2.88	.99			
Posttest	2.60	.99			

* $p < .05$, ** $p < .001$; one tailed

Table 20

*Coefficients for Model Variables Difference Scores and Systolic Blood Pressure (*n = *42).*

	B	β	*t*	*p*	Bivariate *r*	Partial *r*
Meds	-1.874	-.069	-.373	.711	-.044	-.063
BDI-II-diff	-.036	-.020	-.105	.917	-.087	-.018
STAXI- diff	-.320	-.144	-.743	.463	-.132	-.125
PRI- diff	.137	.123	.680	.501	.159	.114
PPI-diff	.614	.047	.257	.799	.098	.043
Coh-diff	.042	.120	.620	.539	.077	.104

Table 21

*Coefficients for Model Variables: Difference Scores and Diastolic Blood Pressure (*n = *42).*

	B	β	*t*	*p*	Bivariate *r*	Partial *r*
Meds	-2.813	-.159	-.864	.393	-.129	-.145
BDI-II-diff	-.071	-.061	-.317	.753	-.157	-.054
STAXI-diff	-.249	-.172	-.893	.378	-.179	-.149
PRI-diff	.032	.043	.242	.810	.089	.041
PPI-diff	.366	.043	.236	.814	.046	.040
Coh-diff	.029	.127	.661	.513	.042	.111

Table 22

*Correlation Coefficients for Continuous Variable Difference Scores used as Predictors (*n = *42).*

Scale	1	2	3	4	5	6	7
BDI-II difference score	1	.43**	.05	.16	-.08	-.09	-.16
STAXI difference score		1	-.03	.01	.24	-.13	-.18
PRI difference score			1	.36**	.06	.16	.09
PPI Difference Score				1	.18	.10	.05
Coherence Difference Score					1	.08	.04
Systolic Blood Pressure						1	.64**
Diastolic Blood Pressure							1

**p* < .01

Table 23

Correlations of Difference Scores on Outcome Measures for Those Not Coherent at Posttest

Scale	1.	2	3	4
1. BDI-II difference score	1	.37	-.05	.14
2. STAXI difference score		1	.11	.06
3. PRI difference score			1	.39*
4: PPI difference score				1

* *p* < .05 one tailed

Table 24

Correlations of Posttest Scores on Outcome Measures for Those Not Coherent at Posttest

Scale	1.	2	3	4
1. BDI-II difference score	1	.50*	.14	.41*
2. STAXI difference score		1	-.09	.27
3. PRI difference score			1	.34
4: PPI difference score				1

* *p* < .05 one tailed

Table 25

Correlations of Difference Scores on Outcome Measures for Those who were Coherent at Posttest

Scale	1	2	3	4
1. BDI-II difference score	1	.54**	.08	.18
2. STAXI difference score		1	-.05	.004
3. PRI difference score			1	.34
4: PPI difference score				1

* $p < .01$ one tailed

Table 26

Correlations of Posttest Scores on Outcome Measures for Those who were Coherent at Posttest

Scale	1	2	3	4
1. BDI-II difference score	1	.81**	.43*	.43*
2. STAXI difference score		1	.34	.37*
3. PRI difference score			1	.66**
4: PPI difference score				1

* $p < .05$ one tailed, ** $p < .01$ one tailed

Table 27

Correlations among Dependent Variable Pretest Scores for Those Whose Systolic Blood Pressure Declined from Pre to Posttest

	1	2	3	4	5	6	7
1. Systolic Blood Pressure	1	.78**	.34*	.17	.19	.20	.14
2. Diastolic Blood Pressure		1	.23	.11	.02	.08	.08
3. BDI-II			1	-.02	.40	.34	-.08
4. STAXI				1	-.03	.01	.05
5. PRI					1	.63**	.003
6. PPI						1	.24
7. Coherence							

* $p < .05$ one tailed, ** $p < .01$ one tailed

Table 28

Correlations among Dependent Variable Posttest Scores for Those Whose Systolic Blood Pressure Declined from Pre to Posttest

	1	2	3	4	5	6	7
1. Systolic Blood Pressure	1	.59**	.09	-.03	.19	.10	.04
2. Diastolic Blood Pressure		1	.20	-.12	.31	.11	-.05
3. BDI-II			1	.72**	.47**	.54**	.05
4. STAXI				1	.26	.46*	.29
5. PRI					1	.51**	.15
6. PPI						1	.24
7. Coherence							

* $p < .05$ one tailed, *$p < .01$ one tailed

Table 29

Correlations among Dependent Variable Pretest Scores for Those Whose Diastolic Blood Pressure Declined from Pre to Posttest

	1	2	3	4	5	6	7
1. Systolic Blood Pressure	1	.85**	.29	.15	.17	.31	.002
2. Diastolic Blood Pressure		1	.23	.23	-.04	.23	-.03
3. BDI-II			1	.28	.53**	.46*	-.03
4. STAXI				1	.26	.34*	-.01
5. PRI					1	.76**	.10
6. PPI						1	.32
7. Coherence							

* $p < .05$ one tailed, ** $p < .01$ one tailed

Table 30

Correlations among Dependent Variable Posttest Scores for Those Whose Diastolic Blood Pressure Declined from Pre to Posttest

	1	2	3	4	5	6	7
1. Systolic Blood Pressure	1	.61**	.15	-.02	.17	.20	.15
2. Diastolic Blood Pressure		1	.27	.09	.31	.22	.09
3. BDI-II			1	.71**	.53**	.47*	.12
4. STAXI				1	.27	.38*	.38*
5. PRI					1	.71**	.24
6. PPI						1	.42*
7. Coherence							

* $p < .05$ one tailed, ** $p < .01$ one tailed

APPENDIX A

INFORMED CONSENT

University of North Texas Institutional Review Board

Informed Consent Form

Before agreeing to participate in this research study, it is important that you read and understand the following explanation of the purpose and benefits of the study and how it will be conducted.

Title of Study: Heart Rhythm Variability in Persons with Chronic Pain

Principal Investigator: LaDonna Saxon, a graduate student in the University of North Texas (UNT) Department of Psychology.

Purpose of the Study:

You are being asked to participate in a research study, which is designed to determine the usefulness of the Freeze Framer training program to decrease pain intensity and symptoms of anger and depression in persons experiencing chronic pain. Your pulse rate will be recorded to measure changes in heart rhythm during the training sessions. Additionally, your blood pressure will be monitored to determine if the Freeze Framer program is effective in decreasing blood pressure among those with hypertension.

Study Procedures:

You will be asked to complete a brief interview, a pain questionnaire, the State Trait Anger Expression Inventory, and the Beck Depression Inventory-Second Edition. Your blood pressure readings will then be taken. Next, your pulse rate will be obtained via a non-invasive sensor placed at the tip of the index finger of your non-dominant hand. Pulse rate will be recorded using the Freeze Framer software. Prior to recording, you will be asked to sit quietly during a 5 minute rest period and refrain from talking, falling asleep, or engaging in exaggerated body movements. It is anticipated that these baseline measures will take approximately 35 minutes to complete.

Then, you will either be assigned to receive the usual treatment (UT) offered at the clinic or the usual treatment plus Freeze Framer Training condition (UT+FF). Participants in the UT+FF group will then receive the usual treatment offered at the pain clinic plus six sessions of Freeze Framer training lasting 15-20 minutes each session.

At the end of a two-week period, you will once again be asked to complete the questionnaires listed above and have your blood pressure and pulse taken as previously described. It is anticipated that the final measures will take approximately 30 minutes to complete.

Foreseeable Risks:
The foreseeable risks of this study are minimal. The study utilizes procedures that have previously been used with persons with and without medical conditions. There is the potential that some items in the questionnaires regarding anger or depression may cause discomfort. Should you experience extreme distress, the staff psychologist will be consulted. Referral to a local hospital for evaluation will occur if needed.

Benefits to the Subjects or Others:
We expect the project to benefit you by teaching you an additional tool to cope with negative thoughts and chronic pain. Additionally, this study is expected to contribute to knowledge in the field of psychology. If effective, there is the potential that the Freeze Framer system will be integrated into the routine treatment protocol at pain treatment centers, which will benefit future clients seeking treatment for chronic pain. It is expected that the benefits of this study will far outweigh any potential risks to individual participants.

Procedures for Maintaining Confidentiality of Research Records:

Signed consent forms will be stored in a locked cabinet in the research office of the Principal Investigator's research supervisor. All other materials will be assigned a numbered code and will not contain any personally identifiable information, and will be stored in a separate locked cabinet in the research office of the Primary Investigator. All records will be maintained until the completion of the study. Also, the confidentiality of your individual information will be maintained in any publications or presentations regarding this study.

Questions about the Study

If you have any questions about the study, you may contact LaDonna Saxon at telephone number 940-565-2671 or Joseph Doster, Ph.D., UNT Department of Psychology, at telephone number 940-565-2671.

Review for the Protection of Participants:

This research study has been reviewed and approved by the UNT Institutional Review Board (IRB). The UNT IRB can be contacted at (940) 565-3940 with any questions regarding the rights of research subjects.

Research Participants' Rights:

Your signature below indicates that you have read or have had read to you all of the above and that you confirm all of the following:

- LaDonna Saxon has explained the study to you and answered all of your questions. You have been told the possible benefits and the potential risks and/or discomforts of the study.
- You understand that you do not have to take part in this study, and your refusal to participate or your decision to withdraw will involve no penalty or loss of rights or benefits. The study personnel may choose to stop your participation at any time.
- You understand why the study is being conducted and how it will be performed.
- You understand your rights as a research participant and you voluntarily consent to participate in this study.
- You have been told you will receive a copy of this form.

_____
Printed Name of Participant

_____ _____
Signature of Participant Date

For the Principal Investigator or Designee:

I certify that I have reviewed the contents of this form with the participant signing above. I have explained the possible benefits and the potential risks and/or discomforts of the study. It is my opinion that the participant understood the explanation.

_____ _____
Signature of Principal Investigator or Designee Date

APPENDIX B

DEMOGRAPHICS QUESTIONNAIRE

Participant ID # _____
Treatment Condition _____

DEMOGRAPHIC INFORMATION / PARTICIPANT TRACKING FORM

(For researcher use only)

Age ____ Gender _____

Race / Ethnicity _____ Marital Status_____

Years of Education _____ Household annual income_____

Date of injury / pain onset _____ Work status _____

Litigation Status: never litigated litigation ongoing litigation concluded

Medical Diagnosis:_____

Current Medications:_____

Nicotine use: _____

Caffeine Use: _____

Participant did not complete study:

Date: _____

Reason: _____

Participant ID # _____
Treatment Condition _____

PARTICIPANT TRACKING FORM (SIDE 2)

Date of intake:_____ Date of post-treatment measures:_____

Blood pressure at intake: ____/____ (mean)

Reading 1:___/___ Reading 2:___/___ Reading 3:___/___ (if required)

Blood Pressure at post-treatment:_____/_____(mean)

Reading 1:___/___ Reading 2:___/___ Reading 3:___/___ (if required)

Scores at intake:

BDI-II:_____ STAXI:_____ PRI:_____ PPI:_____

Scores at post-treatment:

BDI-II:_____ STAXI:_____ PRI:_____ PPI:_____

Date of Baseline /training session:_____
Coherence Ratio:____(LF) ____(MF) ____(HF) - baseline
Coherence Ratio:____(LF) ____(MF) ____(HF) - training

Date of training session:_____
Coherence Ratio:____(LF) ____(MF) ____(HF)

Date of training session:_____
Coherence Ratio:____(LF) ____(MF) ____(HF)

Date of training session:_____
Coherence Ratio:____(LF) ____(MF) ____(HF)

Date of training session:_____
Coherence Ratio:____(LF) ____(MF) ____(HF)

Date of Posttest /training session:_____
Coherence Ratio:____(LF) ____(MF) ____(HF)

APPENDIX C

UT+HRC TRAINING SCRIPT

UT+HRC Training Script*

Shift your thoughts away from your worries or pain and allow your focus to on the area around your heart.

Breathe deeply into your heart center.

Begin to recall a time when you truly enjoyed yourself or experienced feelings of compassion, love or appreciation. Perhaps this is an enjoyable vacation, or a special moment spent with friends or family members, or even a special moment when you were alone, feeling peaceful and content. Recall this moment fully in your mind.

Picture the things you could you see around you. Recall and particular smells or sounds are associated with this time. For example, this could be the smell of freshly cut grass or the sound of someone's voice. Perhaps you may even recall physical sensations, such as the touch of someone's hand or the feel of the warm sun on your shoulders. Also re-experience the emotions that accompany this moment such as love, contentment, peace or joy.

If you have difficulty recalling a time of appreciation or enjoyment at least try to become neutral in your emotions, as this will help provide your body and mind a time-out from your worries.

Once you have taken this time out for yourself, gently send out feelings of appreciation to yourself and others. Picture each person who is important to you as you radiate sincere thoughts of appreciation, love or peace towards them. Be sure to also radiate wishes for contentment, peace and appreciation toward yourself.

* Content adapted from the works of Childre, 1998; McCraty, Atkinson, & Tomasino, 2003; Tiller, McCraty, & Atkinson, 1996

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