University of Alberta

The Influence of Anxiety and Hostility on Heart Rate Recovery in the Cardiac Patient

by

Lea Alison Carlyle 

A thesis submitted to the Faculty of Graduate Studies and Research
in partial fulfillment of the requirements for the degree of Master of Arts

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled **The Influence of Anxiety and Hostility on Heart Rate Recovery in the Cardiac Patient** submitted by **Lea Alison Carlyle** in partial fulfillment of the requirements for the degree of **Master of Arts**.

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Abstract

This study sought to examine the association between anxiety, hostility and heart rate (HR) recovery. A total of 123 participants were recruited with 52 participants meeting eligibility criteria. State and trait anxiety were assessed using the State Trait Anxiety Inventory with cut-off values > 39 used to define high and low anxiety groups. The Hostile Attitude Scale was used to assess hostility with a cut-off value > 78 used to determine high and low hostility groups. An abnormal HR recovery was indicated by a HR that did not decrease by more than 12 beats within the first minute after the exercise stress test. Results of analysis of variance revealed that there were no differences between individuals reporting high and low state or trait anxiety or hostility and HR recovery values. Also, none of the groups demonstrated an abnormal HR recovery. It will be important to continue to assess the relationships among psychological and physiological characteristics as they both have an affect on health.

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The Influence of Anxiety and Hostility on Heart Rate Recovery in the Cardiac Patient

The role of the autonomic nervous system (ANS) in health and disease has been the focus of recent research (Rosenwinkel, Bloomfield, Arwady, & Goldsmith, 2001). The complex interactions between the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS) allows the body to adapt to changes in the environment and maintain internal balance by making short-and long-term physiological adjustments (Vander, Sherman, & Luciano, 1994, p. 226). An extensive literature exists demonstrating a strong association between chronic imbalance between the PNS and SNS and mortality (Lauer, 2003) mostly due to a decrease in parasympathetic activity. Conditions that affect autonomic balance include cardiac transplantation, myocardial infarction (MI), high blood pressure (BP), diabetes, normal aging (Curtis & O'Keefe, 2002), and psychological factors (Sloan et al., 1994; Stein et al., 2000; Watkins, Grossman, Krishnan, & Sherwood, 1998).

Psychological factors such as anxiety, hostility, depression, and social isolation have been associated with increased all-cause mortality, coronary heart disease (CHD) incidence, and mortality after an MI (Barefoot, Dahlstrom, & Williams, 1983; Barefoot & Schroll, 1996; Frasure-Smith, Lesperance, & Talajic, 1995). The mechanisms through which psychological factors exert their effects still remains unclear, but behavioural, neuroendocrine and pathophysiological mechanisms have been proposed (Horsten et al., 1999). Recent research has identified that there may be a pathological alteration in cardiac autonomic balance and that this may be the mechanism by which psychological variables increase the risk for mortality (Rozanski, Blumenthal, & Kaplan, 1999). This imbalance could involve either an increase in sympathetic activity, which has been linked

to onset of life-threatening cardiac arrhythmias and sudden death (Lown, 1982) or impaired parasympathetic control, which has been linked to increased cardiac mortality (Rich et al., 1988).

The majority of studies that have looked at the associations between psychological factors and increased risk of cardiac mortality have used 24-hour Holter recordings of heart rate variability (HRV) and baroreceptor sensitivity (BRS) to measure parasympathetic activity (Nissinen et al., 2003). More recently, researchers have evaluated the usefulness of heart rate (HR) recovery immediately after an exercise stress test (EST) in a number of populations including normal healthy, cardiac, diabetic, and elderly. The rate at which the HR decreases immediately after an EST and 1 minute into recovery is thought to be due mostly to parasympathetic reactivation and it has been found that a delayed fall in the HR during the first minute after an EST is associated with an increased risk of mortality (Nishime, Cole, Blackstone, Pashkow, & Lauer, 2000). To date, researchers have not examined the influence of psychological factors on cardiovascular outcomes using HR recovery as a measure of parasympathetic activity. Further research is needed to examine whether HR recovery would be a useful measure to use to capture the effects of psychological factors on health, and more specifically on autonomic balance. Heart rate recovery is simple to calculate as it is contained within the results of the EST and may prove to be beneficial in assessing the risk of patients in routine clinical practice (Cole, Blackstone, Pashkow, Snader, & Lauer, 1999).

The following review of the literature critically examines the associations between autonomic dysfunction and increased mortality, as measured by HRV and HR recovery, as well as the mechanisms by which anxiety and hostility may increase CHD

risk. This review will also establish the role that HR recovery has as a simple measure of autonomic function in relation to anxiety and hostility.

Statement of Problem

There is little data examining the relationship of psychological factors to autonomic function using HR recovery in cardiac patients. The majority of research has measured autonomic function using HRV or BRS analysis. Therefore, this study set out to examine the effects of anxiety and hostility on autonomic function assessed by measuring the change in HR one minute after an EST.

The first objective of this study was to examine the relationship between anxiety and HR recovery. Comparisons were made between individuals with high and low trait anxiety as well as between those with high and low state anxiety. The second objective of the study was to examine the relationship between hostility and HR recovery as well as to assess differences in individuals with high and low hostility scores. It was hypothesized that individuals with high levels of trait anxiety or hostility would have a poor HR recovery after the EST. This is based on previous research that has found that psychological factors such as anxiety and hostility have an affect on autonomic function as demonstrated by low HRV (Sloan et al., 1994; Watkins et al., 1998).

The third objective was to determine if there were differences in demographic or clinical characteristics between individuals with a normal HR recovery compared to those with an abnormal HR recovery. It was hypothesized that individuals displaying an abnormal HR recovery would have poorer clinical characteristics.

Delimitations

Delimitations and limitations of studying the influences of anxiety and hostility on HR recovery in the cardiac patient include:

1. heart rate recovery measures autonomic function
2. there are physiological mechanisms by which hostility and anxiety may affect cardiovascular health and outcomes
3. trait anxious cardiac patients will also experience state anxiety prior to the EST
4. since anxiety and hostility affect autonomic balance when measured by heart rate variability that similar results will occur when heart rate recovery is used to measure the effects of anxiety and hostility on autonomic balance
5. cardiac patients recruited from a single cardiac rehabilitation program which included mostly men.

Limitations

1. there are ways to measure anxiety and hostility and that the measures are reliable and valid
2. participants will answer questionnaires honestly.

Literature Review

Heart rate variability and mortality

Cyclic variation of the intervals between successive normal QRS complexes occurs due to the control of the sinoatrial node by the ANS and are commonly referred to as heart rate variability (Martin et al., 1987). Heart rate variability analysis provides a means of assessing the beat-to-beat changes in heart rate (R-R intervals) in response to alterations in cardiac sympathetic-parasympathetic autonomic balance (Task Force of the European Society of Cardiology (ESC) and the North American Society of Pacing and Electrophysiology, 1996). The first component, high-frequency power (HF), provides an index of parasympathetic tone and the second, low-frequency power (LF), component is primarily influenced by sympathetic activity and it is the LF-to-HF ratio that is an index of cardiac sympathetic-parasympathetic balance (Task Force of the ESC and the North American Society of Pacing and Electrophysiology, 1996). It is advantageous for individuals to have a high HRV as it signifies that the ANS is able to respond quickly and adapt to changes in the environment.

Post-myocardial infarction patients (Bigger et al., 1992; La Rovere, Bigger, Marcus, Andrea, & Schwartz, 1998) and patients with congestive heart failure (CHF) (Nolan et al., 1998) have been shown to have decreased parasympathetic activity and increased sympathetic activity. The increase in sympathetic activity is a compensatory mechanism to improve cardiac contractility in individuals with CHF and investigators have noted eventual exhaustion of this system (Cheitlin, Sokolow, & McIlroy, 1993). Heart rate variability has been found to be substantially reduced immediately following a MI, but increases spontaneously after an acute cardiac event (Bigger, Fleiss, Rolnitzky,

Steinman, & Schneider, 1991). However, even after 12 months following a MI, HRV remains reduced by one half to one third of that seen in age-and sex-matched healthy controls (Bigger et al., 1995). The data suggest that a low HRV following a MI reflects depressed vagal activity and is not just a reflection of sympathetic overdrive and/or vagal withdrawl due to left ventricular (LV) dysfunction (De Ferrari, Vanoli, & Schwartz, 1995, p. 423). The depressed vagal activity has a strong association with the development of ventricular arrhythmias and sudden cardiac death (De Ferrari et al., 1995, p. 423). Kleiger et al. (1987) studied 808 patients who survived a MI to test the hypothesis that HRV was a predictor of long-term survival. Further, even after adjustments for other prognostic variables such as LV function and ventricular ectopic beats, individuals with HRV of less than 50 milliseconds (ms) had a relative risk of mortality 5.3 times higher than individuals with HRV of more than 100 ms. Martin et al. (1987) analyzed data from 5 patients who died suddenly during ambulatory electrocardiographic monitoring, three of those patients had HRV analyzed. Sudden death was found to be due to ventricular fibrillation which was preceded by ventricular tachycardia in all cases (Martin et al., 1987). Heart rate variability was found to be significantly lower in the patients who died suddenly than in the normal subjects. The authors concluded that HRV analysis may be useful in identifying patients that are at an increased risk of sudden death. Bigger, Fleiss, Rolintzky, and Steinman (1993) also found that power spectral measures of HRV were also strong predictors of all-cause mortality and sudden cardiac death in individuals who had suffered a MI the previous year.

Heart rate recovery and mortality

Recently, a great deal of attention has focused on HR recovery as a measure of parasympathetic activity following exercise (Imai et al., 1994; Ohuchi et al., 2000; Pierpont, Stolpman, & Gornick, 2000). A number of studies have shown that an abnormal HR recovery is an independent predictor of mortality in a number of different populations (Cole et al., 1999; Nishime et al. 2000; Cheng et al., 2003; Nissinen et al., 2003). There is an increasing interest in using HR recovery as a prognostic tool in clinical settings because it provides practitioners with a measure of vagal tone that is easy to calculate and may help identify patients at high risk for future cardiac events (Cole et al., 1999). Identifying higher-risk patients would allow clinicians to provide interventions to these individuals to decrease the risk of future complications.

The contribution of the PNS and the SNS to increase HR during exercise and decrease it after exercise is related to the intensity of the exercise (Imai et al., 1994). At the beginning of exercise, there is withdrawl of the parasympathetic system and as exercise progresses to moderate and vigorous levels, an increase in HR is a result of continued parasympathetic inhibition and the addition of sympathetic activation (Pierpont et al., 2000). Once exercise stops, the parasympathetic system is reactivated quickly within the first minute and maintains its effects over the next 4 to 10 minutes of recovery (Kannankeril, Le, Kadish, & Goldberger, 2004). Withdrawl of sympathetic activation also contributes to early HR recovery (Kannankeril et al., 2004). However, activation of the SNS contributes to maintaining high heart rates during the recovery period after high intensity exercise despite the reactivation of the PNS (Pierpont et al., 2000). Research has

also demonstrated that the slowing of the HR in response to parasympathetic reactivation may follow a first order exponential decay (Pierpont et al., 2000; Imai et al., 1994).

In some individuals the HR does not decrease immediately after exercise. This may be an indication that there is a disruption in the balance between the SNS and the PNS characterized by either a decrease in parasympathetic activity or increase in sympathetic activity or both (Chaitman, 2003). A decrease in parasympathetic activity may result in unopposed sympathetic dominance placing the individual at an increased risk for life-threatening arrhythmias (Billman, Schwartz, & Stone, 1982). Activation of the parasympathetic division of the ANS is thought to exert a protective and anti-fibrillatory effect on the heart (Schwartz, Vanoli, Stramba-Badiale, De Ferrari, Billman, & Foreman, 1988). An abnormal HR recovery is usually defined as a HR that fails to decrease by more than 12 beats per minute (bpm) in the first minute after an EST for protocols that use a post-test cool-down such as slow walking (Cole et al., 1999). Individuals that are more likely to have an attenuated HR recovery after exercise tend to be older, and have a history of hypertension, diabetes, chronic obstructive pulmonary disease, and MI (Chaitman, 2003).

Recently published data have demonstrated that a delayed HR recovery in the first minute after exercise in healthy adults referred for submaximal exercise testing (Cole et al., 1999) and in post-MI patients (Nishime et al., 2000) is associated with a significantly increased risk of mortality. Cole et al (1999) followed 2428 adults for 6 years who had undergone symptom-limited exercise testing for diagnostic purposes. The authors found that 26% of the patients had an abnormal HR recovery which was strongly predictive of mortality. An abnormal HR recovery was found to be associated with higher

mortality among women, the elderly, patients with and without prior history of CHD, patients with and without nuclear perfusion defects and patients taking beta (β)-blockers (Cole et al., 1999). The authors concluded that a delayed HR recovery within the first minute after the cessation of exercise was a powerful predictor of overall-mortality, independent of workload, the presence or absence of myocardial perfusion defects, and changes in HR during exercise. In a similar study by Cole, Foody, Blackstone, and Lauer (2000), a delayed HR recovery after submaximal exercise testing in a cohort of adults free of cardiovascular disease (CVD) was associated with increased all-cause mortality in individuals with risk factors for CVD. An abnormal HR recovery was defined as a HR that did not decreased by ≤42 beats within the first 2 minutes after the EST. An abnormal HR recovery was associated with an increase in all-cause mortality.

Nissinen et al. (2003) examined the prognostic value of HR recovery after an EST among 229 survivors of an acute myocardial infarction (AMI). At the time of the graded exercise test all patients were on β-blocking medication and those with an ejection fraction less than 40% were also taking angiotensin-converting enzyme (ACE) inhibitors (Nissinen et al., 2003). Heart rate recovery was measured as a decrease in HR by 12 beats within the first minute upon termination of the bicycle EST. The results showed that the patients who had not survived had an abnormal HR recovery as well as a low maximal HR during the test compared to those who had survived (Nissinen et al., 2003). The simple measure of HR recovery provided more powerful information on the risk of death than the usual markers of autonomic activity, HRV and BRS (Nissinen et al., 2003). Heart rate variability and BRS showed only modest, non-significant predictions of mortality (Nissinen et al., 2003).

The Heart and Soul study, a prospective study of psychological factors and health outcomes in patients with stable CHD, examined the association of the metabolic syndrome with treadmill exercise capacity (METS) and HR recovery. After adjusting for obesity, physical activity, β-blocker use, diabetes, and hypertension, the authors found that the metabolic syndrome was associated with poor exercise capacity and poor HR recovery (Spies et al., 2005).

The mechanisms as to how an abnormal HR recovery increases mortality is still unclear, but researchers speculate that it may be related more to a disruption between the SNS and the PNS, leaving the heart vulnerable to arrhythmias, than to the presence or extent of CHD (Chaitman, 2003).

The research reviewed provides support for the use of HR recovery as a potential measure of vagal tone as well as a predictive measure of mortality among healthy populations referred for exercise testing (Cole et al., 1999), among survivors of an AMI (Nissinen et al., 2003) and in patients with stable CHD (Spies et al., 2005).

Heart rate recovery and β-blockade

Beta-blockers are commonly used for the treatment of cardiovascular disorders including hypertension, abnormal heart rhythms, angina, and CHF (Opie, Sonnenblick, Frishman, & Thadani, 1995). Their main function is to decrease the work of the heart through a decrease in HR, contractility of the heart muscle and peripheral resistance (Opie et al., 1995). Because of the nature of the population being studied, the influence of β-blockers on HR and on HR recovery is of some concern and an explanation is warranted.

Research has shown that an impaired HR recovery still predicts risk of death in patients taking β-blocking medication when measured using the cut-off value of 12 beats within the first minute after the cessation of a modified or standard Bruce EST (M.S. Lauer, personal communication, May 4, 2004). While a HR recovery of 12 beats or less in the first minute after the cessation of exercise is considered abnormal in the absence of β-blockade, the threshold would most likely be lower with β-blockade and it may be that a cut-off value of 8 beats in the first minute is more accurate in patients undergoing an upright cool down protocol (M.S. Lauer, personal communication, May 31, 2004). However, the cut-off value in patients on β-blockade has not been researched in detail as of yet and it is therefore difficult to comment on the appropriate cut-off value at this time (M.S. Lauer, personal communication, May 4, 2004).

Psychological factors and autonomic balance

Psychological factors, such as anxiety, hostility, depression, and social isolation have been linked to poor health status in numerous populations (Barefoot & Schroll, 1996; Dembroski, MacDougall, Costa, & Grandits, 1989; Kawachi et al., 1994; Orth-Gomer, Rosengren, & Wilhelmsen, 1993). Specifically, anxiety and hostility have been implicated in increasing the risk of mortality among healthy individuals as well as those with CHD. The following is a review of the literature of the pathophysiologic mechanisms underlying the relationship between anxiety, hostility and mortality as well as the relationship between hostility and the development and severity of CAD.

Anxiety.

Anxiety can be defined as an emotion that has biological as well as psychological components. It consists of unpleasant feelings of tension, apprehension,

nervousness, and worry, and activation of the ANS. The physiological response to anxiety generally includes increased BP and HR (palpitations or tachycardia), sweating, dryness of the mouth, nausea, dizziness, irregularities in breathing, muscle tension, as well as restlessness, tremors, and feelings of weakness (Spielberger & Rickman, 1990).

Among the numerous theories that define anxiety, Speilberger's State-Trait anxiety theory provides a conceptual framework which specifies the relationship between two anxiety concepts and other variables related to stress and anxiety (Speilberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). Speilberger (1966) made the distinction between state anxiety and trait anxiety stating that the trait component of anxiety is believed to be a stable behavioural tendency to experience anxiety and the state component is often short-term and situationally provoked. The transitory emotions of state anxiety include consciously perceived feelings of tension and apprehension (Speilberger et al., 1983). People who have high trait anxiety are most likely to perceive stressful situations as being personally dangerous or threatening and to respond to such situations with elevations in state anxiety (Spielberger & Rickman, 1990).

Anxiety has been associated with fatal CAD, particularly sudden cardiac death. It has not been associated with MI, suggesting that ventricular arrhythmias may be the cause for cardiac death among individuals with anxiety disorders (Rozanski et al., 1999). Kawachi, Sparrow, Vokonas, and Weiss (1994) assessed the symptoms of anxiety and risk of CHD in a subgroup of 2271 men in the Normative Aging Study. In the 32 years of follow-up, the authors found that men reporting two or more symptoms of anxiety had elevated risks of fatal CHD, and sudden cardiac death with the age-adjusted odds ratio being 3.20 and 5.73 for fatal CHD and sudden death, respectively (Kawachi, Sparrow et

al., 1994). Kawachi, Colditz et al. (1994) examined the association between self-reported symptoms of phobic anxiety and subsequent risk of CHD in a cohort of 33, 999 US male health professionals, who were free of diagnosed CVD at baseline. The results showed that 128 cases of non-fatal MI and 40 cases of fatal CHD occurred and the risk of fatal CHD increased with higher levels of phobic anxiety at the 2-year follow up (Kawachi, Colditz et al., 1994). When fatal CHD was further categorized into sudden and non-sudden coronary death, the excess risk was confined to sudden death and no association was found between phobic anxiety and risk of non-fatal MI (Kawachi, Colditz et al., 1994). After adjusting for a variety of cardiovascular risk factors, the findings remained essentially unchanged (Kawachi, Colditz et al., 1994). The authors' findings support a strong association between phobic anxiety and fatal CHD in which the excess risk of mortality may have resulted from ventricular arrhythmias.

It has also been observed that individuals with anxiety disorders have a low HRV which may suggest that anxious individuals have an impaired ANS (Kawachi, Sparrow, Vokonas, & Weiss, 1995). This alteration could involve either increased sympathetic stimulation, which has been linked to the occurrence of arrhythmias and sudden death (Lown, 1982) or impaired vagal control, which has also been linked to increased cardiac mortality (Farrell et al., 1987; Rich et al., 1988). It is postulated that reductions in central parasympathetic activity may also contribute to decreased HRV and increased HR during periods of anxiety as demonstrated by the induction of panic attacks during placebo procedures in the laboratory (Goetz et al., 1993). Again, when parasympathetic activity to the heart is reduced, sympathetic tone is unopposed, placing the heart at risk for developing fatal arrhythmias (Gorman & Sloan, 2000).

To test the hypothesis that individuals with anxiety disorders may have reduced HRV increasing the odds of sudden cardiac death, Kawachi et al. (1995) assessed symptoms of anxiety and measured HRV in 581 men from the Normative Aging Study. The results showed that men with higher levels of phobic anxiety had higher resting heart rates and, after adjusting for age, mean HR, and body mass index (BMI), were also found to have a lower HRV (Kawachi et al. 1995). The finding that cardiac autonomic balance is altered in individuals with phobic anxiety is consistent with the literature that demonstrates that an alteration in cardiac autonomic balance is linked to sudden cardiac death (Kawachi et al., 1995).

Watkins et al (1998) examined whether anxiety was associated with reduced vagal control of HR in 93 healthy men and women. Anxiety was assessed using the Speilberger State Trait Anxiety Inventory (STAI) and baroreflex control (BRC) of HR and respiratory sinus arrhythmia (RSA) were used to estimate vagal control. The authors found that participants with high trait anxiety (trait anxiety score > 41) had a significant reduction in BRC and RSA, 36 % and 8%, respectively, indicating reduced vagal control of the HR compared to participants with low trait anxiety (trait anxiety score <31). Trait anxiety scores were also found to be negatively correlated with levels of BRC and RSA and heart rate and state anxiety were significantly higher in trait anxious participants. This study identifies that reduced vagal control is not limited to individuals with diagnosed anxiety disorders, but also occurs in healthy men and women.

Although the association between anxiety and vagal control of HR has not been evaluated using HR recovery, previous studies examining the relationship between anxiety and autonomic balance as measured by HRV have found that anxious individuals

have a low HRV indicating an imbalance in autonomic control of the heart. Like HRV, HR recovery is also thought to be a measure of autonomic function, mostly of parasympathetic activity, and it can be hypothesized that anxious individuals may also display an abnormal HR recovery after an EST which may indicate impairment of the ANS.

In furthering the support of using HR recovery, Shishehbor, Baker, Blackstone, and Lauer (2002) found that educational status was independently associated with an abnormal HR recovery. It has been hypothesized that individuals with a lower educational level are more likely to experience psychological and behavioural stress due to ineffective coping strategies, lower sense of control, and feelings of powerlessness (McLeod & Kessler, 1990). These chronic stressful situations result in an imbalance in ANS function (Langewitz, Ruddel, & Schachinger, 1994; Rostrup, Westheim, Kjeldsen, & Eide, 1993). Lynch, Kaplan, and Salonen (1997) also found higher rates of hopelessness, depression, and cynical hostility among individuals with a lower educational level. Stressful situations as experienced by individuals with a lower education level appear to include components of anxiety such as helplessness or loss of a sense of control which has been associated with an abnormal HR recovery.

In summary, symptoms of anxiety are predictive of subsequent fatal CHD, independent of conventional medical risk factors, and this risk is specific for sudden cardiac death. A reduced HRV has also been observed in anxious patients indicating autonomic dysfunction in this group. Although research has not looked at the influence of anxiety on autonomic function using HR recovery, it can be speculated that anxious

individuals may also display an abnormal HR recovery which may signify an impairment in their autonomic function.

Hostility.

Hostility has been recognized as the "toxic" component of the previously studied type A behaviour pattern (TABP) which is characterized by competitiveness, impatience and hostility. Hostility is characterized by anger, cynicism, and mistrust (Rozanski et al., 1999) and is assumed to be a stable psychological trait that has been associated with CAD severity, occurrence of new CHD events, and CHD deaths (Williams et al., 1980)

In assessing the relationship between TABP, hostility and presence and severity of CAD, Williams et al. (1980) found that a significantly greater proportion of type A individuals (compared to non-type A individuals) had an occlusion of 75% or greater in at least one artery. The authors also found that the patients in all groups who scored higher than 10 on the Cook-Medley Hostility Scale showed a 70% higher rate of significant disease. The hostility score was found to be more strongly related to severity of CAD than the TABP (Williams et al., 1980). Shekelle, Ostfeld, and Paul (1983) assessed the relationship of hostility scores to the risk of CHD and death. Using data from the Western Electric Study, Shekelle et al. (1983) conducted a follow-up of the participants, looking at the 10-year incidence of major coronary events (MI and CHD death) and found that the higher hostility scores were prospectively associated with increased CHD morbidity. The odds of a major cardiac event occurring was 1.47 times greater in men with scores greater than 10 on the Cook-Medley scale compared to men with lower scores (Shekelle et al.,1983). In a 25-year follow-up study on 255 physicians,

Barefoot et al. (1983) found that high hostility scores were more predictive of both clinical coronary disease incidence and total mortality.

Results from the above studies indicate that hostility is associated with CAD and mortality, but how does hostility exert its effects on the cardiovascular system and increase the risk of mortality? Behavioural, pathophysiologic and autonomic control mechanisms have been proposed.

Hostility is associated with a higher concentration of unhealthy lifestyle behaviours, including smoking, poor diet, obesity, and physical inactivity (Everson et al., 1997) which directly increases risk factors for CVD. Hostile individuals are more likely to manifest other psychological factors associated with CAD, such as social isolation (Blumenthal, Barefoot, Burg, & Williams, 1987), and an accumulating body of evidence suggests physiological responses to stressors link hostility to CAD. For example, during an interpersonal stressor, high hostile men and women compared to non-hostile individuals exhibited a greater increase in diastolic BP and total peripheral resistance and a smaller increase in cardiac output (Davis, Matthews, & McGrath, 2000). The authors also found that individuals higher in hostility had enhanced vascular reactivity not only during the social challenge but also during the preparation period of the task. Suarez and Blumenthal (1991) also found that post-MI patients with high hostility scores exhibited higher systolic BP responses to increased emotional stress, but this was not the case for individuals with low hostility scores.

Preliminary data suggest that hostile individuals may also manifest diminished vagal control of heart function (Sloan et al., 1994). Sloan et al (1994) examined the relationship between cardiac autonomic control and hostility among 38 healthy subjects.

The results from the study showed that in normal subjects under 40 years of age, hostility was significantly related to a decrease in parasympathetic control of the heart and an increase in sympathetic dominance when measured by 24-hour heart period variability (Sloan et al., 1994). The relations were found to be much stronger during daytime hours than at night which is consistent with Smith's transactional hypothesis (Sloan et al., 1994). The transactional hypothesis states that hostile individuals interact with their environment in ways that create interpersonal conflict resulting in more stressful life experiences (Smith, 1992). Hostile individuals are more likely to have an increased BP and HR in reaction to these stressful situations.

Hostility has been shown to be the most prominent component of the TABP and affects CVD risk either directly through its effects on the vasculature or indirectly through behavioural mechanisms. It has also been found that hostile individuals demonstrate autonomic dysfunction putting them at an increased risk of death.

Summary

An extensive literature exists attesting to a strong association between autonomic imbalance and mortality. The studies reviewed on HRV, HR recovery, anxiety, and hostility provides evidence for their association with mortality in healthy individuals as well as in cardiac patients. The review of the literature on anxiety and hostility support the findings that these psychological factors may have an influence on vagal tone either directly or through an increase in sympathetic responses to the emotions that characterize negative psychological states. The influence that anxiety and hostility have on autonomic function is evident in the identification of a low HRV.

More recently, HR recovery has also been identified as a measure of parasympathetic activity. It is being used, along with other EST clinical variables such as the Duke exercise treadmill score (a quantitative means of expressing cardiac risk derived from the exercise ECG) and the chronotropic response to exercise (response of the HR to graded exercise), to identify individuals at an increased risk of mortality (Lauer, 2003). If HR recovery is found to be sensitive enough to pick up the effects of emotions on autonomic function, it may prove to be useful in identifying anxious or hostile individuals at an increased risk for future cardiac events or mortality.

Method

The Northern Alberta Cardiac Rehabilitation Program

The Northern Alberta Cardiac Rehabilitation Program (NACRP) is an out-patient program that generally begins within 4 to 6 weeks after a patient is discharged from the hospital following a cardiac event. The program combines medically supervised exercise sessions with education classes on nutrition, cardiovascular risk factors, and stress management as well as behaviour-modification education. Patients attend the program for approximately 4 to 8 weeks, 1 to 3 times per week for about 1 hour. Patients exercise on a variety of exercise equipment including treadmills, stationary bikes and rowing machines, as well as incorporating a resistance training program. Adjustments to the patients' program are made by a physician, exercise specialist and other medical personnel.

Participants

Eligible participants included those who were referred by a physician and completed an EST at the NACRP, had a history of MI, angina, or revascularization procedures such as percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG) surgery. Patients with atrial fibrillation, CHF, recent valve surgery, paced rhythms, Wolff-Parkinson-White (WPW) syndrome, EST complications or who failed to return the questionnaire prior to the EST at the NACRP were excluded from the study. It was calculated that a given sample size of 64 with an alpha set at .05 would give the study a power of .80 for tests of analysis of variance (Cohen, 1992).

Over 10 months, 123 participants, 92 men (mean age = 60.10 ± 10.70 years) and 31 women (mean age = 67.26 ± 10.28 years) were recruited. Approximately 50% of all participants had an admitting diagnosis of MI, with 50% and 20% undergoing PCI and

CABG procedures, respectively. Approximately 11% of participants had suffered a previous MI. The most common medications that participants were taking were antiplatelets (90.24%), β-blockers (89.43%), lipid lowering medication (89.43%), and ACE inhibitors (74.79%). About 12% of participants were current smokers.

Of the 123 participants recruited, 71 participants were ineligible for the study due to the following medical reasons: recent valve surgery (11%), atrial fibrillation (8%), CHF (7%), pacemaker (6%), or WPW syndrome (1%). The mean age of ineligible male and female participants was 61.77 ± 11.10 and 66.39 ± 10.41, respectively. Approximately 41% of participants were admitted to hospital with a MI with 38% and 21% undergoing PCI and CABG, respectively. Cardiovascular risk factors and other health-related problems included high cholesterol (58.5%), arthritis (51%), high BP (49%), diabetes (21%), stomach problems (19%), and asthma (11%).

The final analysis included data from 52 participants, 44 men (mean age = 58.25 ± 9.90 years) and 8 women (mean age = 69.75 ± 10.12 years). Sixty-two percent of participants were admitted to hospital with a MI, with 67% and 19% undergoing PCI and CABG, respectively. Approximately, 8% of participants had suffered a previous MI. The most common cardiovascular risk factors and health-related problems participants suffered from were high cholesterol (69%), arthritis (63%), high BP (44%), diabetes (21%), and asthma (12%). About 10% of participants were current smokers.

Measurements

This study included measures of HR, BP, electrocardiograph (ECG) recordings of the heart, exercise capacity, HR recovery, state and trait anxiety and trait hostility. Patient demographics and medical history were also recorded.

Patient demographics and clinical data.

The patient's age, sex, height, weight, percent body fat, education level, marital and employment status were obtained from medical records. Percent body fat was assessed by a dietician using skinfold measurements and calculated based on the Jackson and Pollock method (Jackson & Pollock, 1985). Clinical data such as current medication use, cardiac risk factors, previous cardiac events and procedures and other cardiac and non-cardiac diagnoses were also obtained from medical records.

Blood pressure.

Blood pressure was measured manually prior to the stress test in a sitting, supine and standing position and every 3 minutes throughout the EST. Blood pressure was assessed by the ECG technologist or physician, using a sphygmomanometer and stethoscope. Systolic pressure was measured at the first Korotkoff sound and diastolic pressure was measured at Phase 5 when there was an absence of sound.

Exercise testing.

The EST generally occurred within 10 days of entry into the CR program. Exercise testing was performed using standard or modified Bruce protocols and was conducted by a physician or the ECG technologist. The standard Bruce protocol is a graded exercise treadmill test that has 6 stages. Speed and grade increase every 3 minutes until volitional exhaustion. Speed increases from 1.7 mph in the first stage to 5.5 mph in

the sixth stage with the grade starting at 10% and increasing by 2% every stage (Bruce, Kusumi, & Hosmer, 1973). The modified Bruce protocol is also a graded exercise treadmill test, however, it starts at 1.7 mph and a 0% grade and increases in speed and grade every 3 minutes until volitional exhaustion (Bruce et al., 1973). The modified Bruce is often used for cardiac patients who have limited functional capacity or other health problems that may prevent them from walking on an incline on the treadmill. Each patient was prepared with a routine 12 lead ECG. Blood pressure, HR and ECG were recorded immediately prior to starting exercise in a resting supine and standing position and at regular intervals throughout the test. The ECG was continually monitored throughout the test as well as 5 minutes into recovery. The symptom limited stress test was terminated when one or a combination of the following occurred: the patient reached their target HR, the patient developed symptoms such as angina, dizziness, dyspnea, leg claudication or marked tiredness, the patient developed ECG changes, or if there was a fall in blood pressure or drop in HR. Exercise capacity was reported in metabolic equivalents (METS) and minutes completed on the treadmill. Metabolic equivalents is a unit used to estimate the metabolic cost or energy requirements of physical activity. One MET is defined as an oxygen uptake of 3.5mL of oxygen per kilogram body weight per minute and is considered a resting metabolic rate obtained during quiet sitting (Jette, Sidney, & Blumchen, 1990).

Heart rate recovery.

Once patients reached volitional exhaustion, they walked on the treadmill for 1 minute at 1.0 mph and a 0% grade. The patient then rested in a supine position on a stretcher for 5 minutes until HR, BP and ECG returned to near resting values. These

values were continuously recorded for the 5 minutes. The value for the recovery HR was defined as the reduction in heart rate from the rate at peak exercise to the rate after walking at 1.0 mph, 0% grade for 1 minute during the cool down period (Cole et al., 1999). The heart rate at peak exercise and at 1 minute into recovery were measured from the ECG rhythm strip using a rate ruler upon completion of the EST. Heart rate recovery was calculated by subtracting the HR at 1 minute into recovery from the peak HR. Heart rate recovery was assessed as a continuous variable as well as a categorical variable using a cut-off value of < 12 bpm which has been associated with increased mortality in previous studies (Cole et al, 1999).

Anxiety.

Anxiety was assessed using the STAI developed by Speilberger et al. (1983). The STAI has been used to measure anxiety in a number of populations such as working adults, college students, military recruits, medical and psychiatric patients. It has proven to be useful in both clinical work and research. The STAI is a self-report assessment tool that clearly differentiates between the temporary condition of state anxiety and the more general and long-standing quality of trait anxiety.

The STAI Trait Anxiety (T-Anxiety) scale (Appendix A) consists of 20 statements that ask people to describe how they generally feel (Speilberger et al., 1983). Subjects respond to each item by rating themselves on a four-point scale ranging from 1 (*almost never*) to 4 (*almost always*). The STAI State Anxiety (S-Anxiety) scale (Appendix B) consists of 20 statements that ask people to rate the intensity of how they feel at that particular moment by rating themselves on a four-point scale ranging from 1 (*not at all*) to 4 (*very much so*) (Speilberger et al., 1983). Cronbach's alpha values of .91

and .93 have been found for the trait anxiety and state anxiety scales, respectively, in male and female working adults. Correlations between trait anxiety and state anxiety scores have been found to be .65. This correlation tends to be quite strong because individuals with high trait anxiety tend to respond with higher state anxiety in situations that they are trait anxious for.

Both scales consist of items addressing the *presence* and *absence* of anxiety. A score of 4 on any item indicates a high level of anxiety for 10 S-Anxiety items and 11 T-Anxiety items (e.g., "I feel upset"). For the remainder of the statements on each scale, a high score indicates the absence of anxiety (e.g., "I feel relaxed"). Scores for the anxiety absent items were reversed. To obtain a score for the S-Anxiety and T-Anxiety scales, the mean weighted score was determined for the scale items and then multiplied by 20 to account for omitted responses. Participants in this study with a mean score greater than 39 on the STAI were categorized as having high anxiety. High and low anxiety groups were formed based on this cutoff value. Previous studies have established that a score of 36 represents the norm for working adults, whereas a mean score greater than 39 indicates that anxiety may be a clinical problem (Spielberger et al., 1983). Mean scores between 45 and 55 are typical of psychiatric patients with symptoms of anxiety (Spielberger et al., 1983). Frasure-Smith et al (1995) used cut-off values of greater than or equal to 40 when assessing anxiety with the STAI in a cardiac population. Cronbach's alphas, in this sample, were found to be .96 and .95 for the state anxiety and trait anxiety scales, respectively.

Hostility.

The Hostile Attitude Scale (HAS), developed by Arthur, Garfinkel, and Irvine (1999), was used to measure hostility (Appendix C). Hostility has primarily been measured by the Cook-Medley Hostility Scale (Cook and Medley, 1954) and the Type A Structured Interview (SI) (Rosenman, 1978). The Cook-Medley Hostility Scale tends to assess the behavioral component of hostility such as anger-proneness and aggressive responding (Barefoot et al., 1991; Smith & Frohm, 1985) whereas the SI tends to focus on the affective component of hostility (e.g., resentment, contempt, annoyance) (Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985). The HAS focuses on the cognitive aspects of hostility rather than on the behavioural or affective components. It is thought that the behavioural and affective components of hostility are primarily situational responses, whereas the cognitive component is more stable and comprehensive because it involves our attitudes (Arthur et al., 1999). Higher HAS scores have been associated with an increased amount of atherosclerosis as measured by coronary angiography (Arthur et al., 1999). The HAS is composed of 18-items that use a 7-point scale with responses ranging from 1 (*strongly disagree*) to 3 (*mildly disagree*) to 7 (*strongly agree*). Previous research using this scale found mean hostility scores varied between 75 and 78 (SD = 14 or 15) and individuals with scores greater than 1 SD above the mean, 89, were categorized as having high hostility. The HAS has been found to be valid and reliable with a Chronbach's alpha of .80 for the 18-items.

Scores for the HAS were obtained by determining the mean of the 18 weighted items and multiplying the score by 18 to account for omitted responses. In this sample, participants were categorized as having high hostility if they had a mean score equal to or

greater than 78 which was 1 SD above the mean (60±18) in this sample. Cronbach's alpha for the HAS in this sample was .86.

Procedures

Questionnaires were mailed to patients who were referred to the NACRP. The questionnaire package included a letter from the NACRP supporting the research, a letter explaining the details of the study, consent forms, and the baseline questionnaires including the STAI T-Anxiety scale and the HAS. Participants were asked to fill out the questionnaires prior to attending the orientation if they would like to participate in the study and to contact the researchers if they had any questions or concerns. Patients were asked to return the completed questionnaires and informed consent document at their initial orientation to the NACRP approximately 3 to 5 weeks later.

The researcher attended the orientation to meet participants, answer any questions or concerns about the research study or questionnaires and to collect completed questionnaires. Individuals who agreed to participate were met by a researcher 1 to 2 weeks later at the participant's initial EST when they were asked to fill out the STAI S-Anxiety scale. The STAI S-Anxiety scale was administered about 15 minutes prior to the patients' EST.

Ethical approval for this study was granted by the Health Research Ethics Board at the University of Alberta and the Office of Research Services at the Glenrose Rehabilitation Hospital. Support was also received from the Medical and Regional Directors of the NACR program.

Statistical Analysis

Anxiety and heart rate recovery

To achieve the first objective, participants (N=52) were divided into high (>39 on the STAI) versus low (< 39 on the STAI) trait anxiety to examine differences between these two groups. One-way analysis of variance and chi-square were used to test for differences in demographic, clinical and EST characteristics in participants with high and low trait and state anxiety. Pearson correlations were also conducted to look at the relationship between trait anxiety and HR recovery.

Hostility and heart rate recovery

To achieve the second objective, participants were divided into groups based on high and low hostility scores. One-way analysis of variance and chi square were used to test for differences in demographic, clinical and EST characteristics in participants with high and low hostility. Pearson correlations were conducted to assess the relationship between hostility and HR recovery.

Characteristics of participants with a normal versus abnormal heart rate recovery

The third objective was to examine differences on baseline demographic, clinical, and EST characteristics in individuals with a normal and an abnormal HR recovery. Two groups were formed based on a HR cut off value of ≥ 12 bpm and < 12 bpm. One-way analysis of variance and chi square were conducted to test for differences between these two groups.

Statistical analyses were performed with SPSS version 13.0 software.

Results

Characteristics of all men and women

One-way analysis of variance, and chi square were conducted to evaluate differences between men and women in this sample of 123 participants. Gender differences are known to exist in CR programs. Statistical differences were found between men and women's age, percentage of body fat, health related problems, admitting diagnoses, clinical chemistry, EST characteristics and psychological measures. These results can be found in Tables 1 - 3.

Women were found to be older (mean age = 67±10 years) than men (mean age = 60±11 years), $F(1,121) = 10.63$, $p = 0.001$, which is a common finding in CR programs. Women generally present with cardiovascular disease about 10 years later than men (Lerner & Kannel, 1986). Women also had a higher percentage of body fat than men (31±6%, 25±4%), $F(1,98) = 35.90$, $p < 0.01$. Women were found to have more arthritis (74% compared to 43%), $X^2(1, N=123) = 8.76$, $p = 0.003$, but men and women experienced other cardiovascular risk factors and health-related problems such as asthma, diabetes, high BP and stomach problems at about the same rate. Although men and women had similar rates of angiography and PCI, men were more likely to undergo CABG (25%) than women (6%), X^2 (1, N=123) = 4.93, $p = 0.03$. Previous research has also found that men undergo more invasive procedures than women (Shaw et al., 1994). Women had higher HDL cholesterol levels (1.18±.36 mmol/l) compared to men (0.99±0.20 mmol), $F(1, 115) = 13.33$, $p < 0.01$.

An examination of the differences between men and women on the psychological variables revealed that women had significantly higher state anxiety scores (39±9)

compared to men (32±10), $F(1, 75) = 5.71$, $p = 0.02$, but men had significantly higher hostility scores (62±16) than the women (53±16), $F(1, 121) = 6.56$, $p = 0.01$. Men and women did not differ significantly on trait anxiety scores, although women's scores were slightly higher.

Table 1

Frequency and percent of baseline characteristics of all men and women

Characteristics	Men	Women	*p*
	Frequency (% of sample)		
No. of patients	92(75)	31(25)	
Health-related problems present			
high BP	41(45)	19(61)	0.11
high cholesterol	50(54)	22(71)	0.10
diabetes	22(24)	4(13)	0.19
asthma	10(11)	4(13)	0.76
arthritis	40(44)	23(74)	0.003
stomach	18(20)	5(16)	0.67
Current medication use			
β-blocker	85(92)	25(81)	0.07
anticoagulant	19(21)	5(16)	0.58
antiplatelet	84(91)	27(87)	0.50
lipid lowering	82(89)	28(93)	0.85
ace inhibitor	68(74)	24(77)	0.70
anti-depressant	3(3)	3(10)	0.15
anti-anxiety	1(1)	-	0.56
Admitting diagnoses			
MI	45(49)	16(51.61)	0.80
angiogram	84(91)	27(87.10)	0.50
PCI	48(52)	14(45.16)	0.64
CABG	23(25)	2(6.45)	0.03
bundle branch block	5(5)	-	-
angina	15(61)	5(6.20)	0.98
Previous MI	13(14)	1(3.23)	0.10
Smoking status			
current smoker	12(13)	2(6)	0.05
former smoker	49(53)	12(39)	
never smoked	27(29)	17(55)	

Note: ACE, angiotensin converting enzyme; BP, blood pressure; CABG, coronary artery bypass graft; MI, myocardial infarction; PCI, percutaneous coronary intervention.

Table 2

Baseline characteristics of all men and women

Characteristics	Men	Women	p	η_p^2
	Mean±SD			
Age (years)	60±11	67±10	0.001	0.08
Body fat	25±4	31±6	0.000	0.27
Body mass index (kg/m^2)	30±5	29±7	0.53	0.00
Days from cardiac event to EST	58±28	64±39	0.43	0.00
Clinical chemistry (mmol/l)				
total cholesterol	3.76±0.95	4.10± 0.90	0.09	0.02
triglycerides	1.67±0.98	1.48± 0.66	0.34	0.01
HDL cholesterol	0.99±0.20	1.18±0.36	0.000	0.10
LDL cholesterol	2.01±0.70	2.27± 0.60	0.78	0.03
Psychological variables				
state anxiety score	32±10(N=59)	39±9(N=18)	0.02	0.07
trait anxiety score	35±11(N=92)	37±10(N=31)	0.28	0.01
hostility score	62±16(N=92)	53±16(N=31)	0.01	0.05

Note: EST, exercise stress test; HDL, high-density lipoprotein; LDL, low-density lipoprotein; η_p^2, partial Eta squared.

When examining differences on EST characteristics (Table 3), men were found to have longer EST times (7:36±2:21 minutes, 5:01±2:21 minutes), $F(1, 109) = 24.17, p < 0.01$, and achieved higher METS (8.6±3.0 METS, 5.2±2.6 METS), $F(1, 108) = 27.68, p < 0.01$, than women. Effect sizes were $\eta_p^2 = 0.18$ and $\eta_p^2 = 0.20$, respectively.

Table 3

Exercise stress test characteristics for men and women

Characteristics	Men	Women	p	η_p^2
	Mean±SD			
Test duration (min)	7:36±2:21(N=85)	5:01±2:21(N=26)	0.000	0.18
METS	8.6±3.0(N=85)	5.2±2.6(N=25)	0.000	0.20
Resting supine HR (bpm)	65±13(N=81)	66±12(N=25)	0.81	0.00
Supine SBP (mmHg)	121±18(N=78)	124±21(N=18)	0.54	0.00

Table 3 (continued)

Characteristics	Men	Women	*p*	η_p^2
Supine DBP (mmHg)	66±8(N=78)	67±9(N=18)	0.80	0.00
Pre-ex standing HR (bpm)	71±15(N=63)	71±13(N=18)	0.96	0.00
Peak HR (bpm)	124±24(N=79)	114±19(N=24)	0.06	0.03
% of maximum HR achieved	78±13(N=78)	77±13(N=24)	0.83	0.00

Note: bpm, beats per minute; DBP, diastolic blood pressure; HR, heart rate; METS, metabolic equivalents; min, minutes; pre-ex, pre-exercise; SBP, systolic blood pressure; η_p^2, partial Eta squared.

Characteristics of eligible and ineligible participants

Of the 123 participants who completed the initial questionnaire, 71 participants did not meet the criteria to be included in the study. Participants were excluded due to clinical characteristics such as CHF, atrial fibrillation, paced rhythms, recent valve surgery and WPW which accounted for 21% of participants being excluded. Other reasons for exclusion included the EST not being completed at the NACRP (9%), improper EST protocol (9%), EST complicated by arrhythmias (5%), unable to obtain HR recovery measures (8%) and initial questionnaires being handed in after the EST (5%). Percentages represent the percent of individuals of the total sample (N=123) who were excluded based on specific exclusion criteria. Demographic and clinical data as well as scores on the psychological measures can be found in Tables 4 and 5 for eligible and ineligible participants.

Results of the ANOVA and chi square tests revealed no significant differences in demographic, clinical, or psychological variables between participants who were eligible or ineligible for the study except on EST characteristics.

Table 4

Frequency and percent of baseline characteristics for ineligible and eligible participants

Characteristics	Ineligible	Eligible	*p*
	Frequency (% of sample)		
No. of patients	71(58)	52(42)	
Health-related problems present			
high BP	60(49)	23(44)	0.39
high cholesterol	72(59)	36(69)	0.04
diabetes	26(21)	11(21)	0.99
asthma	14(11)	6(12)	0.96
arthritis	63(51)	33(63)	0.02
stomach	23(19)	14(27)	0.05
Current medication use			
β-blocker	62(87)	48(92)	0.37
anticoagulant	15(21)	9(17)	0.60
antiplatelet	60(85)	51(98)	0.01
lipid lowering	61(86)	49(94)	0.14
ace inhibitor	49(69)	43(83)	0.08
Admitting diagnosis			
MI	29(41)	32(62)	0.02
angiogram	60(85)	51(98)	0.01
PCI	27(38)	35(67)	0.001
CABG	15(21)	10(19)	0.80
CHF	5(7)	-	
valve surgery	8(11)	-	
pacemaker	4(6)	-	
atrial fibrillation	6(8)	-	
wpw	1(1)	-	
bundle branch block	1(1)	4(8)	0.08
cardiomyopathy	2(3)	-	
angina	8(11)	12(23)	0.08
Previous MI	10(14)	4(8)	0.27
Smoking status			
current smoker	9(13)	5(10)	0.81
former smoker	35(52)	26(51)	
never smoked	24(35)	20(39)	

Note: ACE, angiotensin converting enzyme; BP, blood pressure; CABG, coronary artery bypass graft; MI, myocardial infarction; PCI, percutaneous coronary intervention.

Table 5

Baseline characteristics of ineligible and eligible participants

Characteristics	Ineligible	Eligible	p	η_p^2
	Mean±SD			
Age (years)	63±11	60±11	0.11	0.02
Body fat (%)	27±6	26±5	0.16	0.02
Days from cardiac event to EST	64± 31	55±29	0.16	0.02
Number of medications	6±2	7±2	0.15	0.02
Clinical chemistry (mmol/l)				
total cholesterol	3.81±0.94	3.89±0.96	0.68	0.00
triglycerides	1.65±0.78	1.59±1.06	0.75	0.00
HDL cholesterol	1.05±0.30	1.03±0.20	0.66	0.00
LDL cholesterol	2.03±0.67	2.13±0.70	0.44	0.01
Psychological variables				
state anxiety score	31±10	35±10	0.16	0.03
trait anxiety score	34±10	37±12	0.09	0.02
hostility score	59±16	60±18	0.78	0.00

Note: EST, exercise stress test; HDL, high-density lipoprotein; LDL, low-density lipoprotein; η_p^2, partial Eta squared.

Using the Bonferroni approach to control for Type I error across the 8 EST variables, a p value of less than 0.006 (0.05 / 8 = 0.006) was required for significance. Significant differences were found between the two groups on exercise test duration, $F(1,109) = 9.19, p = 0.003$, and METS achieved, $F(1,108) = 11.26, p = 0.001$ (Table 6). Total exercise time and METS achieved was lower in the ineligible group (6:19 ± 2:29 minutes, 6.9±3.0 METS) compared to the eligible group (7:46 ± 2:30 minutes, 8.9±3.2 METS)

Table 6

Exercise stress test characteristics for ineligible and eligible participants

Characteristics	Ineligible	Eligible	*p*	η_p^2
	Mean±SD			
Test duration (min)	6:19±2:29	7:46±2:30	0.003	0.08
METS	6.9±3.0	8.9±3.2	0.001	0.10
Resting supine HR (bpm)	66±13	64±12	0.34	0.01
Supine SBP (mmHg)	123±1	120±20	0.45	0.01
Supine DBP (mmHg)	66±8	66±9	0.98	0.00
Pre-ex standing HR (bpm)	70±15	70±15	0.94	0.00
Peak HR (bpm)	116±24	126±21	0.02	0.05
% of maximum HR achieved	76±14	79±12	0.32	0.01

Note: bpm, beats per minute; DBP, diastolic blood pressure; HR, heart rate; min, minutes; METS, metabolic equivalents; pre-ex, pre-exercise; SBP, systolic blood pressure; η_p^2, partial Eta squared.

Characteristics of eligible men and women

Differences were found in exercise stress test duration, $F(1, 50) = 9.11$, $p = 0.004$, and MET values, $F(1, 50) = 14.37$, $p < 0.01$ between men and women in the eligible group (Table 7). Men had higher EST times and MET scores (8:11 2:21 minutes, 9.48± 2.81 METS) compared to women (5:29±2:07 minutes, 5.39±2.79 METS) with effect sizes of $\eta_p^2 = 0.15$ and $\eta_p^2 = 0.22$, respectively. There were no significant differences on HR recovery values between men and women (20±10 vs 18±12; $F(1, 50) = .216$, p = 0.64).

Table 7

Exercise stress test characteristics for eligible men and women

Characteristics	Men	Women	p	η_p^2
	Mean ± SD			
Test duration (min)	8:11±2:21	5:29±2:07	0.004	0.15
METS	9.5±2.8	5.4±2.8	0.000	0.22
Resting supine HR (bpm)	64±12	63±11	0.80	0.00
Supine SBP (mmHg)	118±19	136±23	0.06	0.08
Supine DBP (mmHg)	65±8	72±10	0.14	0.05
Pre-ex standing HR (bpm)	72±15	69±12	0.66	0.01
Peak HR (bpm)	129±20	113±22	0.06	0.07
% of maximum HR achieved	80±12	75±11	0.31	0.02
HR recovery (bpm)	20±10	18±13	0.64	0.00

Note: bpm, beats per minute; DBP, diastolic blood pressure; HR, heart rate; METS, metabolic equivalents; pre-ex, pre-exercise; SBP, systolic blood pressure; η_p^2, partial Eta squared.

There were significant correlations between state anxiety and trait anxiety, $r(39) = .72, p < 0.01$, trait anxiety and hostility, $r(42) = .45, p < 0.01$, age and METS, $r(42) = 0.40, p < 0.01$, and METS and HR recovery, $r(42) = .68, p < 0.01$, in eligible men, but not in eligible women (Table 8). There was a trend for higher anxiety and hostility scores to be negatively related to HR recovery in women. Correlations may not have reached statistical significance in women due to the low power in this group (N = 8).

Table 8

Correlation matrix among baseline demographic, EST, and psychological measures for eligible male participants (N=44)

Variable	1	2	3	4	5	6
1.Age	-					
2.S-Anxiety	-.17	-				
3.T-Anxiety	-.33*	.72**	-			
4.HAS	.02	.15	.45**	-		
5.HR recovery	-.29	-.10	-.11	.02	-	
6.METS	-.40**	-.08	-.21	-.17	.68**	-

Note: S-Anxiety, state anxiety; T-Anxiety, trait anxiety; HAS, Hostile Attitude Scale; HR, heart rate; METS, metabolic equivalents.
*$p < .05$; **$p < .01$

Relationship between demographic, clinical and psychological variables for eligible participants

The results of the correlation analyses, presented in Table 9, show that there were significant correlations between HR recovery and age, $r(50) = -.35, p < .05$, peak HR achieved on the EST, $r(50) = .50, p < .01$, supine HR before the EST, $r(49) = -.42, p < .01$, and METS, $r(50) = .53, p < .01$.

Correlation coefficients were computed among the three psychological variables and HR recovery for the 52 eligible participants. The results show that state anxiety scores were positively correlated with trait anxiety scores, $r(46) = .65, p < .01$, and trait anxiety scores were positively correlated with hostility scores, $r(50) = .34, p < .05$, both reaching statistical significance. There was no significant correlation between any of the three psychological variables and HR recovery.

Table 9

Correlation matrix among baseline demographic, EST and psychological measures for eligible participants (N=52)

Variable	1	2	3	4	5	6	7	8
1.Age	-							
2.S-Anxiety	-.05	-						
3.T-Anxiety	-.25	.65**	-					
4.HAS	-.06	.04	.34*	-				
5.HR recovery	-.35*	-.17	-.12	.02	-			
6.Supine HR	.17	.16	.13	.06	-.42**	-		
7.Peak HR	-.38**	-.03	-.08	.01	.50**	.22	-	
8.METS	-.55**	-.19	-.21	-.007	.62**	-.41**	.48**	-

Note: S-Anxiety, state anxiety; T-Anxiety, trait anxiety; HAS, Hostile Attitude Scale; HR, heart rate; METS, metabolic equivalents.
*$p < .05$; **$p < .01$

Characteristics of participants with high and low trait and state anxiety scores

To test the first objective, eligible participants were divided into groups based on high and low trait anxiety. Participants were categorized as having high trait anxiety or high state anxiety if they had a mean score on the STAI > 39 (Speilberger et al., 1983). Analysis of variance and chi square were used to examine differences between these two groups. Baseline demographic, clinical, and EST characteristics for high and low trait anxious participants can be found in Tables 10, 11, and 12.

Participants reporting higher trait anxiety were slightly younger, had a higher proportion of MIs and previous MIs, and were admitted to hospital with a diagnosis of angina more frequently. Trait anxious participants had higher state anxiety before the EST $F(1,50) = 30.12, p < .01$ with an effect size of $\eta_p^2 = 0.42$. A higher proportion were women, current and former smokers and had higher cholesterol levels. A smaller proportion of participants reporting high trait anxiety received PCI. Participants reporting higher trait anxiety achieved slightly fewer METS on the EST. Heart rate recovery was

the same between the two groups. Differences were not found to be significant on any of these variables except on state and trait anxiety scores.

Table 10

Frequency and percent of high and low trait anxious participants with baseline characteristics

Characteristics	High trait anxiety	Low trait anxiety	*p*
	Frequency (% of sample)		
No. of patients	18(35)	34(65)	
Female sex	5(28)	3(9)	
Health-related problems present			
high BP	9(50)	14(41)	0.54
high cholesterol	14(78)	22(65)	0.33
diabetes	2(11)	9(26)	0.20
asthma	5(28)	1(3)	0.008
arthritis	15(83)	18(53)	0.03
stomach	6(33)	8(24)	0.45
Current medication use			
β-blocker	17(94)	31(91)	0.67
anticoagulant	3(17)	6(18)	0.93
antiplatelet	18(100)	33(97)	0.46
lipid lowering	18(100	31(91)	0.19
ace inhibitor	14(78)	29(85)	0.50
anti-depressant	1(6)	2()	0.96
anti-anxiety	1(6)	-	
Admitting diagnosis			
MI	12(67)	20(59)	0.58
angiogram	17(94)	34(100)	0.17
PCI	11(61)	24(71)	0.49
CABG	3(17)	7(21)	0.73
bundle branch block	1(6)	3(89)	0.67
angina	4(22)	8(9)	0.92
Previous MI	2(11)	2(6)	0.50
Smoking status			
current smoker	3(17)	2(6)	0.45
former smoker	9(50)	17(50)	
never smoked	6(33)	14(41)	

Note: ACE, angiotensin converting enzyme; BP, blood pressure; CABG, coronary artery bypass graft; MI, myocardial infarction; PCI, percutaneous coronary intervention.

Table 11

Baseline characteristics of participants with high and low trait anxiety

Characteristics	High trait anxiety	Low trait anxiety	*p*	η_p^2
	Mean±SD			
Age (years)	56±10	62±11	0.09	0.06
Body fat (%)	27±7	25±4	0.31	0.02
Days from cardiac event to EST	48±30	59±29	0.24	0.03
Clinical chemistry (mmol/l)				
total cholesterol	4.14±1.00	3.75±0.92	0.17	0.04
triglycerides	1.97±1.20	1.39±0.92	0.06	0.07
HDL cholesterol	1.01±0.12	1.03±0.24	0.75	0.00
LDL cholesterol	2.18±0.58	2.10±0.76	0.72	0.00
Psychological variables				
state anxiety score	44±10(N=17)	30±7(N=31)	0.000	0.42
trait anxiety score	51±8(N=18)	30±5(N=34)	0.000	0.75
hostility score	62±21(N=18)	59±17(N=34)	0.56	0.01

Note: EST, exercise stress test; HDL, high-density lipoprotein; LDL, low-density lipoprotein, η_p^2, partial Eta squared.

Table 12

Exercise stress test characteristics for high and low trait anxious participants

Characteristics	High trait anxiety	Low trait anxiety	*p*	η_p^2
	Mean ± SD			
Test duration (min)	7:17±2:24	8:01±2:33	0.32	0.02
METS	8.3±2.9	9.2±3.3	0.35	0.02
Resting supine HR (bpm)	66±151	63±10	0.50	0.01
Supine SBP (mmHg)	120±19	120±21	0.93	0.00
Supine DBP (mmHg)	66±10	66±8	0.94	0.00
Pre-ex standing HR (bpm)	71±15	71±140	0.84	0.00
Peak HR (bpm)	127±17	126±23	0.92	0.00
% of maximum HR achieved	78±9	80±13	0.64	0.00
HR recovery (bpm)	19±9	19±12	0.99	0.00

Note: bpm, beats per minute; DBP, diastolic blood pressure; HR, heart rate; METS, metabolic equivalents; min, minutes; pre-ex, pre-exercise; SBP, systolic blood pressure; η_p^2, partial Eta squared.

State anxiety.

To test the first objective of the research, the high and low state anxiety groups were compared in terms of HR recovery. Individuals reporting higher state anxiety before the stress test had a lower EST time, achieved fewer METS and a lower peak HR on the test than those experiencing low state anxiety. Higher state anxious participants also had higher resting supine HRs and pre-exercise standing HRs and a poorer HR recovery than those reporting lower state anxiety. These differences were, however, not found to be significant.

Table 13

Exercise stress test characteristics for high and low state anxious participants

Characteristics	High state anxiety	Low state anxiety	*p*	η_p^2
	Mean ± SD			
Test duration (min)	6:59±2:38	8:09±2:22	0.12	0.05
METS	7.7±3.2	9.4±3.1	0.08	0.06
Resting supine HR (bpm)	68±15	62±10	0.12	0.05
Supine SBP (mmHg)	119±16	121±22	0.76	0.00
Supine DBP (mmHg)	64±8	67±9	0.36	0.02
Pre-ex standing HR (bpm)	75±17	69±12	0.17	0.05
Peak HR (bpm)	123±26	128±19	0.45	0.01
% of maximum HR achieved	77±13	80±11	0.41	0.01
HR recovery (bpm)	14±9	22±11	0.02	0.11

bpm, beats per minute; DBP, diastolic blood pressure; HR, heart rate; METS, metabolic equivalents; min, minutes; pre-ex, pre-exercise; SBP, systolic blood pressure; η_p^2, partial Eta squared.

Characteristics of participants with high and low hostility scores

To address the second objective, an examination of the differences between those with high hostility scores and those with low hostility scores was done using ANOVA and chi square. ANOVA revealed that individuals scoring higher on the HAS, had higher total cholesterol (4.64±1.00 mmol/l vs 3.75±0.89 mmol/l), $F(1,49) = 6.41$, $p = 0.02$ and LDL cholesterol (2.72±0.90 mmol/l vs 2.04±0.62 mmol/l), $F(1,47) = 6.30$, $p = 0.02$ than those reporting lower hostility scores with effect sizes of $\eta_p^2 = 0.12$ and $\eta_p^2 = 0.12$, respectively. Also, those reporting higher hostility scores, had higher trait anxiety scores (48±12 vs 35±11), $F(1,50) = 9.16$, $p = 0.004$ with an effect size of $\eta_p^2 = 0.15$. State anxiety scores were also higher, but not found to be significant. There was a higher combined proportion of participants that were current and former smokers (75% vs 57%). There were no other significant differences found between the two groups. These results can be found in Tables 14 and 15.

Table 14

Frequency and percent of baseline characteristics for high and low trait hostile participants

Characteristics	High trait hostility	Low trait hostility	*p*
	Frequency (% of sample)		
No. of patients	8(15)	44(85)	
Female sex	1(13)	7(16)	
Health-related problems present			
high BP	5(63)	18(41)	0.26
high cholesterol	6(75)	30(68)	0.70
diabetes	2(25)	9(20)	0.77
asthma	4(50)	2(5)	0.000
arthritis	6(75)	27(61)	0.46
stomach	2(25)	12(27)	0.89

Table 14 (continued)

Characteristics	High trait hostility	Low trait hostility	*p*
Current medication use			
β-blocker	8(100)	40(91)	0.38
antiplatelet	8(10)	43(98)	0.67
lipid lowering	6(75)	42(65)	0.38
ace inhibitor	5(63)	38(86)	0.10
anti-depressant	-	3(7)	0.45
anti-anxiety	-	1(2)	0.67
Admitting diagnoses			
MI	5(63)	27(61)	0.95
angiogram	8(100)	43(98)	0.67
PCI	4(50)	31(70)	0.26
CABG	2(25)	8(18)	0.65
bundle branch block	1(13)	3(7)	0.58
angina	1(13)	11(25)	0.44
Previous MI	1(13)	3(6)	0.58
Smoking status			
current smoker	1(13)	4(9)	0.67
former smoker	5(63)	21(48)	
never smoked	2(25)	18(41)	

ACE, angiotensin converting enzyme; BP, blood pressure; CABG, coronary artery bypass graft; EST, exercise stress test; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MI, myocardial infarction; PCI, percutaneous coronary intervention.

Table 15

Baseline characteristics of high and low trait hostile participants

Characteristics	High trait hostility	Low trait hostility	*p*	η_p^2
	Mean±SD			
Age (years)	64±12	59±10	0.29	0.02
Body fat (%)	27±5	25±5	0.40	0.02
Clinical chemistry (mmol/l)				
total cholesterol	4.64±1.00	3.75±0.89	0.02	0.12
triglycerides	1.91±1.18	1.53±1.04	0.36	0.02
HDL cholesterol	1.02±0.12	1.03±0.22	0.90	0.00
LDL cholesterol	2.72±0.90	2.04±0.62	0.02	0.12

Table 15 (continued)

Characteristics	High trait hostility	Low trait hostility	p	η_p^2
Psychological variables				
state anxiety score	40±7	34±11	0.15	0.04
trait anxiety score	48±12	35±11	0.004	0.15
hostility score	88±11	55±14	0.000	0.45

Note: EST, exercise stress test; HDL, high-density lipoprotein; LDL, low-density lipoprotein; η_p^2, partial Eta squared.

Participants having higher hostility scores had a higher supine resting HR, a lower peak HR, and a lower HR recovery after the EST than those having lower hostility scores. These differences were not found to be significant (Table 16).

Table 16

Exercise stress test characteristics for participants with high and low trait hostility

Characteristics	High trait hostility	Low trait hostility	p	η_p^2
	Mean ± SD			
Test duration (min)	7:11±1:25	7:52±2:39	0.48	0.01
METS	8.1±2.1	9.0±3.3	0.49	0.01
Resting supine HR (bpm)	68±10	63±13	0.34	0.02
Supine SBP (mmHg)	117±19	120±20	0.73	0.00
Supine DBP (mmHg)	66±9	66±9	0.89	0.00
Pre-ex standing HR (bpm)	75±10	70±15	0.49	0.01
Peak HR (bpm)	120±20	128±22	0.34	0.02
% of maximum HR achieved	76±10	80±12	0.41	0.01
HR recovery (bpm)	15±8	20±11	0.20	0.03

Note: bpm, beats per minute; DBP, diastolic blood pressure; HR, heart rate; METS, metabolic equivalents; min, minutes; pre-ex, pre-exercise; SBP, systolic blood pressure; η_p^2, partial Eta squared.

Characteristics of participants with a normal and abnormal HR recovery

To address the third objective of the study, eligible participants were divided into groups based on cut-off values for measures of HR recovery (normal HR recovery ≥12 bpm and abnormal HR recovery < 12 bpm) to assess differences in baseline clinical characteristics, EST values, and measures of anxiety and hostility. These cut-off values have been used in previous research (Cole et al. 1999). Participants with an abnormal HR recovery had a similar frequency of undesirable health conditions. Table 17 represents the frequency and percent of sample presenting with high levels of the designated health indices.

Significant differences were found on state anxiety scores, $F(1, 46) = 7.55$, $p = 0.01$, and hostility scores, $F(1,50) = 4.62$, $p = 0.04$, between the two groups when p was set at 0.05. Participants with an abnormal HR recovery had higher state anxiety scores, but lower hostility scores compared to participants with a normal HR recovery. There were no significant differences in trait anxiety scores between those with a normal HR recovery and those with an abnormal HR recovery (Table 18). Differences in men and women could not be examined because of the small sample size of women.

Table 17

Frequency and percent of baseline characteristics for participants with a normal and abnormal heart rate recovery

Characteristics	Normal HR recovery (≥12 bpm) N=38	Abnormal HR recovery (<12 bpm) N=14	*p*
	Frequency (% of sample)		
Female sex	5(13)	3(21)	
Health-related problems			
high BP	15(39)	8(57)	0.26
high cholesterol	25(66)	11(79)	0.38
diabetes	6(16)	5(36)	0.12
asthma	4(11)	2(14)	0.71
arthritis	23(61)	10(71)	0.47
stomach	11(29)	3(21)	0.59
Medication			
Number of medications			
β-blocker	34(89)	14(100)	0.21
anticoagulant	6(16)	3(21)	0.63
antiplatelet	38(100)	13(93)	0.10
lipid lowering	37(97)	12(86)	0.11
ace inhibitor	30(79)	13(93)	0.24
Admitting diagnosis			
MI	24(63)	8(57)	0.69
angiogram	37(97)	14(100)	0.54
PCI	27(71)	8(57)	0.34
CABG	6(16)	4(29)	0.30
Previous MI	3(8)	1(7)	0.93
Smoking status			
current smoker	5(13)	-	0.35
former smoker	18(47)	8(57)	
never smoked	15(39)	5(36)	

Note: ACE, angiotensin converting enzyme; BP, blood pressure; CABG, coronary artery bypass graft; EST, exercise stress test; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MI, myocardial infarction; PCI, percutaneous coronary intervention.

Table 18

Baseline characteristics of participants with a normal and abnormal HR recovery

Characteristics	Normal HR recovery (≥12 bpm)	Abnormal HR recovery (<12 bpm)	*p*	η_p^2
	Mean±SD			
Age (years)	59±10	64±12	0.12	0.05
Body fat (%)	25±4	27±6	0.33	0.02
Number of medications	6±2	7±3	0.20	0.03
Clinical chemistry (mmol/l)				
total cholesterol	3.96±0.96	3.69±0.97	0.38	0.02
triglycerides	1.59±1.07	1.60±1.05	0.99	0.00
HDL cholesterol	1.05±0.21	0.97±0.19	0.23	0.03
LDL cholesterol	2.22±0.76	1.92±0.44	0.21	0.03
Psychological variables				
state anxiety score	32±9	41±12	0.01	0.14
trait anxiety score	36±11	39±13	0.50	0.01
hostility score	63±17	51±19	0.04	0.08

Note: EST, exercise stress test; HDL, high-density lipoprotein; LDL, low-density lipoprotein; η_p^2, partial Eta squared.

Significant differences were found, however, between the two groups when examining EST characteristics (Table 19). Critical *p* values were adjusted using the Bonferroni approach ($p < 0.005$). Differences in METS, $F(1, 50) = 17.26$, $p < 0.01$, and standing pre-exercise HR, $F(1, 35) = 8.96$, $p = 0.005$ were significant with effect sizes of $\eta_p^2 = 0.26$ and $\eta_p^2 = 0.20$. Participants with an abnormal HR recovery achieved lower METS, and had a higher standing pre-exercise HR. Although not significant, participants with an abnormal HR recovery had higher resting supine heart rates before the EST and achieved fewer minutes and a lower peak HR on the EST.

Table 19

Exercise stress test characteristics of participants with a normal and abnormal heart rate recovery

Characteristics	Normal HR recovery (≥12 bpm) N=38	Abnormal HR recovery (<12 bpm) N=14	*p*	η_p^2
	Mean ± SD			
Test duration (min)	8:14±2:22	6:29±2:27	0.02	0.10
METS	9.8±2.7	6.2±2.8	0.000	0.26
Resting supine HR (bpm)	61±10	72±13	0.006	0.14
Supine SBP (mmHg)	120±20	120±19	0.93	0.00
Supine DBP (mmHg)	66±9	67±9	0.80	0.00
Pre-ex standing HR (bpm)	67±12	82±16	0.005	0.20
Peak HR (bpm)	130±18	115±27	0.03	0.10
% of maximum HR achieved	81±11	73±13	0.03	0.09
HR recovery (bpm)	24±8	7±3	0.000	0.53

bpm, beats per minute; DBP, diastolic blood pressure; HR, heart rate; METS, metabolic equivalents; min, minutes; pre-ex, pre-exercise; SBP, systolic blood pressure; η_p^2, partial Eta squared.

Discussion

Characteristics of all men and women

An initial analysis was done on baseline demographic, clinical and psychological variables examining differences between men and women (N=123). The group was then split into eligible and ineligible participants for further analysis.

Previous research has identified demographic, physiological, and psychological differences between men and women with heart disease. The results of ANOVA and chi square revealed that men and women attending the NACRP differed significantly on exercise performance and psychological characteristics.

Women in this sample were older than the men by about 7 years. The age difference found in this study is typical of findings from other CR programs that include men and women. The onset of CVD in women is about 10 years later than it is in men (Lerner & Kannel, 1986) which is generally thought to be due to the protective effects of estrogen (Stevenson, Crook, & Godsland, 1993). The prevalence of CVD in men between the ages of 45 and 65 is several times that of age-matched women (Dubey, Imthurn, Barton, & Jackson, 2005). However, after menopause these gender differences decrease and generally disappear by about 75 years of age (Dubey et al., 2005). Estrogen has been shown to increase serum HDL cholesterol in women (The Writing Group for the PEPI Trial, 1995) which may be why women in this sample were found to have higher HDL levels than men. Other co-morbidities often exist in women with CVD, generally due to their older age. In this sample, women only experienced higher incidences of arthritis compared to men. Men and women in this sample would also both be considered

overweight according to their BMI. A normal BMI would be between 18.5 and 24.9 kg/m^2.

Men in this study underwent significantly more CABG and PCI procedures than women. Previous research has shown that women undergo fewer non-invasive and invasive diagnostic and revascularization procedures than men (Shaw et al., 1994) despite the established benefits of these procedures in women (Lansky et al., 2005). However, women tend to experience more complications after CABG and PCI procedures than men, and this may deter physicians from referring women for these invasive procedures, thus accounting for the lower rates of CABG in women.

In regards to EST characteristics, women achieved fewer minutes and METS on the EST than men. Previous research looking at differences between men and women have found that women have lower exercise capacities which have included lower exercise tolerance (Cannistra, Balady, O'Malley, Weiner, & Ryan, 1992; O'Farrell, Murray, Huston, LeGrand, & Adamo, 2000), peak oxygen uptake and shorter time on the treadmill (Ades, Waldmann, McCann, & Weaver, 1992). Differences have been attributed to smaller muscle mass, lower capillary density, and lower oxidative potential in women (Reybrouck & Fagard, 1999), but an older age in this sample may also be a contributing factor.

Women had significantly higher state anxiety scores before the EST than men with trait anxiety scores being slightly higher but not significant. This is not surprising since the prevalence of anxiety in women is estimated to be 2 to 3 times higher in women than in men (Wittchen, Zhao, Kessler, & Eaton, 1994). Research has shown that women have higher anxiety then men during the early hospitalization phase for AMI (Kim et al.,

2000), throughout the preoperative and postoperative cardiac surgery phases (Duits et al., 1998; McCrone, Lenz, Tarzian, & Perkins, 2001) and even at 1 year post CABG surgery (Holahan, Moos, Holahan, & Brennan, 1995; Wiklund et al., 1993).

Men were found to have significantly higher hostility scores. This finding supports past research that has found that men experience higher levels of hostility than women (Frasure-Smith et al., 1995; Williams, Paton et al., 2000). In this sample, 42% of men were employed full-time at the time of their cardiac event compared to 16% of women. The work place may be an environment that fosters hostile tendencies. Research looking at how tension in the workplace can increase CVD risk has found that high work demand and low reward (Siegrist, Peter, Junge, Cremer, & Seidel, 1990) as well as low job control (Johnson, Stewart, Hall, Fredlund, & Theorell, 1996) predict cardiac events. The responses from the HAS ("I usually wonder about the motives of people around me"; "If you don't compete, you don't achieve anything in life today") may reflect a work environment that is highly competitive or has more pressure to succeed, creating conflict among co-workers. Overall, the group as a whole was not hostile compared to other studies that have used the HAS. The mean (60±18) was well below previous findings.

Characteristics of eligible and ineligible participants

It was important to look at the differences between participants who were eligible and ineligible for the study as there may have been some important clinical differences between the two groups. More than half of the participants in this sample were not eligible for the study. Unfortunately, many participants were excluded only because they did not return their questionnaire prior to their EST or they had an EST at a different

facility. About 21% of participants were excluded because they met the clinical exclusion criteria of this study.

Significant differences were found on EST characteristics for the eligible and ineligible participants. Ineligible participants achieved fewer minutes on the EST than the eligible participants. The group of ineligible participants included individuals with CHF, paced rhythms, atrial fibrillation, WPW, and recent valve surgery. These patients are often more sick and more deconditioned than patients that have had a MI without intervention, PCI or CABG. Therefore, it is not surprising to find that ineligible participants did not do as well on the EST as the eligible participants.

Relationships among demographic, exercise, and psychological variables for eligible participants

Analyses of the relationships among psychological variables revealed that individuals reporting higher scores on the trait anxiety scale had higher scores on the state anxiety scale before the EST. This positive correlation is consistent with the literature (Speilberger & Rickman, 1990). Speilberger & Rickman (1990) have found that individuals with high trait anxiety are more likely to perceive stressful situations as being personally dangerous or threatening and to respond to these situations with increased state anxiety. If a cardiac event occurred while exercising, trait anxious participants may have perceived the EST as a source of danger and responded with an increase in state anxiety.

Trait anxiety scores were also positively correlated with hostility scores. If we think about Smith's transactional hypothesis, that hostile individuals interact with their environment in ways that create interpersonal conflict resulting in more stressful life

experiences, the increased stressful life experiences may lead to increased anxiety. Anxiety may be brought on by being unable to have control over future events or situations such as their cardiac event or financial difficulties. Anxiety has been found to be highly correlated with other affective states such as depression and research has shown that psychological risk factors have a tendency to cluster together (Rozanski et al., 1999; Williams, Barefoot, & Schneiderman, 2003). The clustering of these psychological risk factors may explain the positive correlation between anxiety and hostility in this sample.

An examination of the relationship between HR recovery, demographic and other EST characteristics revealed that HR recovery was significantly related to age, supine HR, peak HR, and METS where there was a negative association with age and supine HR and a positive association with peak HR and METS. These relationships may be indicative of a better fitness level. A higher HR recovery has been found to be related to other EST characteristics such as a lower HR before exercise and higher peak exercise HRs. Individuals who were younger were able to achieve a higher workload and therefore a higher peak HR since HR increases linearly with workload. Generally, individuals who are more fit have a lower resting HR and a quicker HR recovery after exercise because of an increase in parasympathetic dominance due to habitual endurance exercise (McCardle, Katch, & Katch, p.343). This phenomenon is commonly seen in athletes. The negative association found between a resting supine HR and HR recovery in this population, may not necessarily be an indication of a higher fitness level in this sample, but may be a result of medication. Medications, such as β-blockers, are prescribed to cardiac patients to decrease the HR and the amount of work the heart has to do. Many cardiac patients tend to be older and lead sedentary lifestyles both which contribute to poor functional

capacity indicated by fewer METs on the EST and a lower peak HR. Instability on the treadmill or co-morbidities such as arthritis may also lead to premature cessation of the EST resulting in fewer METs.

An examination of the relationship between the psychological variables and HR recovery indicated that there was no significant relationship between state anxiety, trait anxiety or hostility with HR recovery. However, there was a trend for participants reporting higher state and trait anxiety scores to have achieved fewer minutes on the EST, a lower peak HR and to have a higher supine resting HR as well as a low HR recovery. Increases in supine resting HR and BP may be in response to increased state anxiety before the EST.

Characteristics of participants with trait anxiety

Although there were no significant findings between those reporting high trait anxiety scores and those with low anxiety scores, some interesting findings were found that are worth discussing. The first objective of this study was to assess the relationship between trait anxiety and HR recovery. Results of the ANOVA revealed that there were no significant differences in HR recovery values between those with high trait anxiety and those with low anxiety and neither group had a HR recovery value that would indicate an impaired autonomic function (e.g., < 12 bpm). This finding is in contrast to other studies that have found that anxiety does affect the balance of the ANS (Kawachi et al., 1995; Watkins et al., 1998). Differences in findings between this study and others may be attributed to the use of different measures of autonomic function and trait anxiety with other studies using HRV analysis or BRS. Also, HR recovery values may not have differed between the two groups because high and low trait anxious participants achieved

similar results on the EST (similar METS and peak HR values). It would be expected that HR recovery values would be similar between groups that achieved similar peak HRs since HR recovery is affected by the intensity of the exercise (Imai et al.,1994).

There were a higher proportion of women with high trait anxiety scores. As mentioned previously, women suffer from anxiety more often than men. The higher trait anxiety in this sample of women may be due to a lack of social support. Women in CR programs are often widowed and therefore lack the support of not only a significant other, but potentially extended family. They may have lost the support they once received from co-workers once they retired. A lack of social support has been shown to be associated with an increase in anxiety in patients with chronic disease (Sherbourne & Hays, 1990). Women of this age often have difficulty with transportation and finances which may increase their level of anxiety. Despite more women having high trait anxiety, participants reporting higher anxiety were found to be younger. Previous research has found that a younger age in men and women recovering from heart surgery predicted increased anxiety more than gender (McCrone et al., 2001). Older individuals may be more prepared to cope with chronic diseases because they expect them with increasing age. Younger individuals, however, are not expecting to experience a cardiac event because they are younger and it may be this incongruence that increases their anxiety during the recovery process.

It is interesting that those reporting higher trait anxiety scores were admitted to hospital due to angina and MI more often than those with lower anxiety scores. A higher proportion of participants with high anxiety had also experienced a prior MI. Previous research has found that anxiety increases the risk of future cardiac complications in

cardiac patients which may include arrhythmias (Moser & Dracup, 1996) or re-infarction (Frasure-Smith, Lesperance, & Talajic, 1995). It has also been shown that acute negative emotional states, such as anger, anxiety or frustration are associated with myocardial ischemia (Gullete et al., 1997). It may be that those with higher trait anxiety scores were reacting to daily life events with more state anxiety inducing myocardial ischemia and potentially leading to a recurrent cardiac event.

The slightly lower EST times and METS achieved may indicate that participants with higher anxiety were more sedentary prior to their cardiac event and from the time of their cardiac event until they entered CR. Sullivan, LaCroix, Baum, Grothaus, and Katon (1997) found that cardiac patients who reported higher anxiety had lower levels of physical functioning which included having difficulty with basic and intermediate activities of daily living. Participants with higher anxiety may have feared that exercise would precipitate another cardiac event and, therefore, remained sedentary in order to prevent that from occurring. Participants reporting higher trait anxiety also reported higher state anxiety before the EST and slightly higher hostility scores. These findings are congruent with the literature which has found that psychological factors tend to cluster in individuals and in groups (Williams, Barefoot, et al., 2003).

State anxiety.

High anxiety before the EST was associated with a poorer result on the EST where participants with higher state anxiety achieved fewer METs and a lower peak HR on the EST. They also had higher resting supine heart rates, pre-exercise heart rates and a lower HR recovery. These findings were not found to be significant, but the finding that state anxiety, not trait anxiety, was associated with a lower HR recovery is interesting

because previous research has also found that recent emotional stress was found to be associated with blunted cardiac-vagal modulation of HRV independent of the individuals level of trait anxiety or physical fitness (Dishman et al., 2000). It may be that state anxiety or recent stressful life events upset the balance between the PNS and the SNS more than trait anxiety or hostility.

Characteristics of participants with hostility

The second objective of this study was to determine if there was an association between high hostility and HR recovery. Participants reporting higher hostility had a lower HR recovery than those with lower hostility scores, but ANOVA revealed the finding was not significant. Although the HR recovery value was lower, the participants would not be categorized as having an abnormal HR recovery with a HR recovery value of 14 bpm. It still may demonstrate that hostile people have poorer autonomic function than participants with low hostility. The lower HR recovery may be a result of participants reaching a lower maximum HR during the EST whereby the HR does not have to decrease by many beats within the first minute to return to baseline.

Participants with high hostility scores in this sample had significantly higher total cholesterol and triglyceride levels. A higher proportion of hostile participants were also current or former smokers (75% vs. 58%) and had a higher percentage of body fat (27% vs 25%) compared to low hostile participants. Previous studies have found that hostility is positively associated with waist-to-hip ratio, BMI, hypertension (Yan et al., 2003), total cholesterol level, increased triglycerides, and high fasting glucose levels (Lavie & Milani, 2005). It has also been shown that hostile persons are more likely to smoke, have poor nutritional habits, be overweight, and be inactive (Siegler, Peterson, Barefoot, &

Williams, 1992; Raikkonen & Keltikangas-Jarvinen, 1991). It seems that the unhealthy lifestyle behaviours (e.g., smoking) may be contributing to the poor clinical profile (e.g., high cholesterol) in this sample.

Overall, it appears that participants with higher hostility scores did not do as well on the EST achieving fewer METS, having higher resting supine heart rates, and a lower peak HR. They did, however, have slightly lower supine BP than those with low hostility. It might be expected that hostile participants would also display a higher BP in this situation. Previous research has shown that hostile people have elevated ambulatory blood pressure during daily life (Suarez & Blumenthal, 1991) and exhibit elevated blood pressure and HR response to annoying circumstances presented in laboratory settings (Sul & Wan, 1993). It is difficult to explain why hostile participants in this sample had a lower BP, but may be a result of the different physicians measuring BP.

The finding that individuals with higher hostility scores also had higher trait anxiety is congruent with the literature and may be attributed to the clustering of psychological risk factors.

The small number of participants in the high hostility group may be an indication that those that are more hostile are not referred to CR programs or choose not to attend. It is well known that cardiac patients with depression are less likely to attend CR programs (Lane, Carroll, Ring, Beevers, & Lip, 2001) and this phenomenon may be occurring in hostile patients as well. At the time of referral, patients may have become confrontational with the health care professional and the physician may have chosen not to refer the patient onto the CR program. Other possible explanations could be that hostile

individuals succumbed to the health consequences of hostility or they may have been more likely to decline to participate in the research study.

Characteristics of participants with a normal and an abnormal HR recovery

Objective 3 was to investigate the differences between individuals who had a normal HR recovery compared to those with an abnormal HR recovery. Results of the ANOVA identified significant differences on EST characteristics and psychological variables. Participants with an abnormal HR recovery achieved significantly fewer METS, and had significantly higher resting supine and standing heart rates before the EST. Theses clinical indicators have been shown to independently increase the risk of mortality (Snader et al., 1997; Palatini, Casiglia, Julius, & Pessina, 1999). Research has shown that individuals who achieve fewer than 5.0 METS on an EST double their risk of death compared to individuals who achieve more than 8.0 METS (Myers et al., 2002). Although participants with an abnormal HR recovery achieved fewer METS, they achieved greater than 5.0 METS on the EST with a mean of 6.2 ±2.8 METS.

Participants with an abnormal HR recovery were also found to have an overall adverse clinical profile. These participants were slightly older, were taking more medications, had lower HDL, higher BMI, and underwent more CABG surgery than PCI procedures. Previous research has found that individuals with an abnormal HR recovery generally have a poorer clinical profile (Cole et al.,1999; Nishime et al., 2000).

Results of the ANOVA revealed that participants with an abnormal HR recovery had significantly higher state anxiety scores and significantly lower hostility scores. Although it may appear that the participants with an abnormal HR recovery had lower hostility scores, the hostility score for both groups would not actually categorize either

group as having high hostility and these scores may have been a result of splitting the group on the HR recovery variable. The previous correlations identified that there was no relationship between hostility scores and HR recovery. Higher state anxiety may be contributing to the higher resting supine and standing heart rates in participants with an abnormal HR recovery similar to what was found in participants with high hostility.

General Discussion

Anxiety and hostility.

Over the last decade, work on psychological risk factors has identified that these risk factors do not work independently to adversely affect health, but tend to cluster together in the same individual and in the same group of people such as individuals of low socioeconomic status (SES) (Williams, Barefoot et al., 2003). This study also found anxiety and hostility to be highly correlated with each other in this sample of cardiac patients, but neither one was associated with an abnormal HR recovery as hypothesized. It was not possible to assess whether anxiety and hostility together would be associated with an impaired HR recovery because of the small sample size.

Participants with higher levels of state and trait anxiety and hostility displayed a more adverse clinical profile. The clinical profile included higher cholesterol levels and lower HDL, and a greater likelihood of being a current or former smoker, more medications, increased resting supine and pre-exercise heart rates, and lower METS achieved. Previous research has identified that individuals with psychological risk factors for CVD tend to engage in unhealthy behaviours such as smoking, poor nutritional habits, and sedentary behaviour (Siegler et al., 1992; Raikkonen et al., 1991). The poor clinical

profile found in the high hostility and high anxiety groups may reflect participation in these unhealthy behaviours.

Heart rate recovery.

Heart rate recovery has not previously been used to assess the influence of psychological factors on autonomic function and, therefore, further discussion is provided to try and explain the null findings between anxiety, hostility and HR recovery.

As described previously, the amount that the HR will decrease after exercise depends on the intensity of the exercise or the peak HR achieved (Imai et al., 1994). Although all efforts were made to push patients to volitional exhaustion, patients in this setting were not accustomed to graded exercise and may have stopped prematurely due to muscular fatigue, balance problems on the treadmill, fear of overexertion or poor motivation. The EST was also administered by several different physicians who may have had an influence as to when the patient stopped. Patient and physician factors may have influenced the peak HR achieved during the EST.

Heart rate recovery is also influenced by numerous variables including age, medications, high BP, diabetes, and physical inactivity. The sample size was not large enough to control for all of these variables, so it is difficult to know which factors might have influenced parasympathetic function in these cardiac patients.

Heart rate recovery may not be a sensitive enough measure to pick up the effects of emotions on autonomic balance. Studies investigating how psychological factors influence autonomic balance have used HRV analysis which has the capability to determine which part of the ANS is being affected. Heart rate recovery is thought to be more of an estimate of parasympathetic function rather than a direct measure because it is

likely mediated by both parasympathetic reactivation and sympathetic withdrawl (Kannankeril et al., 2004).

The number of ineligible participants for the study due to clinical exclusion criteria raises the question about using HR recovery in a cardiac population. Previous epidemiological research in this area has established that an abnormal HR recovery increases the risk of mortality in asymptomatic individuals, but limited studies have been performed in populations that already demonstrate autonomic dysfunction. Despite the lack of studies assessing HR recovery in patients with autonomic dysfunction, it may not be a question of how useful HR recovery is in these patients, but perhaps the focus should move towards how to increase HR recovery or parasympathetic function in these individuals since they are at a known increased risk of mortality.

There are, however, many unanswered questions about HR recovery including the best way to incorporate HR recovery into the risk stratification and management of cardiac patients, the usefulness of using HR recovery in select populations such as heart failure patients and the differences in cut-off values for patients taking and not taking β-blocking medication (Gibbons, 2002). Researchers have begun to examine these questions, but the unknowns about HR recovery have lead researchers to recommend that HR recovery be used in conjunction with other valuable information from the EST such as exercise capacity, chronotropic incompetence and the Duke treadmill score when risk stratifying cardiac patients. This will provide a more complete clinical profile of the patient and help determine the most appropriate intervention for each individual.

Limitations

There are important limitations in the present study that need to be addressed. The small sample size of eligible participants significantly limited the power of this study. It reduced the ability to examine differences between men and women because only 15% of the sample were women. However, this percentage is typical of women attending CR programs in Canada. This also limited the power to analyze differences within each of the three groups. There was limited ability to control for factors known to affect autonomic function such as age, fitness level, and diabetes for example.

This sample is taken from a single CR program in which patients have been referred and volunteered to participate in the study. It is known that individuals who are depressed, hostile or suffer from anxiety are less likely to attend CR programs and therefore the scores on the STAI or HAS might have been biased towards the lower range of scores. Participants may have modified their responses on the STAI or HAS to appear more socially desirable.

The measures used to assess anxiety, hostility and autonomic function differ from other studies. Differences in findings between studies have commonly been attributed to differences in how anxiety and hostility are measured. However, the STAI has been used previously in cardiac patients and in research assessing how anxiety affects autonomic function (e.g., Watkins et al., 1998). The Cook-Medley Hostility scale has most often been used to assess hostility in research but assesses the behavioural component of hostility which is thought to be a situational response. The HAS was selected because it measures the cognitive aspect of hostility and is considerably shorter which eliminated patient burden. The HAS has been used in cardiac populations and is valid and reliable.

Most studies assessing the influence of psychological factors on autonomic function have used HRV analysis not HR recovery. The use of different measures makes it difficult to compare the results from this study to results of other studies.

Future Directions

Research needs to continue to examine the relationship between psychological factors and cardiac health and to investigate the pathophysiological mechanisms as to how psychological factors affect health. It would be a benefit to take SES into consideration as SES has been shown to influence health. Research has shown that a lower education level is associated with higher levels of chronic stress which may disrupt the balance of the ANS (Shishehbor et al., 2002). Education level may be a mediating factor between psychological factors such as anxiety, depression, or hostility and risk of CVD.

It would be advantageous to assess autonomic function with multiple methods, such as resting HR, peak HR during exercise, HR recovery after exercise and HRV analysis to have a better overall assessment of autonomic function. Studies looking at how HRV and HR recovery are correlated might also be advantageous.

Although there was no association between the psychological variables and HR recovery in this study, it is still important to look at ways of improving HR recovery and psychological variables since they all adversely affect health. Cardiac rehabilitation programs offer psychological counseling and management as well as structured exercise which have proven to be successful in decreasing anxiety, hostility and depression in cardiac patients (Lavie, & Milani, 2004; Lavie, & Milani, 2005). A recent study by Tiukinhoy, Beohar, and Hsie (2003) demonstrated that exercise training in CR was

associated with improvement in HR recovery from baseline to the follow-up EST. Techniques such as relaxation training and slowed respiration have also been shown to increase parasympathetic function (Sakakibara, & Hayano, 1996; Sakakibara, Takeuchi, & Hayano, 1994). It may also be important to include interventions to individuals prior to their EST since it was found that participants reporting higher state anxiety had a poorer result on the EST. A major link in participants being able to receive the numerous benefits of CR programs is the referral process. Health care professionals need to continue to refer all patients to CR programs if one exists in their area.

Conclusions

In an attempt to identify the physiological mechanisms as to how psychological factors affect health, researchers have examined the influence of psychological factors on autonomic function. It is well known that individual psychological factors such as anxiety, hostility and depression as well as autonomic imbalance increase one's risk of CVD or recurrent cardiovascular events. It has also been shown that individuals with anxiety, hostility and depression display autonomic imbalance. Although findings from this study did not demonstrate autonomic imbalance in individuals with anxiety or hostility, it does demonstrate the importance of taking into consideration the complete clinical and psychological profile of the patient, given that these variables appeared to be associated with a poorer clinical profile.

This study demonstrated that anxiety and hostility existed in patients attending CR and that these psychological factors were highly correlated. Previous research has identified that psychological variables tend to cluster together rather than existing independently (Williams, Barefoot et al., 2003). Participants with high state anxiety, trait

anxiety or hostility did not display an impaired HR recovery as hypothesized, however, but they did display a more adverse overall clinical profile.

Heart rate recovery was also highly correlated with other exercise test characteristics and participants displaying an abnormal HR recovery also had a more adverse clinical profile as hypothesized. It will be important to provide interventions such as exercise, not only to improve HR recovery, but functional capacity as well. This will improve the prognosis of the patient.

Because of the small sample size in this study, it is difficult to say if HR recovery is a suitable measure to investigate the effects of emotions or personality characteristics on autonomic function. Further studies are needed to identify the relationship between these variables.

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Appendix A

STAI - Trait

A number of statements which people have used to describe themselves are given below. Read each statement and circle the appropriate number to the right of the statement to indicate how you **generally** feel.

		Almost never	**Some-times**	**Often**	**Almost always**
1.	I feel pleasant	1	2	3	4
2.	I feel nervous and restless	1	2	3	4
3.	I feel satisfied with myself	1	2	3	4
4.	I wish I could be as happy as others seem to be	1	2	3	4
5.	I feel like a failure	1	2	3	4
6.	I feel rested	1	2	3	4
7.	I am "calm, cool, and collected"	1	2	3	4
8.	I feel that difficulties are piling up so that I cannot overcome them	1	2	3	4
9.	I worry too much over something that doesn't really matter	1	2	3	4
10.	I am happy	1	2	3	4
11.	I have disturbing thoughts	1	2	3	4
12.	I lack self-confidence	1	2	3	4
13.	I feel secure	1	2	3	4
14.	I make decisions easily	1	2	3	4
15.	I feel inadequate	1	2	3	4
16.	I am content	1	2	3	4
17.	Some unimportant thoughts run through my mind and bothers me	1	2	3	4
18.	I take disappointments so keenly that I can't put them out of my mind	1	2	3	4
19.	I am a steady person	1	2	3	4
20.	I get in a state of tension or turmoil as I think over my recent concerns and interests	1	2	3	4

Appendix B

STAI – State

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you feel **right** now, **at this moment**. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems best to describe your present feelings best.

		Not at all	Some-what	Moderately	Very much so
1.	I feel calm	1	2	3	4
2.	I feel secure	1	2	3	4
3.	I am tense	1	2	3	4
4.	I feel strained	1	2	3	4
5.	I feel at ease	1	2	3	4
6.	I feel pretty upset	1	2	3	4
7.	I am presently worrying over possible misfortunes	1	2	3	4
8.	I feel satisfied	1	2	3	4
9.	I feel frightened	1	2	3	4
10.	I feel comfortable	1	2	3	4
11.	I feel self-confident	1	2	3	4
12.	I feel nervous	1	2	3	4
13.	I am jittery	1	2	3	4
14.	I feel indecisive	1	2	3	4
15.	I am relaxed	1	2	3	4
16.	I feel content	1	2	3	4
17.	I am worried	1	2	3	4
18.	I feel confused	1	2	3	4
19.	I feel steady	1	2	3	4
20.	I feel pleasant	1	2	3	4

Appendix C

Hostile Attitude Scale

We are interested in your thoughts about the statements that follow below. Please read each statement and then circle the number, from 1 to 7, which best represents your position. Please use the scale below to guide your responses to the next set of questions.

1	2	3	4	5	6	7
Strongly disagree		Mildly disagree		Mildly agree		Strongly agree

1.	I really like an argument with people who think they know it all.	1	2	3	4	5	6	7
2.	Since I am usually right, I consider it a duty to point out people's errors to them.	1	2	3	4	5	6	7
3.	Other people get opportunities that never seem to come my way.	1	2	3	4	5	6	7
4.	I would never purposely lose an argument with another person	1	2	3	4	5	6	7
5.	I tend to compare myself with others who are doing better than I am.	1	2	3	4	5	6	7
6.	I believe that I deserve more out of life than I have.	1	2	3	4	5	6	7
7.	If you don't compete, you don't achieve anything in life today.	1	2	3	4	5	6	7
8.	I can't help being a little unpleasant with people I don't like.	1	2	3	4	5	6	7
9.	I am always entitled to give my opinion to things.	1	2	3	4	5	6	7
10.	I usually wonder about the motives of people around me.	1	2	3	4	5	6	7
11.	When someone is bossy, I often think about doing the opposite of what he or she wants.	1	2	3	4	5	6	7
12.	I have frequently met people who were supposed to be experts but who didn't impress me.	1	2	3	4	5	6	7
13.	I am against people in authority who like to demonstrate their power.	1	2	3	4	5	6	7
14.	When I play, I play to win.	1	2	3	4	5	6	7
15.	You can't trust people to do what they say they will do.	1	2	3	4	5	6	7
16.	Rules were made to be broken.	1	2	3	4	5	6	7
17.	Often I do not receive the kind of treatment or service that I deserve.	1	2	3	4	5	6	7
18.	I like to make sure that nobody really gets to know me.	1	2	3	4	5	6	7