DEPRESSION, ANXIETY, AND SOCIAL SUPPORT FAIL TO PREDICT HEART RATE RECOVERY IN EXERCISE STRESS TEST PATIENTS

A thesis submitted to Kent State University in partial fulfillment of the requirements for the degree of Master of Arts

by

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Introduction

Cardiovascular disease has been the leading cause of death for both men and women in the United States since 1900, currently accounting for 34.3% of all deaths (American Heart Association, 2006; Lloyd-Jones, 2010). This translates to one out of every 2.7 deaths in the U.S., more than cancer, chronic lower respiratory disease, and accidents combined (American Heart Association, 2006). Approximately 2,300 Americans die every day as a result of cardiovascular disease making it responsible for one death every 38 seconds (Lloyd-Jones, 2010). In 2006, an estimated 81.1 million Americans were living with some form of cardiovascular disease and as of 2003, 37% of adults in the United States had two or more risk factors for cardiovascular disease including inactivity, obesity, and smoking (American Heart Association, 2006; National Center for Health Statistics, 2009). In addition, it is estimated that the economic impact of cardiovascular disease will have cost the U.S. more than $503.2 billion in 2010 alone due to medical treatment, medication, and lost wages. This is nearly double the cost created by cancer, which cost the U.S. approximately $228 billion in 2008, making cardiovascular disease one of the most significant health concerns for our country (Lloyd-Jones, 2010).

Cardiovascular disease and depression

Cardiovascular disease often leaves patients with high levels of emotional distress (Moser et al., 2010). However, the emotional response experienced is far greater than
imagined, with the prevalence of major depression in those who have suffered an acute myocardial infarction (MI) nearly three times that of the healthy population (Thombs et al., 2006). The *Diagnostic and Statistical Manual of Mental Disorders-IV-TR* estimates the prevalence of major depression to be 2-9% in the general population while rates for survivors of acute MI’s are thought to be 19.8% (4th ed., text revision, American Psychiatric Association, 2000; Thombs et al., 2006). In addition to this increased rate of depression following a cardiac event, it is important to note that depression is also related to the development of coronary heart disease (CHD). Specifically, depression is a strong risk factor for the development CHD with a relative risk of nearly 2.0 (Lett et al., 2004; Rugulies, 2002). This risk is even greater for those diagnosed with a major depressive disorder as opposed to those with milder depressive symptoms (Carney & Freedland, 2003).

In addition to being a risk factor for the development of CHD, depression is associated with much poorer prognoses for those patients already diagnosed with CHD (Carney & Freedland, 2003). Specifically, CHD patients diagnosed with comorbid clinical depression have a two times greater risk of mortality in the two years following diagnosis as compared to those without depression (Barth, Schumacher, & Herrmann-Lingen, 2004; Carney & Freedland, 2003). Similarly, depressed patients who recently suffered a MI have a two times increased risk of mortality in the 12 months following the initial MI (van Melle et al., 2004). In addition, depression continues to be a strong risk factor for mortality up to five years after the initial MI (Carney et al., 2008).
Increased mortality is not the only risk factor associated with depression in CHD patients. Those with CHD and depression were also found to have 31% more cardiac events such as heart failure, MI, stroke, or death than those non-depressed patients (Whooley et al., 2008). Additional research has found depression to be the strongest predictor of future cardiac events in those diagnosed with coronary artery disease (CAD; Carney et al., 1988) with depression also strongly predicting cardiac events in the first 12 months following an MI (Frasure-Smith, Lesperance, & Talajic 1995). In sum, cardiac patients experience increased rates of depression, which is associated with an increased risk for subsequent cardiac events and even death.

Cardiovascular disease and social support

Just as depression is a risk factor for both future cardiac events and mortality in those with CHD, a lack of social support has also been found to be a risk factor in those with cardiovascular disease (Murphy et al., 2008). For example, Berkman, Leo-Summers, & Horwitz discovered that recent MI sufferers who had no one to rely on for emotional support had two times greater risk of mortality than those with available emotional support. This increased risk of mortality was present in the hospital directly following the MI and remained present for at least one year post-MI (1992). A similar increased risk of mortality has been found to extend to those recent MI patients who are unmarried and live alone (Williams et al., 1992). A prognostic study by Kawachi et al. revealed that those men who lacked a social support network were found to have a twofold risk of dying from cardiovascular diseases when compared to their socially supported counterparts (1996). In addition to an increased risk of mortality, those recent MI patients...
who live alone experience twice as many cardiac events compared to those who live with others (15.8% vs. 8.8%, Case, Moss, Case, McDermott, & Eberly, 1992), indicating that, as with depression, social support is related to both increased morbidity and mortality.

**Cardiovascular disease and anxiety**

Anxiety, which is highly comorbid with and related to depression (Cameron, Abelson, & Young, 1991), has also been investigated as a potential risk factor for mortality or future cardiac events in those with cardiovascular disease. Specifically, in a sample of CAD patients, Frasure-Smith and Lesperance found that 20 to 25% of those meeting criteria for Major Depressive Disorder (MDD) or Generalized Anxiety Disorder (GAD) met criteria for both disorders. In addition, both the presence of depression and anxiety predicted an increased risk for future cardiac events (2008). In a separate study, Frasure-Smith, Lesperance, & Talajic showed that the risk of future cardiac events in MI patients associated with anxiety is as great as the risk associated with depression (1995). Additionally, the increased risk of cardiac events associated with anxiety extends to those patients undergoing coronary artery bypass surgery (CABG), with both pre and post-surgery anxiety levels being associated with an increased risk of cardiac events and mortality (Pignay-Demaria et al., 2003).

**Randomized clinical trials**

As described above, several psychosocial variables (i.e. depression, anxiety, and social support) are associated with worse outcomes for those with cardiovascular disease. Due to these important relationships, several randomized clinical trials have focused on
ways to treat depression in cardiovascular disease patients in hopes of reducing these relationships. One of these trials, the Sertaline Antidepressant Heart Attack Randomized Trial (SADHART) Group (Glassman et al., 2002), investigated the safety and efficacy of Sertaline, a selective serotonin reuptake inhibitor (SSRI), in recent MI patients and those with unstable angina. Hoping to decrease depression levels, the SADHART trial randomly assigned 369 depressed MI patients to receive Sertaline or placebo for 24 weeks. Overall, Sertaline was found to be safe for MI patients and those with unstable angina, with no cardiac functioning differences (e.g. LVEF, blood pressure, heart rate, or serious cardiac events) found between the groups. While Sertaline was found to be safe for those patients with cardiovascular disease, it did not significantly reduce depression for those experiencing their first depressive episode; however depression was significantly reduced for those with reoccurring depression (Glassman et al., 2002).

Whereas the SADHART trial investigated the safety and efficacy of SSRI’s in MI and unstable angina patients, the “Enhancing Recovery in Coronary Heart Disease” (ENRICHD) randomized clinical trial investigated whether the treatment of depression and low social support would decrease mortality after a MI in depressed patients (Berkman et al., 2003). Overall, 2481 MI patients with depression, low social support, or both were randomly assigned within 28 days of infarction to receive cognitive-behavioral therapy (CBT) or care as usual for six months. Both the treatment and care as usual groups showed improvements in psychosocial variables (i.e. depression and social support) and the treatment group saw a significant, albeit modest improvement over the care as usual group (49% reduction in depression scores for treatment group versus 33%
reduction in depression scores for care as usual group). Whereas the treatment did reduce depression and increase social support, there were no group differences in cardiac outcomes such as cardiac mortality and events at a 29-month follow-up.

Finally, the Canadian Cardiac Randomized Evaluation of Antidepressant and Psychotherapy Efficacy (CREATE) trials (Lesperance et al., 2007) examined both the use of a SSRI (Citalopram) versus a placebo and interpersonal therapy (IPT) versus case management in CAD patients meeting DSM-IV criteria for major depressive disorder (MDD). After 12 weeks, Citalopram was superior to placebo at reducing depression, however no difference in the number of cardiac events was found between the groups. Interpersonal therapy was found to be slightly less effective than case management; however this finding was not statistically significant.

Overall, interventions targeting the treatment of depression and social support have not proven effective in decreasing mortality or cardiac events in cardiovascular disease patients with depression. This could be due to the lack of understanding of the relationship between psychosocial factors and cardiovascular disease, such as how they are physiologically connected and influence one another, ultimately resulting in increased cardiac events and mortality.

**Potential mechanisms**

Carney, Freedland, Miller, & Jaffe describe mechanisms potentially contributing to the relationship between depression and cardiovascular disease (2002). They categorized these potential explanations into those that are linked to both psychosocial factors and cardiovascular disease, but not causally and those mechanisms that imply a
casual relationship between the two. Possible non-casual mechanisms discussed include antidepressant cardiotoxicity, association with major cardiac risk factors, and severity of the cardiovascular disease. Possible causal mechanisms discussed include non-adherence to cardiac prevention and care, platelet reactivity, and dysregulation of the autonomic nervous system (ANS).

Antidepressant cardiotoxicity is one proposed non-causal mechanism. Specifically, tricyclic antidepressants have been shown to be relatively unsafe for use in CHD patients and are known to increase HR, slow cardiac conduction, and increase the risk of cardiac morbidity and mortality (Roose & Miyazaki, 2005). Whereas this could account for a portion of the relationship between depression and increased cardiac events and mortality, it is not likely to be a major factor due to the fact that depression is rarely diagnosed by physicians, and when it is SSRI’s have been shown to be a safe alternative to tricyclic antidepressants (Carney et al. 2002; Glassman et al., 2002).

Another possible mechanism proposed by Carney et al. is the association between depression and major cardiac risk factors (2002). Depressed patients are more likely to smoke (Husky, Mazure, Paliwal, & McKee, 2008), have diabetes (Golden, 2007), and lead sedentary lifestyles (Bonnet, et al., 2005). While these are all known risk factors for cardiovascular disease, the relationship between depression and cardiovascular morbidity and mortality still exists even after controlling for these factors.

A final non-causal mechanism proposed by Carney et al. is disease severity (2002). This intuitive explanation contends that more severe diseases are more depressing for patients and this could account for the existing relationship. However, despite the
logical appeal of this explanation, there does not appear to be a relationship between cardiovascular disease severity and level of depression (Carney et al., 2002). For example, even after controlling statistically for disease severity, depression was still the best predictor of subsequent cardiac events in a sample of coronary artery disease patients (Carney et al., 1988).

In addition to the above potential explanations, several possible causal mechanisms have been proposed to explain the relationship between depression and CHD. The first of these is adherence to medical treatment and cardiac prevention. Depressed patients are less likely to take their medications, such as aspirin, as prescribed, which can lead to an increase in cardiac events and potentially mortality (Gehi, Haas, Pipkin, & Whooley, 2005). In addition, depressed patients are less likely to complete cardiac rehabilitation (Casey, Hughes, Waechter, Josephson, & Rosneck, 2008) and follow doctor recommended dietary guidelines (Luyster, Hughes, & Gunstad, 2009). This remains a plausible explanation, and additional research is underway.

A second potential causal explanation is platelet reactivity. Specifically, healthy depressed patients have been found to have increased platelet activation and reactivity (Glassman & Shapiro, 1998; Musselman, Evans, & Nemeroff, 1998). Platelet aggregation in CHD patients is related to an increased risk for coronary thrombosis and cardiac events (Carney et al., 2002; Musselman, Evans, & Nemeroff, 1998). This is also a plausible explanation accounting for part of the relationship between depression and cardiovascular disease with further research being conducted. Additionally, SSRI’s have been found to reduce platelet aggregation in depressed patients with CHD (Serebruany et al., 2003),
making this an attractive avenue for further exploration. Finally, Carney et al. suggest altered cardiac autonomic tone as a possible causal mechanism (2002).

**ANS functioning and depression**

Specifically, the cardiovascular system is regulated by the autonomic nervous system which consists of the sympathetic nervous system and the parasympathetic nervous system (PNS; Mussleman, Evans, & Nemeroff, 1998). Additionally, chronic imbalance of these two systems, such as excessive sympathetic activation or decreased PNS activation, leads to increased cardiac workload and increases the likelihood of cardiac events and even death (Carney et al., 2002; Curtis & O’Keefe, 2002). Two ways commonly used to measure PNS functioning include resting heart rate (HR) and heart rate variability (HRV), the variation in beat-to-beat intervals (Olshansky, Sabbah, Hauptman, and Colucci, 2008). For example, using these measures Dekker et al. followed 900 healthy men and women with no indication of CHD and discovered that those with lower HRV and higher resting HR had a significantly increased risk of CHD and all-cause mortality (2000). Additionally, MI patients with low HRV have been found to have a 64% greater mortality rate than those with high HRV (Camm et al., 2004). Similar findings have linked lowered HRV to higher morbidity and mortality in CHD patients (Camm et al., 2004; Carpeggiani et al., 2005; La Rovere, Bigger, Marcus, Mortara, & Schwartz, 1998; Stein, Domitrovich, Huikuri, & Kleiger, 2005). For example, LaRovere et al. followed 1284 CHD patients for an average of 21 months. Those patients with decreased HRV had a 3.2 times greater risk of cardiac mortality than those with well-
preserved HRV (1998), indicating a clear link between HRV and cardiac morbidity and mortality.

In addition to a connection between HRV and cardiac outcomes in cardiac patients, HRV abnormalities have been found in healthy depressed populations as well. For example, in a sample of 415 healthy men and women Pizzi, Manzoli, Mancini & Costa found depression to be significantly related to reduced HRV (2008). Similarly, Udupa et al. compared HRV in 40 CHD-free individuals diagnosed with MDD with 40 healthy controls, matched for age and gender. As with previous investigations, depressed individuals were found to have significantly lower HRV when compared to the non-depressed controls (2007).

In CHD samples, Carney et al. discovered that patients with elevated depression symptoms had poorer cardiovascular functioning, including increased HR and exaggerated HR response, compared to CHD patients with no depression symptoms (1999). Similar results have been found in other investigations (Carney et al., 1995; Carney et al., 2001; Watkins & Grossman, 1999) further implicating altered PNS functioning, in the relationship between cardiovascular disease and depression. For example, Carney et al., through the use of 24-hour HRV monitoring, found that coronary artery disease patients with comorbid major or minor depression had significantly lower HRV than non-depressed coronary artery disease patients. This finding remained significant even after controlling for disease severity, age, and gender (1995). Watkins and Grossman found similar results using baroreflexive sensitivity as a measure of PNS functioning. Those CHD patients with increased depression levels had reduced
baroreflexive sensitivity compared to those CHD patients with low levels of depression (1999). These findings all indicate impaired ANS functioning, and specifically impaired PNS functioning, in those CHD patients with increased depression.

**ANS functioning and psychosocial variables**

As before, depression is not the only psychosocial variable to affect this relationship (Gorman & Sloan, 2000). Specifically, decreased HRV has been found in healthy men with phobic anxiety (Kawachi, Sparrow, Vokonas, & Weiss, 1995), healthy men and women with trait anxiety (Dishman et al., 2000; Watkins, Grossman, Krishnan, & Sherwood, 1998), and MI patients with anxiety disorders (Martens, Nyklicek, Szabo, & Kupper, 2008). In addition, in healthy adults, heart rate variability has been found to be inversely related to perceived emotional distress (Dishman et al., 2000), state and trait anxiety (Shinba et al., 2008), panic (Friedman and Thayer, 1998) and generalized anxiety disorder (Friedman and Thayer, 1998). For example, Thayer, Friedman, and Borkovec (1996) compared HRV in 34 healthy adults with generalized anxiety disorder to 32 non-anxious healthy adults during baseline, relaxation, and worry periods. They discovered that while HRV decreased for both groups during the worry period, those participants with generalized anxiety disorder had decreased heart rate variability, when compared with the controls, in all three periods. Additionally, worry’s impact on HRV has been found to remain for several hours after the worry period has passed (Pieper, Brosschot, Van der Leeden, and Thayer, 2010).

Far fewer investigations have looked into the effects of social support, however; Randall, Bhattacharyya, and Steptoe found unmarried CAD patients to have significantly
reduced HRV compared to those CAD patients who were married or living with a partner (2009). These findings indicate that other psychosocial variables, in addition to depression, should be explored as potentially contributing to the relationships described above. Overall, altered ANS functioning due to depression or other psychosocial variables could lead to the progression of CHD and eventually cardiac events (Carney et al., 2002; Musselman, Evans, & Nemeroff, 1998).

Heart rate recovery

Whereas heart rate variability is a common method used to measure parasympathetic nervous system functioning, heart rate recovery (HRR) after exercise is another simple measure of PNS functioning (Pierpont & Voth, 2004). As mentioned above, both the sympathetic and parasympathetic nervous systems are involved in regulating the cardiovascular system, including the rate at which the heart returns to resting HR levels after exercise (Mussleman, Evans, & Nemeroff, 1998). The return of HR to resting levels after exercise is primarily accomplished through the activation of the PNS making HR recovery a good marker of ANS functioning, and specifically PNS, functioning (Imai et al., 1994; Pierpont, Stolpman, and Gornick, 2000).

Delayed heart rate recovery has been shown to predict mortality and the presence of CAD (Lipinski, Vetrovec, and Froelicher, 2004). For example, using a treadmill exercise test to acquire heart rate recovery, Nishime, et al. found that diminished HRR predicted mortality in CHD patients (2000). Specifically, HRR data was acquired for 9,454 cardiac patients. Twenty percent of these patients had abnormal HRR following an exercise stress test. After an average follow-up time of 5.2 years, those with abnormal
HRR saw an 8% mortality rate compared to only a 2% mortality rate for those with normal HRR. Overall, HRR was a strong predictor of mortality with an odds ratio of 4.16, indicating that those with abnormal HRR have an increased probability of dying (Nishime, et al., 2000). Additionally, increased HRR has been linked to sudden cardiac death (Jouven et al., 2005) and cardiac mortality (Cole, Foody, Blackstone, and Lauer, 2000) even after controlling for disease severity (Vivekananthan, Blackstone, Pothier, and Lauer, 2005).

Although, heart rate recovery has been shown to be an effective way to measure PNS functioning (Pierpont & Voth, 2004), few investigations have used this method to examine the relationship between cardiovascular disease and depression. One investigation that did utilize this method is Hughes et al., 2006. Specifically, 260 CHD patients reported depression symptoms using the Beck Depression Inventory (BDI) prior to completing a treadmill exercise stress test. The stress test resulted in resting heart rate, maximum heart rate, and heart rate two minutes after the end of the test. Heart rate recovery was defined as the difference between maximum heart rate and heart rate two minutes after the test. Depression was found to significantly predict heart rate recovery in the CHD sample and exercise capacity was found to mediate this relationship. This was the first study to look at depression and HRR in a CHD sample.

In a second investigation Hughes, et al. again found depression to significantly predict HRR. Specifically, 188 CAD patients were administered the BDI prior to completing an exercise stress test. Upon cessation of the stress test, HRR was again calculated by taking the difference between maximum heart rate and heart rate two
minutes after completion. As before, BDI scores were negatively associated with HRR, indicating that as depression symptoms increase HRR is attenuated. Beck Depression Inventory scores accounted for 3.5% of the variance in HRR, even after controlling for age (2008).

Heart rate recovery appears to be a simple way to assess PNS functioning while examining the relationship between cardiovascular disease and depression however; few researchers have utilized this methodology. In addition, no investigations have explored the relationship between anxiety or low perceived social support and HRR in a CHD sample. The current study seeks to investigate possible relationships between depression, anxiety, and low perceived social support with heart rate recovery in a CHD sample. It is expected that all three psychosocial variables will be negatively correlated with heart rate recovery such that as depression, anxiety, and low perceived social support increase heart rate recovery will be reduced. Additionally, it is expected that all three variables will be significant predictors of heart rate recovery.

**Hypothesis 1.** Patients reporting higher depression levels will exhibit slower heart rate recovery.

**Hypothesis 2.** Patients reporting higher state and trait anxiety levels will exhibit slower heart rate recovery.

**Hypothesis 3.** Patients reporting lower perceived social support will exhibit slower heart rate recovery.
Method

Participants

Participants consisted of 144 patients (109 men and 35 women) completing cardiac stress tests at Summa Health Center’s Heart and Lung center in Akron, OH. In order to be included in the study patients had to be fluent in English and be undergoing an exercise stress test. Patients ranged in age from 29 to 88 and were primarily Caucasian (93%). Table 1 has further participant demographics. Patients were enrolled from July 7, 2007 to November 4, 2008. One hundred and forty four patients underwent a treadmill exercise stress test and 9 underwent an Adenoscan stress test. Those undergoing an Adenoscan stress test were administered adenosine and a radioactive tracer, resulting in images of their heart and blood flow. Only those patients who underwent a treadmill exercise stress test were used in analyses. Conditions prompting stress tests were predominantly suspected coronary artery disease (54%), followed by ischemia (18%). The remainder of indications consisted of investigation of the extent-severity of disease, cause of dyspnea, and re-evaluation of disease. Fifty-seven percent of participants reported taking a beta-blocking medication. Table 2 lists percent of participants taking other common medications.
Table 1

Clinical and demographic characteristics of study population.

*Values represent the mean ± 1 S.D.*

<table>
<thead>
<tr>
<th></th>
<th>Patients</th>
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<tbody>
<tr>
<td>N</td>
<td>144</td>
</tr>
<tr>
<td>Age</td>
<td>62±12</td>
</tr>
<tr>
<td>Gender (% male)</td>
<td>76%</td>
</tr>
<tr>
<td>Race (% Caucasian)</td>
<td>93%</td>
</tr>
<tr>
<td>Weight (pounds)</td>
<td>192±38</td>
</tr>
<tr>
<td>Body mass index</td>
<td>29±5</td>
</tr>
<tr>
<td>Education</td>
<td>14±3</td>
</tr>
<tr>
<td>Exercise capacity (METS)</td>
<td>10±3</td>
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</table>
Table 2

Percent using medications by type (N = 144)

<table>
<thead>
<tr>
<th>Medication type</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>79</td>
</tr>
<tr>
<td>Beta blockers</td>
<td>57</td>
</tr>
<tr>
<td>Alpha blockers</td>
<td>7</td>
</tr>
<tr>
<td>Diuretics</td>
<td>19</td>
</tr>
<tr>
<td>Statins</td>
<td>60</td>
</tr>
<tr>
<td>SSRI</td>
<td>6</td>
</tr>
<tr>
<td>Anti-anxieties</td>
<td>3</td>
</tr>
<tr>
<td>Anti-depressants</td>
<td>7</td>
</tr>
<tr>
<td>Anti-coagulants</td>
<td>37</td>
</tr>
<tr>
<td>Anti-arrhythmias</td>
<td>1</td>
</tr>
<tr>
<td>ACE-Inhibitors</td>
<td>38</td>
</tr>
<tr>
<td>ARBs</td>
<td>12</td>
</tr>
<tr>
<td>Calcium Channel Blockers</td>
<td>15</td>
</tr>
<tr>
<td>Nitrates</td>
<td>15</td>
</tr>
<tr>
<td>Hypertension, Other</td>
<td>6</td>
</tr>
<tr>
<td>Hyperlipidemia, Other</td>
<td>18</td>
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</tbody>
</table>
Procedure

The Beck Depression Inventory (BDI), Spielberger State-Trait Anxiety Inventory (STAI), and the ENRICHED Social Support Instrument (ESSI) were administered to patients prior to stress testing or during their scheduled break. Patients participated in a Bruce or modified Bruce protocol treadmill exercise stress test resulting in resting HR, maximum HR at termination of test, and HR recovery at 2 minutes after peak exercise. A chart review was conducted in order to obtain medical history, diagnoses, and current medications.

Measures

Depression. Depressive symptoms were measured using the Beck Depression Inventory (Beck et al., 1961). The BDI is a self-report questionnaire designed to assess current levels of depression. It consists of 21 questions, each with 4 response options that correspond to varying degrees of depressive symptoms. For example, response options for question three consist of zero (“I do not feel like a failure”), one (“I feel I have failed more than the average person”), two (“As I look back on my life, all I can see is a lot of failure”), and three (“I feel I am a complete failure as a person”). Patients chose the answer that best reflects their level of depressive symptoms in the past few days. The first thirteen questions of the BDI assess cognitive aspects of depression such as disappointment in oneself, feelings of sadness, and suicidal ideation. The last eight questions assess the physical aspects of depression such as fluctuations in weight, fatigue, and lack of interest in sex. Scores range from zero (no depressive symptoms) to 63 (severe depressive symptoms), with a score of 10 or higher generally used as a cutoff for
the possible presence of clinical depression. Using a cutoff of 10 to diagnose major depression in MI patients, Strik et al. found the BDI to have a sensitivity of 82% and a specificity of 79% (2001).

**Anxiety.** Anxiety symptoms were measured using the Spielberger State-Trait Anxiety Inventory (STAI). The STAI is a 40 item self-report questionnaire used to assess current (state) and stable (trait) anxiety symptoms. The STAI assess anxiety by having respondents rate how closely each statement reflects their level of anxiety. Response options range from one (“almost never”) to four (“almost always”). State anxiety is assessed through 20 statements that ask the patient to refer to their anxiety symptoms right now or at this moment and trait anxiety is assessed through 20 questions that ask the patient to refer to their anxiety symptoms generally. Scores range from 20 to 80 for both the state anxiety portion and the trait anxiety portion. A score of 39-40 or higher is generally considered to be clinical levels of anxiety (Addolorato et al., 2000). Overall, the STAI is found to be reliable, with internal consistency exceeding .80 and test-retest ranging from .73 to .86 (Spielberger, 1983).

**Social Support.** Social support was measured using the ENRICHD Social Support Inventory (ESSI) which was developed for use in the ENRICHD clinical trial (Barefoot et al., 2003). The ESSI is a 6-item self-report questionnaire designed to assess the availability of social support in the patient’s life. Each question asks the patient about the availability of someone in their life to provide social and emotional support. Questions are rated using five response choices that range from one (“none of the time”) to five (“all of the time”). Scores range between 6 and 30, with scores of 18 and lower
generally considered evidence of low perceived social support. A sample item would be “Is there someone available to you who shows you love and affection?”

The ESSI has been found to be psychometrically sound with high test-retest reliability and strong predictive and discriminate validity (Vaglio et al., 2004). Cronbach’s alpha for the ESSI is .86, putting the internal consistency in the acceptable range (Barefoot et al. 2003).

**Exercise stress testing.** A treadmill exercise test was used to obtain resting HR, maximum HR, and HR recovery data. Testing used a GE Marquette (Waukesha, WI) treadmill and followed either a Bruce or modified Bruce protocol. Specifically, during a Bruce protocol patients begin walking at a speed of 1.7 mph with a 10% gradient. Both speed and incline are increased every 3 minutes until the test is stopped or the final stage is reached with a speed of 7 mph and a gradient of 26%. Reasons for test termination included patients’ request, dyspnea, fatigue, dizziness, chest pain, pain other than chest pain, and changes in ECG or blood pressure. A modified Bruce protocol begins at a speed of 1.7 mph and a gradient of 0%. After three minutes the second stage begins and the incline is increased to 5% while the speed remains at 1.7 mph. The third stage is equivalent to the first stage of the Bruce protocol; 1.7 mph and a gradient of 10%. Once again, the test is continued until the final stage is reached (7 mph and 26% incline) or the test is terminated due to patient request, dyspnea, fatigue, dizziness, chest pain, pain other than chest pain, or changes in ECG or blood pressure. A standard 12-lead electrocardiogram (ECG) was recorded continuously from rest until 6 minutes after the termination of the test. The ECG was used to measure HR at rest prior to testing, every 2
minutes during testing, maximum HR upon termination of testing, and at minute 2, 4, and 6 after cessation of testing. Only resting HR, maximum HR, and HR two minutes after termination were used for this study. Heart rate recovery was determined by taking the difference between maximum HR at termination of the test and HR at two minutes after termination of the test.

**Analytic strategy**

Descriptive analyses including frequencies, means, and standard deviations were used to assess the sample characteristics. Next, four bivariate correlations were run to discover the relationships between heart rate recovery and BDI scores, STAI-State scores, STAI-Trait scores, and ESSI scores. Hierarchical multiple linear regressions were conducted next to further investigate the relationships between heart rate recovery and psychosocial variables. Specifically, heart rate recovery was predicted first from BDI scores, followed by STAI-State scores, STAI-Trait, and ESSI scores in a series of four regressions. These regressions controlled for age, sex, and β-blocker usage. These variables were chosen to control for because of their likely impact on heart rate and HRR. For example, it has been shown that maximum heart rate achieved declines with age while HRR increases (Kostis et al., 1982). In a similar vein, women have been shown to have a higher heart rate than men overall and older men are more likely to see changes in heart rate as they age (Stein, Kleiger, and Rottman, 1997). Finally, β-blockers, a medication commonly prescribed to CHD patients, are known to alter parasympathetic functioning in cardiac patients (Lampert, Ickovics, Viscoli, Horwitz, and Lee, 2003). See
Table 3 for results of bivariate correlations between heart rate recovery and control variables.

Table 3

Bivariate correlations between heart rate recovery and age, gender, and β-blocker usage (N = 144)

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Gender</th>
<th>β-blocker usage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Rate Recovery</td>
<td>-.354*</td>
<td>-.057</td>
<td>.236*</td>
</tr>
</tbody>
</table>

* p < .05
Results

Descriptive Statistics

In the current sample, 9% of individuals had scores above the 10 point cut-off, indicating the possible presence of clinically significant levels of depression. Further, Beck Depression Inventory scores ranged from 0 to 19, with a mean of 4.39 ± 3.70. When considering clinically significant levels of anxiety, 17% of individuals had state-anxiety scores over 40 points with 16% of individuals having trait-anxiety scores above this level. State anxiety scores ranged from 20 to 75, with a mean of 30.9 ± 10.70 and trait anxiety scores ranged from 23 to 60, with a mean of 34.92 ± 7.97. Eight percent of individuals reported social support scores lower than 18, the cut-off typically used to define low social support with scores ranging from 9 to 30, with a mean of 25.92 ± 4.89. See Table 4 for a further breakdown of questionnaire scores. Average HR recovery at two minutes was 46.01 ±
Table 4

Psychosocial characteristics of study population (N = 144)

Values represent the mean ± 1 S.D.

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI</td>
<td>total scale</td>
<td>4.4±3.7</td>
</tr>
<tr>
<td></td>
<td>cognitive-affective subscale</td>
<td>1.5±2.1</td>
</tr>
<tr>
<td></td>
<td>somatic subscale</td>
<td>2.9±2.1</td>
</tr>
<tr>
<td>STAI</td>
<td>state anxiety</td>
<td>30.1±10.7</td>
</tr>
<tr>
<td></td>
<td>trait anxiety</td>
<td>34.9±8.0</td>
</tr>
<tr>
<td>ESSI</td>
<td>total scale</td>
<td>25.9±4.9</td>
</tr>
</tbody>
</table>

Bivariate correlation analyses

Bivariate correlation results between heart rate recovery and psychosocial variables (BDI, STAI-State, STAI-Trait, and ESSI scores) are presented in Table 5 and 6. As shown, trait anxiety scores were positively related to heart rate recovery, indicating that those with higher levels of trait anxiety experience greater HRR (r = .23, p > .05). State anxiety scores were also positively related to heart rate recovery, indicating that those with higher levels of state anxiety also experience greater HRR (r = .23, p > .05). Neither depression scores (r = .06, p = .55) nor social support scores (r = -.16, p = .17) were significantly related to heart rate recovery.
Table 5

*Bivariate correlations between depression, social support, trait anxiety, and state anxiety (N = 144)*

<table>
<thead>
<tr>
<th></th>
<th>BDI</th>
<th>STAI-Trait</th>
<th>STAI-State</th>
<th>ESSI</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI</td>
<td>0.601*</td>
<td>0.557*</td>
<td>-0.465*</td>
<td></td>
</tr>
<tr>
<td>STAI-Trait</td>
<td>0.634*</td>
<td></td>
<td>-0.476*</td>
<td></td>
</tr>
<tr>
<td>STAI-State</td>
<td></td>
<td></td>
<td>-0.394*</td>
<td></td>
</tr>
<tr>
<td>ESSI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p < .01

Table 6

*Bivariate correlations between heart rate recovery and depression, anxiety, and social support (N = 144)*

<table>
<thead>
<tr>
<th>Heart Rate Recovery</th>
<th>BDI</th>
<th>STAI-Trait</th>
<th>STAI-State</th>
<th>ESSI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.059</td>
<td>0.229*</td>
<td>0.225*</td>
<td>-0.156</td>
</tr>
</tbody>
</table>

* p < .05
Linear regression analyses

Hierarchical multiple linear regressions were carried out to further investigate the relationships between depression, social support, state and trait anxiety and heart rate recovery. The first step of these analyses regressed age, sex, and β-blocker usage onto heart rate recovery.

When BDI scores were examined as a possible predictor of heart rate recovery the first step, consisting of age, sex, and β-blocker usage, accounted for 11% of the variance \([F(3, 101) = 5.29, p < .01]\). Age significantly predicted heart rate recovery \((\beta = -.36, p < .001)\). When BDI scores were added in step two they were not found to be a significant predictor of heart rate recovery \([\Delta R^2 = .00, F\text{-change (1, 100)} = .132, p = .717]\). See Table 7 for complete regression results.

Table 7

Hierarchical Multiple Linear Regression Analyses Predicting Heart Rate Recovery from Depression (N = 144)

<table>
<thead>
<tr>
<th></th>
<th>(R^2)</th>
<th>(\Delta R^2)</th>
<th>B</th>
<th>SE B</th>
<th>(\beta)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1</td>
<td>.14</td>
<td>.14</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td>-.41</td>
<td>.11</td>
<td></td>
<td>-.36*</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td>-3.16</td>
<td>2.91</td>
<td></td>
<td>-.10</td>
</tr>
<tr>
<td>β-blocker</td>
<td>1.29</td>
<td>2.51</td>
<td></td>
<td></td>
<td>.05</td>
</tr>
<tr>
<td>Step 2</td>
<td>.14</td>
<td>.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI</td>
<td></td>
<td>.12</td>
<td>.34</td>
<td></td>
<td>.03</td>
</tr>
</tbody>
</table>

% with elevated BDI score 9

*p < .01
Next, the relationship between social support and heart rate recovery was examined. The first step accounted for 10.9% of the variance \([F (3, 99) = 5.18, p < .01]\). Age was the only significant predictor of heart rate recovery \((\beta = -.36, p < .001)\). When ESSI scores were added in the second step less than .5% additional variance was predicted \([\Delta R^2 = .017, F\text{-change} (1, 98) = 1.96, p = .165]\) indicating social support did not significantly predict heart rate recovery. See Table 8 for complete regression results.

**Table 8**

*Hierarchical Multiple Linear Regression Analyses Predicting Heart Rate Recovery from Social Support (\(N = 144\))*

<table>
<thead>
<tr>
<th></th>
<th>(R^2)</th>
<th>(\Delta R^2)</th>
<th>(B)</th>
<th>(SE)  (B)</th>
<th>(\beta)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1</td>
<td>.14</td>
<td>.14</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td>-.41</td>
<td>.11</td>
<td>-.36*</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td>-3.16</td>
<td>2.94</td>
<td>-.10</td>
</tr>
<tr>
<td>(\beta)-blocker</td>
<td></td>
<td></td>
<td>1.29</td>
<td>2.53</td>
<td>.05</td>
</tr>
<tr>
<td>Step 2</td>
<td>.15</td>
<td>.02</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESSI</td>
<td></td>
<td></td>
<td>-.36</td>
<td>.26</td>
<td>-.13</td>
</tr>
</tbody>
</table>

% with elevated ESSI score = 8

*p < .01

When the relationship between STAI-Trait and heart rate recovery was examined, the first step accounted for 10.8% of the variance in heart rate recovery \([F (3, 92) = 4.81, p < .01]\). As with previous analyses, age was the only significant predictor of heart rate recovery \((\beta = -.36, p < .001)\). When STAI-Trait scores were added to the model during the second step less than .5% additional variance was accounted for \([\Delta R^2 = .022, F\text{-change} (1, 91) = 2.42, p = .123]\) indicating trait anxiety did not significantly predict heart rate recovery. See Table 9 for complete regression results.
Finally, when the relationship between STAI-State and heart rate recovery was further examined, the first step accounted for 10.9% of the variance in heart rate recovery \[F (3, 98) = 5.13, p < .01\]. Age was found to significantly predict heart rate recovery (\(\beta = -.36, p < .001\)). When STAI-State scores were added to the model during the second step less than .5% additional variance was accounted for \([\Delta R^2 = .023, F (1, 97) = .2.71, p = .103]\) indicating state anxiety did not significantly predict heart rate recovery. See Table 10 for complete regression results.

Table 9

_Hierarchical Multiple Linear Regression Analyses Predicting Heart Rate Recovery from Trait Anxiety (N = 144)_

<table>
<thead>
<tr>
<th></th>
<th>R²</th>
<th>ΔR²</th>
<th>B</th>
<th>SE B</th>
<th>B</th>
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<tbody>
<tr>
<td>Step 1</td>
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<td>.14</td>
<td>-.41</td>
<td>.11</td>
<td>-.36*</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td>-3.16</td>
<td>3.05</td>
<td>-.10</td>
</tr>
<tr>
<td>β-blocker</td>
<td></td>
<td></td>
<td>1.29</td>
<td>2.63</td>
<td>.05</td>
</tr>
<tr>
<td>Step 2</td>
<td>.16</td>
<td>.02</td>
<td>.26</td>
<td>.17</td>
<td>.16</td>
</tr>
<tr>
<td>STAI-Trait</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*p < .01
Table 10

*Hierarchical Multiple Linear Regression Analyses Predicting Heart Rate Recovery from State Anxiety (N = 144)*

<table>
<thead>
<tr>
<th>Step</th>
<th>R²</th>
<th>ΔR²</th>
<th>B</th>
<th>SE B</th>
<th>B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1</td>
<td>.14</td>
<td>.14</td>
<td>.41</td>
<td>.11</td>
<td>-.36*</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>-.316</td>
<td>2.95</td>
<td>-.10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>β-blocker</td>
<td>1.29</td>
<td>2.54</td>
<td>.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Step 2</td>
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<td>.02</td>
<td>.20</td>
<td>.12</td>
<td>.16</td>
</tr>
<tr>
<td>STAI-State</td>
<td></td>
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</tbody>
</table>

*p < .01
Discussion

Summary

The current investigation sought to replicate findings by Hughes et al. (2006; 2008) linking depressive symptoms to heart rate recovery using a heart rate recovery paradigm. Additionally, it also sought to link social support and anxiety to heart rate recovery. Unexpectedly, both state and trait anxiety were positively correlated with heart rate recovery, indicating that as anxiety increases heart rate recovery increases. Also unexpectedly, neither depression nor social support were related to heart rate recovery. Upon further investigation, the hypotheses that depression, social support, and anxiety would predict heart rate recovery were not supported. Results of the descriptive statistics indicate that only a small percentage of the current sample was experiencing elevated depression symptoms or low social support. Overall, the current investigation was unable to replicate previous findings linking depressive symptoms to heart rate recovery utilizing a heart rate recovery paradigm (Hughes et al., 2006; Hughes et al., 2008) and was unable to predict HRR from additional psychosocial variables.

Rates of Depression, Anxiety, and Social Support

The lack of relationship between heart rate recovery and depression, anxiety, and social support in the current investigation is likely due to the low levels of depression and high social support in the current sample. Rates of elevated state or trait anxiety were
slightly higher, but still lower than in previous investigations. Specifically, 9% of the current sample had elevated depressive symptoms with 8% of the current sample having ESSI scores below the cutoff of 18, indicating low levels of social support. Elevated state anxiety symptoms were present in 17% of the current sample and elevated trait anxiety symptoms were present in 16% of the current sample.

Depression rates in CHD samples overall have been estimated to be 20% or more using a diagnostic interview and 30% or more using the BDI (Thombs, et al., 2006). Investigations utilizing only patients enrolled in cardiac rehabilitation have found similar rates of depression, approximately 20% (Milani, Lavie, & Cassidy, 1996; Milani & Lavie, 2007). The current study sample contained only 9% experiencing elevated depressive symptoms.

As with depression, rates of increased anxiety in the current sample were considerably lower than anxiety rates in previous investigations utilizing similar populations. For example, 5% of patients met full criteria for generalized anxiety disorder and 41% of patients had elevated anxiety symptoms in Frasure-Smith and Lesperance’s 2008 investigation whereas in the current sample only 17% were experiencing elevated state anxiety symptoms and 16% were experiencing elevated trait anxiety symptoms.

Additionally, few patients in the current sample reported experiencing low social support. Prior cardiac samples have found rates of low social support to be around 38% (Berkman, Leo-Summers, & Horwitz, 1992) and 26% when using the ESSI, the same social support measure utilized in the current study (Berkman et al., 2003). In the current sample 8% endorsed enough items to indicate they were experiencing low social support,
again a much lower rate than reported in other cardiac samples. In summary, it is likely that the low level of psychological distress in the current sample is accounting for the lack of relationship between these variables and heart rate recovery.

Previous research utilizing the heart rate recovery paradigm supports this idea and indicates that even with higher rates of depression only a small percentage of the heart rate recovery variance is predicted (Hughes et al., 2006; Hughes et al., 2008). In 2006, utilizing a cardiac rehabilitation sample of 260 patients, Hughes et al. found 22% of their sample to have elevated levels of depression. With this level of depression, BDI scores were a significant predictor of heart rate recovery accounting for 3% of the variance. In 2008, Hughes et al. investigated similar relationships in 188 patients with CAD. In this sample, 18% of patients were experiencing elevated levels of depression and BDI scores accounted for 2.6% of the overall variance. Taken together these findings suggest that even with adequate levels of depression present, depression only accounts for a small portion of the variance. This makes it unlikely that significant amounts of the variance would be accounted for with levels of depression as low as those in the current investigation. Additionally, these findings indicate that very high levels of depression are necessary to predict substantial amounts of variance in heart rate recovery. Potential explanations for these low levels of psychological distress include the high levels of social support, low diversity, and the high level of engagement in the current sample.

**Potential Explanations**

Research has shown that social support in general promotes recovery in cardiac patients (Grace et al., 2002). Spouses, in particular, are often the most important source
of social support and those who are not married report far less social support in their lives overall (Brummett et al., 2001; Williams et al., 1992). Additionally, social support in the form of marriage has been shown to predict attendance to cardiac rehab with married individuals one and a half to two times more likely to attend (Beckie & Beckstead, 2010; Molloy, Hamer, Randall, & Chida, 2008). The high level of social support in the current sample is likely influenced by the large percentage of married participants, with 87% reporting having a spouse.

The low levels of depression and anxiety indicate that the current sample may be low in psychological distress overall. This may, in part, be due to the adequate level of social support acting as a buffer against further psychological distress (Grace et al., 2002; Koivula, Paunonen-Ilmonen, Tarkka, Tarkka, & Laippala, 2002; Olstad, Sexton, & Sogaard, 2001). Specifically, using a one-year predictive model, Holahan, Moos, Holahan, & Brennan found that increased social support predicted lower levels of depression in cardiac samples (1995). Similarly, cardiac patients who are unmarried or experiencing low social support report greater levels of depression (Frasure-Smith, Lesperance, Juneau, Talajic, & Bourassa, 1999) and more difficulty coping with their cardiac disease (Holahan, Holahan, Moos, & Brennan, 1997).

Additional support for the idea of social support as a buffer against the increased rates of psychological distress in cardiac patients comes from Frasure-Smith et al. (2000). Specifically, Frasure-Smith et al. found no direct link between social support and cardiac mortality in a sample of 887 MI patients, a relationship previously supported (Berkman, Leo-Summers, & Horwitz, 1992; Williams et al., 1992). An interaction, however, was
discovered between social support and depression such that patients with the lowest level of social support saw a stronger connection between depression and mortality, but those patients with the highest levels of social support had no connection between depression and morality (2000). This further indicates that a strong social support network may be able to act as a buffer against depression.

A separate possible explanation for the low rate of depression in the current sample is the lack of minorities and women. Specifically, the current sample consisted of 93% Caucasian patients and 76% male patients. Research has shown that both minority (Macabasco-O’Connell, Crawford, Stotts, Steward, & Froelicher, 2010; Mead, Andres, Katch, Siegel, & Regenstein, 2010) and female cardiovascular disease patients (Grace et al., 2002; Hunt-Shanks, Blanchard, & Reid, 2009) experience higher rates of depression and anxiety. It is likely that the lack of diversity in the current sample is contributing to the low rate of patients experiencing elevated depression and anxiety symptoms.

In addition, research has shown that actively engaging in the recovery or disease management process acts as a buffer against depression and anxiety (Wrosch, Schulz, Miller, Lupien, & Dunne, 2007). Specifically, the presence of a physical health problem predicted increased depression only in those individuals who reported low levels of engagement in the disease management process. Those who were actively involved in the recovery and management process did not experience an increase in psychological distress when faced with a physical illness (Wrosch et al., 2007). These findings indicate that the patients in the current investigation may be experiencing lower levels of
psychological distress partly due to taking an active role in managing their physical health by engaging in their doctor-recommended stress test.

Related to the idea of engagement in the disease management process is adherence to doctor recommended behaviors. Specifically, research has shown that patients experiencing elevated depression and anxiety or low social support are less likely to adhere to doctor’s recommendations including taking medications as prescribed (Bane, Hughes, & McElnay, 2006; Aggarwal & Mosca, 2010; Garner, 2010), participation in cardiac rehabilitation (McGrady, McGinnis, Badenhop, Bentle, & Rajput, 2009; Deskur-Smielecka et al., 2009; Yohannes, Yalfani, Doherty, & Bundy, 2007), and other risk-reducing behaviors (Kuhl, Fauerbach, Bush, & Ziegelstein, 2009). It would not be surprising if these findings extended to other doctor recommendations, such as the cardiovascular stress test recommended to the patients in the current sample. It is possible that those patients experiencing greater psychological distress and lower social support at the time of their doctor’s recommendation were less likely to follow through and make an appointment for a stress test, thus allowing them to have the opportunity to participate in the current investigation.

A final factor that may be impacting the relationship between psychosocial variables and heart rate recovery is the population used in the current investigation. As mentioned, the current investigation utilized patients receiving a diagnostic stress test, as recommended by their physician. This leaves the possibility that not all the patients had CHD. It could be that their doctor was suspecting the presence of CHD, but it was in fact
not there. Those individuals who may have ended up being CHD-free may also be free of altered PNS functioning seen in individuals with cardiovascular disease.

**Anxiety and Heart Rate Recovery**

The current investigation found a positive relationship between state and trait anxiety and heart rate recovery. This indicates that those with more anxiety experience better heart rate recovery, opposite than what was expected. It is unlikely that those patients with higher levels of anxiety are more physically fit and thus experiencing faster heart rate recovery. It is likely, however, that the current sample is experiencing increased stress and anxiety. The current sample contained patients who were receiving a stress test at the recommendation of their physician. It could be that these individuals were initially more prone to anxiety and worry, thus prompting them to see their physician. Additionally, it could be that these individuals were experiencing elevated concern and anxiety at the time of the investigation due the fact that their physician feels they may have cardiovascular disease. Finally, because these patients not only took the initiative to schedule an appointment with their physician, but also followed through with their recommendations; they may be more worry or anxiety-prone overall. Kuhl, Fauerbach, Bush, and Zeigelstein found that those patients with higher anxiety were more likely to carry potentially helpful medical supplies, such as nitroglycerine, possibly to help ease their anxiety (2009). Participants in the current investigation may have been experiencing elevated anxiety regarding the possible presence of cardiovascular disease and followed through with the recommended stress test to help alleviate their anxiety.
Limitations

The greatest limitation to the current study is the low levels of depression, anxiety, and low social support. In order to avoid this limitation, future investigations should screen for those patients currently experiencing higher levels of psychological distress to help ensure adequate sample sizes of both distressed and non-distressed patients. This would also enable researchers to make direct comparisons of functioning between the two groups.

A second limitation to the current study is the lack of diversity in the sample utilized. Specifically, 76% of the current sample was male and 93% of the current sample was Caucasian. The low level of diversity is potentially contributing to the low levels of psychological distress. Future investigations should consider oversampling for women and minorities to achieve a more diverse sample population.

A final limitation to the current study is the utilization of a convenience sample. This sample population consisted of those patients referred for an exercise stress test by their physicians to investigate the potential presence of coronary artery disease or to assess the severity of current disease. These individuals may not be representative of all CHD patients and it is possible that not all patients had cardiovascular disease.

Strengths

The current investigation is believed to be the first study to examine the link between anxiety and social support and heart rate recovery. While previous research has examined the link between depression and heart rate recovery (Hughes et al., 2006;
Hughes et al., 2008), this paradigm has not be utilized with other psychosocial variables, such as anxiety and social support. Additionally, altered PNS functioning and social support and anxiety have been investigated (Randall, Bhattacharyya, & Steptoe, 2009; Watkins et al., 1998), however; no investigation has examined this link utilizing the heart rate recovery paradigm.

An additional strength of the current investigation is its utilization of a diagnostic patient population. The majority of previous investigations have examined PNS functioning in cardiac rehabilitation samples (Hughes et al., 2006) or hospitalized patient samples (Carney et al., 2001). The current findings suggest that the relationship between psychosocial variables and PNS functioning may be specific to patient populations or to those further along in the disease process. Future investigations should further explore potential differences in these relationships in differing populations and whether certain populations are protected from the PNS-psychosocial variables link.

Conclusion

The current study investigated whether psychosocial variables such as depression, low social support, and anxiety, predict heart rate recovery in a diagnostic sample. Despite previous findings indicating a relationship between depression and heart rate recovery (Hughes et al., 2006; Hughes et al., 2008), none of the psychosocial variables predicted heart rate recovery in the current study. This is likely attributed to the low level of psychological distress in the current sample possibly due to high social support, low diversity, and high engagement. Additionally, the utilization of a diagnostic sample increased the likelihood that not all participants had CHD. Future investigations should
use patient populations and screen for participants with increased levels of anxiety and depression as well as low social support in addition to oversampling women and minorities.
References


Imai, K., Sato, H., Hori, M., Kusuoka, H., Ozaki, H., Yokoyama, H. et al. (1994). Vagally mediated heart-rate recovery after exercise is accelerated in athletes but


