Summer 2012

A Comparison of Relaxation Techniques on Blood Pressure Reactivity and Recovery Assessing the Moderating Effect of Anger Coping Style

Catherine J. Mills
Old Dominion University

A COMPARISION OF RELAXATION TECHNIQUES ON BLOOD PRESSURE REACTIVITY AND RECOVERY ASSESSING THE MODERATING EFFECT OF ANGER COPING STYLE

by

Catherine J. Mills
B.A. December 2002, The University of North Carolina at Greensboro
M.A. May 2008, North Carolina Central University

A Dissertation Submitted to the Faculties of The College of William and Mary, Eastern Virginia Medical School, Norfolk State University, Old Dominion University in Partial Fulfillment of the Requirements for the Degree of

DOCTOR OF PSYCHOLOGY

CLINICAL PSYCHOLOGY

VIRGINIA CONSORTIUM PROGRAM IN CLINICAL PSYCHOLOGY
August 2012

Approved by:

Serina A. Neumann (Director)
Eastern Virginia Medical School

Richard Handel (Member)
Eastern Virginia Medical School

Karen Y. Holmes (Member)
Norfolk State University

Leonard Holmes (Member)
Eastern Virginia Medical School

Robin Lewis (Member)
Old Dominion University

Karen B. White (Member)
Norfolk State University

ABSTRACT

A COMPARISON OF RELAXATION TECHNIQUES ON BLOOD PRESSURE REACTIVITY AND RECOVERY ASSESSING THE MODERATING EFFECTS OF ANGER COPING STYLE

Catherine J. Mills
Old Dominion University, 2012
Director: Dr. Serina A. Neumann

This study examined the relationship of anger coping style and relaxation techniques on cardiovascular reactivity and recovery in blood pressure (BP). Eighty-nine students from Eastern Virginia Medical School in Norfolk, Virginia participated. The participants were instructed to rest for ten minutes (baseline), complete a math task with harassment for 6 minutes (stressor), and engage in one of three recovery conditions (a standard control (SC), diaphragmatic breathing (DB), or mantra recitation (MR) without breathing instructions) for 10 minutes. The Spielberger State-Trait Anger Expression Inventory (STAXI) was utilized to measure trait anger coping style. Participants were compensated $25. It was hypothesized that DB would show the greatest reduction in BP during the recovery period and those individuals with high Anger-In or Anger-Out trait coping style scores would exhibit greater cardiovascular reactivity and slower cardiovascular recovery. It was also hypothesized that high cardiovascular reactivity would be associated with high baseline BP and anger coping style would moderate the effect of relaxation techniques on BP.

A planned (apriori) simple contrast revealed a significant effect for DB on diastolic blood pressure (DBP) during 10 minutes of recovery, $F\ (1, 85) = 6.11, p < .05$, such that DB demonstrated the greatest reduction in DBP in comparison to the SC and MR. Greater physiological responses to stress were not associated with higher baseline BP; baseline BP was

not significantly related to BP reactivity for baseline systolic blood pressure (SBP) and SBP reactivity, $r = .20$, *ns*, for baseline SBP and DBP reactivity, $r = .13$, *ns*, for baseline DBP and DBP reactivity, $r = .03$, *ns*, or baseline DBP and SBP reactivity $r = .16$, *ns*. There were no significant results found to indicate an interaction or main effect of trait anger coping style by recovery condition on recovery BP. Given the significant result for DB's effectiveness at reducing DBP after a stressor, this information may be useful when treating hypertensive patients. Relaxation techniques should be considered an adjunctive treatment for high BP along with hypertensive medication as they can be cost effective at reducing BP.

This dissertation is dedicated to my mother, DeJuanna W. Mills.

TABLE OF CONTENTS

Page

LIST OF TABLES

Table Page

LIST OF FIGURES

CHAPTER I

INTRODUCTION

The necessity to conduct a study concerning alternative treatments for hypertension, like relaxation techniques, becomes apparent when one looks at the prevalence of hypertension in the United States. An estimated 80,000,000 American adults (one in three) have one or more types of cardiovascular disease (CVD). High blood pressure (HBP) is one of the most common forms of CVD, with an estimated 73,600,000 Americans diagnosed with HBP, 35,300,000 males and 38,300,000 females (American Heart Association, 2009).

The current study had four objectives. The first objective was to determine if relaxation techniques can be effective at reducing blood pressure (BP) following stress (i.e. serial subtraction with harassment). The second objective was to determine if there is a certain relaxation technique that is superior or more effective at reducing BP following stress than another (specifically, diaphragmatic breathing (DB) versus mantra recitation (MR) without breathing instructions). The third objective of the present study was to evaluate if a person's trait anger coping style (holding anger in or excessively expressing one's anger) is related to BP at rest, during stress, and during a recovery period from stress. The fourth and final aim was to test whether greater cardiovascular reactors (individuals with higher cardiovascular reactivity during the math task) will have higher resting BP.

The rationale for examining the interrelations discussed above is predicated on the hypothesis that cardiovascular reactivity and prolonged stress responses play a central

role in mediating the association among psychosocial factors such as anger and cardiovascular morbidity and mortality. It is hypothesized that a prolonged stress response causes exaggerated sympathetic nervous system activation that may over time lead to hypertension. This is a result of the sustained constriction of the veins and arteries causing an increase in the pressure of the blood vessels. This process is one element involved in the "fight or flight response". The present study will investigate the effectiveness of relaxation techniques to counter this response. Furthermore, the current study will attempt to reveal the moderating role of anger coping style on BP changes during and following stress which potentially may increase the risk for hypertension. The results of this study may illuminate possible underlying causes of hypertension.

The following sections will introduce the concept and societal impact of CVD in general. Then, there will be a select review of the literature on the pathophysiology of anger coping style as it relates to CVD, followed by a select literature review of studies regarding cardiovascular reactivity and recovery, relaxation techniques, and the influence of these variables on BP. Additionally, the concept of anger will be defined and the role of anger coping style in cardiovascular dysregulation will be reviewed. Finally, there will be a description of this study's methodology, data reduction and data analyses, results, a discussion of the results, conclusions, and suggested future directions.

Cardiovascular Disease

HBP is one of the most prevalent forms of CVD; however, it is important to note there are other forms of CVD commonly found in the United States population. For example, Coronary Heart Disease (CHD) includes heart attack and angina pectoris or chest pain. Atherosclerosis occurs when fat, cholesterol, and other substances build up in

the walls of the arteries and form hard substances called plaque (Gandelman, 2006). Plaque deposits can also break loose forming blood clots that are a common cause of heart attack (myocardial infarction) and stroke. A stroke occurs when a blood vessel that supplies oxygen to a part of the brain is blocked. The affected part of the brain cannot function and neither can the part of the body it controls. A stroke can also occur when a blood vessel supplying blood to a part of the brain ruptures (American Heart Association, 2007). The prevalence of acquiring one of these diseases significantly increases when an individual has hypertension.

Hypertension Defined

Hypertension is the term used to describe HBP. BP readings, measured in millimeters of mercury (mm Hg), are usually written as two numbers in a fraction (Vander, Sherman, & Luciano, 1998). The top number is the systolic blood pressure (SBP), the pressure created when your heart beats. SBP is considered high if it is consistently over 140 mm Hg. The bottom number is the diastolic blood pressure (DBP). The definition of DBP is the pressure inside the blood vessels when the heart is at rest. When DBP is over 90 mm Hg, it is considered high (American Heart Association, 2009; Vander, Sherman, Luciano, 1998). Normal BP is defined as a reading of 120/80 mm Hg. A person is considered to have HBP if they have untreated systolic pressure of 140 mm Hg or higher, diastolic pressure of 90 mm Hg or higher, are taking antihypertensive medicine, or have been told at least twice by a physician or other health professional that they have HBP (American Heart Association, 2009). The definition of HBP has changed in recent years with the introduction of a new term, "prehypertension". A person can be

diagnosed as having prehypertension if they have SBP between 120 and 139 mm Hg or a DBP reading between 80 and 89 mm Hg on multiple readings (Chobanian et al., 2003).

In 2003, the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of HBP released its seventh report (Chobanian et al., 2003). The report cited previous studies that found an increased lifetime risk of developing hypertension and increased risk of cardiovascular complications associated with levels of BP previously considered in the normal range. The purpose of the new classification is to identify individuals in whom early intervention by adoption of healthy lifestyles could reduce BP, decrease the rate of progression of BP to hypertensive levels with age, or prevent hypertension entirely (Chobanian et al., 2003). Prehypertension, not considered a disease category per se, is a designation chosen to identify individuals at high risk of developing hypertension, so that both patients and clinicians are alerted to this risk and encouraged to intervene and prevent or delay the disease from developing (Chobanian et al., 2003).

The Societal Impact of High Blood Pressure

HBP is a major burden to American society; approximately 30% of adults are unaware of their hypertension. Undiagnosed, untreated, and uncontrolled hypertension places a substantial strain on the health care system as well as on the health on the individual (Chobanian, 2003). For instance, HBP is a chief risk factor for heart disease, stroke, congestive heart failure, and kidney disease (Centers for Disease Control [CDC], 2011). According to the CDC (2011), in 2010 HBP cost the United States 76.6 billion dollars in health care services, medications, and missed days of work. Additionally, about one in four American adults have prehypertension that increases their risk for a later diagnosis of HBP (CDC, 2011). Eight percent of hypertensive individuals are

undiagnosed (CDC, 2012). There are usually no symptoms of HBP, as a result HBP is referred to as the silent killer (CDC, 2012).

The cause of 90–95% of the cases of HBP is unknown (American Heart Association, 2008). The term essential hypertension is used to describe elevated BP levels with an unknown cause. HBP that results from a specific condition, habit, or medication is referred to as secondary hypertension (Medline Plus, 2010). Dangerously high HBP is called "malignant hypertension." Symptoms of malignant hypertension include chest pain, confusion, ear noise or buzzing, irregular heartbeat, nosebleeds, tiredness, and vision changes. Having a family history of HBP and being of certain ethnic backgrounds (for example, being an African American) put one at increased risk of developing HBP (Medline Plus, 2010).

Ethnicity and High Blood Pressure

There is a racial disparity in the prevalence of HBP, especially among African Americans or Blacks in the United States. The prevalence of HBP in African Americans in the United States is among the highest in the world and is steadily increasing. From 1988-1994 to 1999-2002, the prevalence of HBP increased from 35.8% to 41.4% among African American adults, and it was particularly high among African American women (44.0%). Prevalence among Caucasians also increased, from 24.3 % to 28.1 % (American Heart Association, 2009). Compared with Caucasians, African Americans develop HBP earlier in life and their average BPs are much higher. As a result, compared with Caucasians, African Americans have a 1.3 times greater rate of nonfatal stroke, a 1.8 times greater rate of fatal stroke, a 1.5 times greater rate of death from heart disease, and a 4.2 times greater rate of end-stage renal disease (American Heart Association, 2009).

Among African Americans, those with the highest rates of HBP are more likely to be middle-aged or older, less educated, overweight or obese, physically inactive, and to have diabetes (American Heart Association, 2009). Some studies suggest that Hispanic Americans have rates of HBP that are similar to or lower than those of non-Hispanic White Americans. According to the National Health Interview Survey (NHIS) studies from 2000 to 2002, Black Hispanics were at slightly greater risk of HBP than white Hispanics (American Heart Association, 2009). The following prevalence estimates are for people age 18 and older:

Table 1
Prevalence Estimates for People Age 18 and Older in the United States in 2007

Race (Ethnicity)	Heart Disease	Coronary Heart Disease	Hypertension	Stroke
Whites or Caucasians	11.4%	6.1%	22.2%	6.0%
Blacks or African Americans	10.2%	6.0%	31.7%	13.7%
Hispanics of Latinos	8.8%	5.7%	20.6%	3.7%
Asians	6.9%	4.3%	19.5%	2.6%
Native Hawaiians or Pacific Islanders	Not Available	Not Available	28.5%	Not Available
American Indians or Alaska Natives	10.5%	5.6%	25.5%	Not Available

Note. This information is adapted from Heart Disease and Stroke Statistics-2009 Update, American Heart Association.

Mortality

Previous evidence has demonstrated that there are millions of Americans living with HBP. Unfortunately, there are also a number of Americans who succumb to this disease. According to the American Heart Association (2009), in 2005, HBP mortality was 57,356 (24,056 males and 33,310 females). The overall death rate from HBP was 18.4. Death rates were 15.8 for Caucasian males, 52.1 for African American males, 15.1 for Caucasian females, and 40.3 for African American females (American Heart Association, 2009). Additionally, about 69% of people who have a first heart attack, 77% who have a first stroke, and 74% with congestive heart failure have BP higher than 140/90 mm Hg (American Heart Association, 2009).

Data from the National Heart, Lung, and Blood Institute's Framingham Heart Study indicate that HBP is associated with shorter overall life expectancy as well as shorter life expectancy free of CVD and more years lived with CVD (American Heart Association, 2009). At age 50, total life expectancy is 5.1 years longer for men with normal BP, and 4.9 years longer for women with normal BP, than in those with hypertension (American Heart Association, 2009).

The estimated direct and indirect cost of HBP for 2009 was 73.4 billion dollars. End-Stage Renal Disease (also called end-stage kidney disease) (ESRD) is a condition that is most commonly associated with diabetes and/or HBP, and occurs when the kidneys can no longer function normally on their own. The incidence of reported ESRD has increased about 40% in the past 10 years. CVD is the leading cause of death for those with ESRD, and CVD mortality is five to thirty times higher in dialysis patients than in individuals from the general population. In every year since 1900, except 1918, CVD

accounted for more deaths than any other single cause or group of causes of death in the United States (American Heart Association, 2009).

How is Hypertension Usually Treated?

The previous information exemplifies the fact that hypertension is a serious disease and is the cause of many deaths. In order to prevent more individuals from suffering from and dying from hypertension, the usual method of treatment may need to be reevaluated. Hypertension is usually treated with medications and/or recommended life style changes. According to the CDC, 68% of hypertensives were treated with antihypertensive medication (Ostchega et al., 2008). Some of the life style changes a doctor may prescribe are: eating a healthy diet, starting an exercise regime, maintaining healthy weight, and managing stress or learning to cope with stress. BP medications work in various ways to lower BP. Some remove extra fluid and salt from the body to lower BP. Others slow down the heartbeat or relax and widen blood vessels.

Why Relaxation Techniques?

Relaxation techniques may have some of the same effects as the medications usually prescribed to reduce BP by causing the blood vessels and muscles in the arteries and veins to relax via a general relaxation response. If the body is relaxed, the amount of norepinephrine produced during the stress response is greatly reduced. Norepinephrine can act as a drug increasing BP by increasing vascular tone through α-adrenergic receptor activation. The general relaxation response will cause the muscles of the veins and arteries to relax along with the other muscles in the body.

Research has shown that interventions that reduce the magnitude of cardiovascular responses to stress are justified, at least in part, by the notion that

exaggerated responses to stress can damage the cardiovascular system (Carroll et al., 2011; Chafin, Roy, Gerin, & Christenfeld, 2004; Neumann et al., 2004). As a means of better understanding the processes by which psychological stressors may influence cardiovascular functioning, there have been efforts to find techniques that limit or reduce the magnitude of the stress response (Chafin et al., 2004). Relaxation techniques have been demonstrated to produce reductions in BP, possibly by reducing the overall stress response by countering this response via a generalized relaxation response (Agras et al., 1982; Bruning & Frew, 1987; Grossman et al., 2001; Lee et al., 2003; Lehnert et al., 1987; Parker et al., 1978; Wallace et al., 1983).

Cardiovascular Reactivity

The exaggerated stress response referred to in the previous section is one aspect of cardiovascular reactivity. Cardiovascular reactivity can be defined as "how aroused people become by particular stressors" (Christenfeld, Glynn, & Gerin, 2000; p. 543), or reactivity can be defined as the magnitude of cardiovascular arousal occurring during acute stress exposure (Linden et al., 1997; Trivedi et al., 2008). In other words, cardiovascular reactivity is a change in cardiovascular responses as a result of psychological experiences. Further, cardiovascular reactivity to stress is typically defined in terms of a change or gain score (i.e. delta), calculated as the difference between measurements during stress and baseline rest (Kelsey et al., 2007). The reactivity hypothesis maintains that exaggerated BP and heart rate (HR) (number of heart beats each minute) responses to stress can damage the cardiovascular system (Manuck et al., 1990). Thus, people who exhibit large cardiovascular responses are at risk for the development of CVD and hypertension (Chafin et al., 2004). Following the

aforementioned exaggerated cardiovascular response is a period when an individual will need to recover from the responses. Cardiovascular reactivity and recovery in tandem may be contributing to the prevalence of CVD.

Cardiovascular Recovery

Cardiovascular recovery is defined as how much time it takes people after a stressor to return to resting levels (Christenfeld, Glynn, & Gerin, 2000). For any cardiovascular parameter, recovery can be defined as either the time required to return to pre-task baseline levels after termination of a stressor, or the degree of elevation above pre-task levels within a predetermined post-task interval (Stewart & France, 2001). While the precise nature of the relationship between cardiovascular recovery and later hypertension development is currently unclear, researchers have proposed that prolonged elevations in cardiovascular activity following stress may be a marker of chronic sympathetic activation which, over time, may result in down-regulation of cardiac and vascular adrenergic receptors (Stewart & France, 2001). Evidence suggests that heightened cardiovascular reactivity to stress and delayed cardiovascular recovery from stress may be important risk factors for hypertension (Bleil et al., 2008; Christenfeld, Glynn, & Gerin, 2000; Manuck et al., 1990; Stewart & France, 2001; Trivedi et al., 2008; Vella & Friedman, 2009). The mechanism behind the maladaptive process of heightened cardiovascular reactivity to stress and delayed cardiovascular recovery from stress may be better understood with a review of the fight or flight response.

Pathophysiology of Anger and Cardiovascular Disease

The fight or flight response, also called the acute stress response, was first described by Walter Cannon in 1914 (Lambert & Kinsley, 2005). This theory states that,

when an animal feels threatened or stressed, the sympathetic nervous system will respond priming the animal to fight or flee the threatening situation. For some individuals the experience and expression of anger is the "stress" that starts the fight or flight response. If these individuals experience anger for prolonged periods of time, their bodies are constantly in the fight or flight mode. It is important to note that there is research revealing that certain types of anger expression, specifically constructive anger verbal behavior, can be adaptive (Davidson, MacGregor, Stuhr, & Gidron, 1999). The current study will focus on maladaptive anger coping styles, the over-suppression of anger and the over-expression of anger.

In the maladaptive experience of anger, psychologically the individual becomes angry; whether they express or suppress this anger, it stresses the body. Individuals can have cardiovascular responses to psychological experiences including stress experienced as a result of a life threatening event or intense emotions such as anger. The brain is the central organ of the stress response and determines what is stressful, as well as the behavioral and physiological responses to potential and actual stressors (McEwen, 2008). For some individuals, the experience of anger is physiologically perceived as stress which activates the hypothalamus. The hypothalamus is located just above the pituitary gland in the brain and is the overall control and integration center of the autonomic nervous system (Van De Graaff, 2000). The hypothalamus functions using motor fibers connected to the brain stem of the spinal cord and the pituitary gland and releases various hormones. The hypothalamus activates the adrenal medulla that produces epinephrine (also known as adrenaline) and norepinephrine. The adrenal cortex is also activated which leads to the release of cortisol. The adrenal medulla and the adrenal cortex are

parts of the adrenal glands that are paired organs that cap the superior borders of the kidneys (Van De Graaff, 2000). They are embedded against the muscles of the back in a productive pad of fat. Each gland consists of the outer adrenal cortex and inner adrenal medulla that function as separate glands (Van De Graaff, 2000). The hormones these structures release are epinephrine, norephinephrine, and cortisol which function to mobilize fuels, including lipids and other body fats during a stress response. Other effects of the activation of the sympathetic nervous system include increased HR, metabolism, respiration, and BP. The increased BP results from the body's vasoconstriction of the veins and arteries which if experienced over prolonged periods of time may lead to hypertension. The response of increased BP is the focus of the present study. Additionally, there is a decrease in digestion and reproductive activity, which in combination facilitate the body's ability to survive a threat (Lambert & Kinsley, 2005).

The adaptive value of the increased sympathetic activity in a physically threatening fight-or-flight situation is obvious. Nevertheless, what purpose does it serve in the psychological stressors so common to modern life, when neither fight nor flight is appropriate (Vander, Sherman, & Luciano, 1998)? The current study attempts to add information supporting that this fight-or-flight reaction, if prolonged, will enhance the development of CVD, through the lasting effects of extended cardiovascular reactivity, specifically increased BP.

The current study added to the mounting body of literature that focuses on using relaxation techniques as an alternative or adjunctive form of treatment for HBP, hypothesizing that relaxation treatment may be a remedy, at least in part, for the HBP

disparity in America. This study also examined the possible moderating role of anger coping style in the BP-relaxation technique interaction.

Research Questions

The current study answered five questions:

1. Can relaxation techniques be effective at reducing BP during a recovery period following a stressful event?
2. Is there one relaxation technique that is superior to the other (specifically, MR versus DB) at reducing BP during a recovery period?
3. How is anger coping style related to BP?
4. Is anger coping style a moderator of resting BP, the reactivity of BP during a stressful event, and during post-stress responses? Does anger coping style interact with the relaxation techniques on the BP recovery responses?
5. Will higher cardiovascular responders have higher mean baseline BPs?

CHAPTER II

REVIEW OF LITERATURE

Historic Development of the Mind/Body Connection

The concept that strong negative emotions and the stressors encountered throughout the course of normal life processes can adversely affect one's health is not a new notion. This conception has existed since biblical times during which people referred to "dying of grief." In "Charmides" by Plato, Socrates stated, "Let no one persuade you to cure his headache until he has first given his soul to be cured, for this is the great error of our day in the treatment of the human body, that physicians separate the soul from the body" (Siegman, 1994, p. 3).

Although there was a rise of modern scientific medicine during the Renaissance period (which spanned from the 14th through the 17th centuries), there were still those who continued to acknowledge the role of psychological factors in physical disease (Siegman, 1994). For example, William Harvey (1628), known as the father of cardiovascular physiology, wrote "A mental disturbance provoking pain, excessive joy, hope or anxiety extend to the heart, where it affects its temper, and rate, impairing general nutrition and vigor" (Siegman, 1994, p. 4). Additionally, Dr. J. Archer, a noted 17th Century physician, wrote: "The observations I have made in the practice of physicks these several years has confirmed me in this opinion, that the origin or cause of most men and women's sickness, disease, and death is first some great discontent which brings a habit of sadness of the mind" (Siegman, 1994, p. 4).

In the 20th and 21st centuries, the anger-health concept evolved into numerous research studies to support the idea that psychological constructs can affect a person's physiological health (Engebretson & Stoney, 1995; Finney, Stoney, and Engebretson, 2002; Johnson & Broman, 1987; Richards, Alvarenga, & Hof, 2000; Suinn, 2001). Emphasizing much attention on the theory of Type A Behavior Pattern or Type A personality, the expression of anger and hostility was weighed as a major coronary-prone component directly associated with CVD. The consensus concerning Type A personality and CVD was that adverse health effects resulted from the over expression (Anger Out) or the under expression (Anger In) of angry feelings.

Anger Defined

Anger is a phenomenon that has received much attention from trying to precisely define it to investigating the physiological affects the body experiences during bouts of anger. Defined by Merriam-Webster Online Dictionary (2009) anger is a strong feeling of displeasure and usually of antagonism; or rage. Spielberger and colleagues (1988) define anger as a term for an emotion with corresponding feelings and physiological arousal closely associated with hostility and aggression. These three constructs (anger, hostility, and aggression) together have been termed the "AHA! Syndrome," studied extensively by Spielberger (Spielberger et al., 1988; Spielberger et al., 1995).

The working definitions Spielberger and his associates (1988) used in the development of the anger scales of the State-Trait Anger Expression Inventory (STAXI), utilized in the present study, were as follows: Anger-In was defined on the basis of how often an individual experiences but does not express angry feelings, rather than in terms of the psychoanalytic construct of anger turned against the ego. Anger-Out was defined in

terms of the frequency that an individual engages in aggressive behavior when motivated by angry feelings (Spielberger et al., 1988, 1995). Anger-Out may be expressed in physical or motor behavior acts such as slamming doors or assaulting others, or in verbal behavior such as in the form of criticisms, threats, insults, or extreme use of profanity. These physical and verbal manifestations of Anger-Out may be directed toward the source of provocation or frustration, or they may be expressed indirectly toward individuals or objects associated with, or symbolic of, the provoking agent (Spielberger et al., 1995). The STAXI also differentiates State Anger (current experience) from Trait Anger (more permanent personality traits).

Anger and General Health

There have been various studies assessing the association of emotions such as fear, depression, anxiety, and anger on various medical disorders that utilized the STAXI (Spielberger et al., 1985). Studies concerning anger and its association with various morbidities such as hypertension (Johnson & Spielberger, 1992), headaches (Venable et al., 2001), the experience and reported severity of pain (Bruehl et al., 2006; Suinn, 2001), anorexia nervosa (Geller et al., 2000), acne (Rapp et al., 2004), and immune responsiveness (Suinn, 2001) have greatly increased our understanding of anger as an emotional condition that is hazardous to health.

Anger and Blood Pressure

For many years, researchers have suspected that anger, conceptualized as a personality factor, is associated with the development of hypertension (Schwartz et al., 1981; Siegman, 1994; Sinha et al., 1992). Three variables of anger along with BP were assessed by Goldstein, Edelberg, Meier, and Davis (1988) using 45 non-medicated

participants with a mean age of 50.2 years. Using the Survey of Affective Stress scale, a seven item questionnaire that asked the subjects to indicate on analog scales the frequency and intensity of experienced affects at work and in home environments (Goldstein et al., 1988). The three variables assessed included Experienced Anger, the product of the rating the subject gave to frequency of anger experienced at work and to the intensity of anger experienced at work plus that experienced at home [work frequency x intensity + home frequency x intensity]; Expressed Anger, the sum of the rating the subject gave to the likelihood others would be aware of their anger at home plus whether others would be aware of their anger at work; and Family Expressed Anger, a rating of the subjects perception of the expression of anger in their families when they were growing up (Goldstein et al., 1988).

Goldstein and associates (1988) found that Expressed Anger was inversely associated with SBP and DBP, while Family Expressed Anger showed this inverse relationship with SBP only (Goldstein et al., 1988). No relation was found for HR and the anger variables (Experienced and Expressed Anger). Although Experienced Anger was not related to the cardiovascular measures, it was significantly associated with Expressed Anger and Family Expressed Anger. The researchers suggested that the inverse relationship between Expressed Anger and BP provides further support for the hypothesis that suppression of anger plays a major role in the tonic [marked by prolonged muscular contraction] elevation of BP (Goldstein et al., 1988). Interestingly, the suppression of anger expression was found to be associated with DBP elevations but not with increased HR (Goldstein et al., 1988).

Attempting to find a relation between psychological variables and hypertension,

Friedman, Schwartz, Schnall, Landsbergis, Pieper, Gerin and Pickering (2001) utilized a sample of 283 men between the ages of 30 and 60 assessing both ambulatory and casual (or clinic) BP measures. However, these researchers failed to find a relationship between anger and BP. A number of psychosocial questionnaires were utilized including the STAXI, the Trait Anxiety, and the Spielberger Anger Expression scale. No psychological variable distinguished between normotensive and mildly hypertensive participants. Additionally, neither casual nor ambulatory diastolic hypertension was associated with a pattern of psychological variables different from that of men with normal pressures (Friedman et al., 2001). The researchers gave a number of possible explanations for their lack of significant findings including assessing the wrong psychological variables, using the wrong cut off for BP to distinguish hypertensives from non-hypertensives, using a mostly white sample, and the lack of using a clinical sample of hypertensives (Friedman et al., 2001).

As evidenced by the two previously reviewed studies, there is inconsistency in the results of studies investigating anger and BP. Some studies show changes in DBP as a result of the experience of anger and some show changes in SBP. In an attempt to find some consistency in the studies relating anger and BP, some researchers have conducted meta-analyses. One such meta-analysis was conducted by Schum, Jorgensen, Verhaeghen, Sauro and Thibodeau (2003) who reviewed fifteen studies published between the years 1993 and 2002, assessing the relationship of trait anger and ambulatory BP. Overall, a significant positive correlation between the experience of anger and SBP was found. However, there was no relationship found for anger and DBP.

Anger and Cardiovascular Reactivity

The studies in the previous section provide evidence that the experience of anger and trait coping style to deal with the anger can affect cardiovascular responses which can include cardiovascular reactivity. Carroll, Phillips, Der, Hunt, and Benzeval (2011) conducted a study that examined the reactivity hypothesis. These researchers collected data at three time points, during the initial baseline (completed in 1987), 5 years later, and their current study which reports results of analyses conducted 12-years after the original baseline. There were 1196 participants involved in the study (73% of the participants for whom baseline cardiovascular reactivity data was available); 645 (55%) women and 551 (45%) men (Carroll et al., 2011). The researchers examined whether BP reactions to acute mental stress predicted future resting BP levels, as well as the temporal drift in resting BP (Carroll et al., 2011).

The acute stress task used by Carroll and associates (2011) was the Paced Auditory Serial Addition Test (PASAT), in which the participant added sequential number pairs while, at the same time, retained the second pair in memory to add to the next number presented. The answers were given orally by the participant and the series of single-digit numbers were presented by audiotape (Carroll et al., 2011). The results of the Carroll and associates (2001) study revealed that SBP reactivity was positively correlated with resting SBP 12 years later, the greater the SBP reactivity, the higher the subsequent resting SBP (Carroll et al., 2011). There was no such correlation between DBP reactivity and subsequent resting DBP (Carroll et al., 2011). SBP reactivity positively predicted risk for hypertension and DBP reactivity did not significantly predict hypertension 12 years later (Carroll et al., 2011).

The influence of stress as experienced by various emotional states, including anger, on cardiovascular reactivity was found to be of interest to researchers. Schwartz, Weinberger, and Singer (1981) conducted a fascinating study assessing the differences in cardiovascular reactivity following experiences of various emotions: anger, happiness, sadness, fear, and relaxation via imagery. The participants included 32 healthy college students with previous acting experience in either high school or college. This criterion was chosen to increase the likelihood that subjects would be relatively comfortable in the otherwise novel task of publicly self-generating emotional states (Schwartz et al., 1981). Each subject participated in six trials, one for each condition, which was counterbalanced across subjects. The imagery conditions differentially affected patterns of cardiovascular activity. For DBP, the mean increase during anger was significantly higher than during any of the five other conditions. Twenty-five of the thirty- two subjects had greater increases in DBP during anger than during the other emotion conditions. Mean arterial pressure was also higher in anger than with the other emotions. The BP increases associated with fear were similar to all of the other emotions, except during relaxation. For SBP, anger produced the highest mean increase, but it was not significantly different from fear, sadness, or happiness (Schwartz et al., 1981). Further, the statistical analyses performed (comparison of means, regression weights, and discriminate functions) highlighted anger as the opposite of relaxation (Schwartz et al., 1981).

Subsequent to the aforementioned study a similar study was conducted by Sinha, Lovallo, and Parsons (1992), in which the researchers examined the differential patterns of cardiovascular reactivity during various emotions, also including joy, sadness, fear, and anger. The cardiovascular measures evaluated in this study were HR, BP, stroke

volume (the volume of blood pumped from one ventricle of the heart with each beat), peripheral vascular resistance (vascular resistance to the flow of blood in peripheral arterial vessels that is the function of the internal vessel diameter), cardiac output (amount of blood pumped by the left ventricle each minute), and indices of myocardial contractility. The indirect measures of myocardial contractility were pre-ejection period (PEP) (the time interval from the beginning of electrical stimulation of the ventricles to the opening of the aortic valve) and left ventricular ejection time (LVET) (the time interval from opening to closing of the aortic valve), assessed using impedance cardiogram (Sinha et al., 1992).

The participants included twenty-seven males between 21 and 35 years of age. The participants created personalized emotion scripts that were screened for their ability to evoke relatively pure emotions. The participants were also screened for imagery ability. Participants received prerecorded relaxation instructions and after the relaxation, the participant engaged in seven imagery trials interspersed by two neutral imagery conditions. The order of the imagery trials was randomized.

After comparing the various emotional states, anger showed the largest effects on the cardiovascular system (Sinha et al., 1992). Increased DBP in anger was associated with maintained levels of peripheral vascular resistance and increased cardiac output and HR compared with changes during neutral imagery (Sinha et al., 1992). Sadness produced moderate increases in BP and vascular resistance and a decrease in cardiac output compared with changes in neutral imagery. Additionally, fear, action, and joy produced similar BP changes in which SBP increased and DBP was relatively unchanged. The measurement of cardiac output and determination of vascular resistance

changes during emotional imagery demonstrate that emotion-specific BP responses are produced by underlying patterns of cardiovascular activation, which differ between the major categories of emotions (Sinha et al., 1992).

The Sinha and associates (1992) and Schwartz and colleagues (1981) studies both compared the influences of various emotions on cardiovascular reactivity and consistently found that anger showed pronounced cardiovascular reactivity. There are studies that focus primarily on the influence of negative emotions, i.e. anger and cardiovascular reactivity. In one such study, Engebretson, Matthews, and Scheier (1989) tested the hypotheses that the influence of anger expression style on psychophysiological responses would be limited to subjects exposed to anger instigation (i.e. a harassing confederate), and that the specific pattern of psychophysiological responses would vary as a function of whether subjects were induced to use their preferred versus non-preferred mode of anger expression styles in response to the instigation. These researchers tested these hypotheses by evaluating seventy-eight male college students who worked on a task with either a harassing (annoying) or a pleasant confederate. The participants then wrote either a negative or a positive evaluation of the confederate in which they interacted. Data were collected on the participant's preferred anger coping style (Anger in or Anger out) and BP and HR were evaluated throughout the experiment.

The results of the Engebretson and associates (1989) study revealed that the subjects who were harassed had greater SBP, DBP, and HR responses than did the non-harassed subjects. Additionally, subjects who characteristically express anger outwardly produced larger SBP responses during the study than did subjects who characteristically hold their anger in. Furthermore, Newman-Keuls post hoc tests indicated that among

harassed participants, acting in a manner consistent with one's usual anger expression style (i.e. anger-in style subjects writing a positive evaluation and anger-out style subjects writing a negative evaluation) led to significant reductions in SBP (Engebretson et al., 1989). Additionally, the more negative the confederate was perceived to be, the greater the magnitude of SBP changes during the experiment and evaluation task (Engebretson et al., 1989).

Neumann, Brown, Waldstein, and Katzel (2006) tested whether a 6-month walking program (aerobic exercise) would attenuate cardiovascular reactivity to anger-provoking stressors in sedentary older adults who manifest exercise-induced myocardial ischemia (SI) and had no history of coronary artery disease (CAD). SI is myocardial ischemia not accompanied by angina and is associated with increased risk for subsequent cardiac events in middle-aged and older individuals. Twenty-five adults (17 men and 8 women; age 56-83 years) were selected because they were at increased risk for future coronary events, but were free of potential confounders (i.e. physiological effects of drug therapy for cardiac symptoms, symptomatic myocardial ischemia, and prior myocardial infarction) (Neumann et al., 2006). The participants were randomized to 6 months of aerobic-exercise intervention (n = 14) or 6 months of wait-list control (n = 11) as they entered the study (staggered enrollment).

The experiment included a cardiovascular reactivity session in which four measurements of DBP, SBP, and HR data were collected every minute. The participants performed three 3-minute tasks, each followed by a 10 minute recovery period. The tasks were as follows: 1) a personally relevant anger-recall task, in which participants were asked to recall and speak about an event that occurred within the preceding year that

made them feel angry, irritated, upset, or frustrated; 2) a standardized role-play scenario designed to elicit anger and hostility by asking the participant to discuss the inadequate care of someone to whom the participant is close (e.g. spouse, parent, or sibling) by a hostile and difficult nursing-home administrator; and 3) mental arithmetic consisting of standardized subtraction of serial 7s with minor levels of harassment (i.e. participants were firmly urged to complete the calculations quickly and accurately complete the task), similar to the current study. The tasks were presented in fixed order (Neumann & Waldstein, 2001; Neumann et al., 2006). There was also an exercise training condition with a goal to increase the participants' VO_{2max} (maximal aerobic capacity) by 10% or more, without changing their weight (Neumann et al., 2006). The participants exercised by walking on a treadmill three times per week for 6 months.

The results of the Neumann and colleagues (2006) study revealed significant increases in SBP, DBP, and HR responses from baseline to task across the three anger inducing task conditions during the first time period. Results also showed a significant interaction effect of Treatment Group × Time on DBP reactivity. Specifically, reductions in DBP reactivity were noted for the exercise group after aerobic conditioning relative to the control group. Overall, this study revealed that anger inducing tasks (a stressor) can result in increased cardiovascular responses.

Anger and Cardiovascular Recovery

The previous section contained studies that reviewed cardiovascular responses during a stressor in relation to anger and anger expression. This next section reviews studies involving how anger and anger expression affect BP following stress, some involving harassment as in the procedure for the current study. Faber and Burns (1996)

conducted a research study assessing the degree to which individuals' expressed anger during harassment would mediate relationships between anger management style and cardiovascular recovery from harassment. The researchers also examined gender differences in anger management style and cardiovascular recovery from harassment. To accomplish their goals, Faber and Burns used a within-subjects design and had the subjects tell stories in response to Thematic Apperception Test (TAT) cards under harassment or non-harassment conditions. The baseline rate of angry words spoken by the participant was assessed during the non-harassment phase. The degree to which words reflecting angry affect increased from the non-harassment to harassment phases was inferred to represent a reaction to anger instigation (Faber & Burns, 1996).

The sample consisted of 31 men and 32 women (age range = 18-30, M = 20.0) (Faber & Burns, 1996). The baseline BP was obtained while the participant sat quietly for five minutes. The testing period, in which the participant told stories based on the TAT cards (with either harassment or non-harassment), was followed by a 5-minute recovery period during which the subject relaxed and BP was measured again (Faber & Burns, 1996). The dependent measures in the Faber and Burns study were SBP, DBP, and HR. Anger management style was assessed using the Anger-Expression Inventory by Spielberger (1985); the anger in and anger out subscales only. Expressed anger was assessed via audiotaped responses to the TAT, coded by two raters. Expressed anger was defined in terms of two kinds of words which were uttered by subjects when telling their stories: words connoting aggressive behavior (e.g. hit, kill, push) and words reflecting angry or hostile affect (e.g. angry, irritated, stupid) (Faber & Burns, 1996). Two distinct

variables were revealed, expressed aggressive behavior (EAB) and expressed angry/hostile affect (EAHA) (Faber & Burns, 1996).

The results indicated that for SBP only, an overall effect for gender emerged, with men showing higher SBP across the baseline, non-harassment, harassment, and recovery phases (Faber & Burns, 1996). SBP and DBP increased significantly from baseline to harassment. HR increased significantly from baseline to non-harassment phase but HR did not increase additionally from non-harassment to the harassment phase. A gender main effect emerged for the EAHA ratio only, which indicated that women used a higher ratio of angry/hostile affect words to total words than men across the non-harassment and harassment phases (Faber & Burns, 1996). Overall, the degree of expressed anger increased when the experimenter became critical of subjects' stories, and this effect was similar for men and women, indicated by an increase in angry words (Faber & Burns, 1996).

The results also indicated that the higher the endorsement of an anger-out anger management style by men and women, the greater was the increase in the expression of words connoting angry/hostile affect from non-harassment to harassment (Faber & Burns, 1996). Additionally, anger out subscale scores were positively associated with SBP changes during harassment (Faber & Burns, 1996). Additionally, the results suggest that both men and women high on anger-out showed the highest SBP elevations during harassment, but that during early and middle phases of recovery, men high on anger-out and women low on anger-out showed levels of SBP sustained above baseline levels or slower SBP recovery (Faber & Burns, 1996). EABCH (EAB and EAHA residualized

change scores) X gender interactions were found for DBP during the recovery period (Faber & Burns, 1996). No significant effects were found for HR during recovery.

Lai and Linden (1992) conducted another study assessing cardiovascular recovery. The stressor utilized in this study was serial subtraction with repeated interruptions and harassment to provoke anger, extending Hokanson's research, which showed that for men anger release after provocation, tends to accelerate cardiovascular recovery (Lai & Linden, 1992). Lai and Linden (1992) tested male and female undergraduate students; 105 subjects (56 females and 49 males) with a mean age was 19 years ($SD = 3$).

The Spielberger Anger Expression Scale was utilized to measure the students' usual way of coping with their anger (anger-in vs. anger-out). A double criterion (i.e. self-report and peer evaluation) was utilized to assess the subjects true method of handling their anger. In addition to the participants rating their usual mode of anger expression, a package was sent to their peers (i.e. roommate, family member, etc.) who assessed the participant's usual mode of anger expression. Only subjects whose self-reported was consistent with their peer's reported opinion of their anger expression style were allowed to participate in the study.

The baseline (adaptation) phase lasted for 15 minutes, during which the participant completed the state portion of the Spielberger State-Trait Anger Scale, and unrelated questionnaires including the Pennebaker's Inventory and the Cognitive portion of the Schwartz Cognitive and Somatic Anxiety Questionnaire (Lai & Linden, 1992). The unrelated questionnaires were used in the study to conceal the true purpose of the study and were not involved in the data analysis. At the end of the math task, the subjects who

were randomly assigned to an opportunity for emotional release condition (N =53) completed a written evaluation for 10 minutes (this was the recovery period). The written instruction required the participant to answer the evaluation in an open and frank manner to improve the design of the study, and they were told that the experimenter would not suffer undesirable consequences (Lai & Linden, 1992). The writing task (or recovery phase) lasted 10 minutes.

HR, SBP, and DBP were measured throughout the experiment. These measurements were assessed every 2 minutes during the adaptation phase and at minutes 3, 7, and 11 (i.e. 1 minute after each harassment) during the experimental provocation phase, and at minutes 1, 5, and 9 of the recovery phase (Lai & Linden, 1992). The results of the Lai and Linden (1992) study revealed no gender differences for baseline HR, DBP, or pre-test state anger ratings (Lai & Linden, 1992). Men showed consistently greater cardiovascular task levels than women for HR, SBP, and DBP. There was no main effect for anger expression style or gender x style interaction for either SBP or DBP, but on HR, there was a trend for a gender x anger style interaction ($p = 0.06$) (Lai & Linden, 1992). Anger in men showed greater HR responses than anger-out men. Additionally, regarding the effectiveness of the experimental manipulation on state anger, a main effect for the period variable was revealed such that increases in state anger from pre-test to post-test were observed. Females given the opportunity for anger expression were angrier at the end of recovery than females not given the opportunity to reveal their anger (Lai & Linden, 1992). In regards to the effectiveness of the written evaluation as a tool for anger expression, there were no main or interaction effects suggesting that all subjects, irrespective of gender and or anger expression style, expressed the same amount of

negative emotion when asked to evaluate the experimenter (Lai & Linden, 1992). Anger-in women reached lower levels of SBP after 10 minutes of recovery than anger-out women (Lai & Linden, 1992). For men, significant effects were noted for HR and on DBP (Lai & Linden, 1992). Men with an opportunity to release anger recovered better on HR than men without an opportunity and men with an opportunity to release anger also showed a similar trend towards faster DBP recovery. Anger-in and anger-out did not affect recovery rates of BP or HR, and no interactions were noted (Lai & Linden, 1992). Men's recovery was affected by the situational manipulation allowing anger release but not by the trait-type anger-in/out distinction; whereas the recovery of women was only affected by the anger-in/anger-out distinction and not by the absence or presence of a situational anger release opportunity (Lai & Linden, 1992).

Evaluating whether laboratory-based BP recovery predicts ambulatory BP (ABP), Trivedi, Sherwood, Strauman, and Blumenthal (2008) postulated that the recovery phase of the stress response is an individual difference characteristic that may predict cardiovascular risk (Trivedi et al., 2008). One hundred and eighty-two employed and healthy adults completed the laboratory and ABP assessments on separate days. The baseline period lasted for 20 minutes, followed by the alternating presentation of four stressors (which were balanced using a Williams' Square Design), and a 10-minute recovery period. The tasks/stressors included an anger recall interview in which the participants had 3 minutes to describe an interpersonal situation which made them angry during the previous week. There was also a reaction time shock avoidance task in which the participants were presented with a loud audible tone presented at varying, unpredictable intervals over a 3 minute period. In the reaction time task, participants were

required to press a key as fast as they could on hearing each tone and were instructed that if the reaction time was considered too slow, a "painful but harmless" electric shock would be delivered immediately by electrodes previously applied to the leg (shocks were never delivered) (Trivedi et al., 2008). There was also a foot cold pressor task in which participants placed one foot in a bucket of ice and water (0-4^0 C) for 2 minutes. The fourth task was a Mirror Trace task in which participants had 3 minutes to outline a star while viewing its reflection in a mirror, as many times as possible, while making a minimum of errors. If the participant deviated from the star, an aversive buzzer was activated and a counter which recorded the number of errors was also activated (Trivedi et al., 2008). During the ABP assessment, participants wore an ABP monitor for 24 hours during a typical workday.

BP recovery was found to account for significant additional variance for daytime SBP, nighttime SBP, daytime DBP, and nighttime DBP, after controlling for baseline and reactivity BP (Trivedi et al., 2008). The study found that post-stress BP recovery was an independent predictor of real-life BP, measured according to daytime and nighttime ABP (SBP and DBP), after controlling for resting BP and BP reactivity (Trivedi et al., 2008). These results suggested that persistence of the BP response following stress maybe a more salient characteristic of the stress response in understanding its potential impact on longer-term cardiovascular regulation (Trivedi et al., 2008). It is noteworthy that the combination of reactivity and recovery accounted for greater variance in the researchers' statistical models predicting ABP, compared with either one individually (Trivedi et al., 2008). As a result, research studies that assess both cardiovascular reactivity and recovery

may provide more beneficial information regarding the anger-CVD relation than studies that investigate either one separately.

Dorr, Brosschot, Sollers, and Thayer (2007) also examined the Hokanson hypothesis, focusing on the mode of anger expression following anger instigation and its differential effects on cardiovascular reactivity and recovery. Dorr and associates (2007) tested differential effects of anger expression versus inhibition on cardiovascular recovery and they examined the extent to which African Americans would exhibit greater reactivity to racist stress (a debate procedure). To manipulate racist stress, both African American and European American participants argued with a European American confederate about a race-related issue, such as racial differences in intelligence. To manipulate non-racist stress, participants argued with the confederate about non-race related issues, such as abortion. To examine the impact of the mode of anger expression on recovery, anger inhibition versus expression condition, participants evaluated the confederate, whereas in the anger inhibition condition, participants evaluated their best friend. The resulting experiment was a 2 (Ethnicity: African American versus European American) x 2 (Debate Stressor: Racist versus Non-racist) x 2 (Anger Inhibition versus Anger Expression) factorial design (Dorr et al., 2007).

The participants were 24 African American and 26 European American male college students from a large predominately European American Midwestern community (Dorr et al., 2007). The physiological measures assessed in this study were SBP and DBP, HR, cardiac output, and pre-ejection period (PEP) were assessed using impedance cardiography (Dorr et al., 2007). Total peripheral resistance (TPR) was computed using a mathematical formula (total peripheral resistance = mean arterial pressure/cardiac

output). The results revealed that African Americans who expressed their anger had slower SBP recovery that was generally greater than their SBP baseline and was reliably greater than African Americans who inhibited their anger (Dorr et al., 2007). European Americans' SBP recovered to baseline levels independent of mode of anger expression. African Americans who expressed their anger had DBP recovery that was slower than both African American Inhibitors and slower than European American Expressors. Additionally, European American Inhibitors showed slower recovery when inhibiting their anger, but this was only evidenced during the first two recovery minutes (Dorr et al., 2007). TPR during anger inhibition was associated with delayed recovery in all participants. The reactivity results revealed that for African Americans, SBP, CO, and HR reactivity was greater during the race debate than the non-race debate. No reliable difference between debates emerged for DBP, TPR, PEP, and heart rate variability (HRV). The difference in European American's reactivity between race and non-race debate was not reliable (*ps* >.10). African Americans also showed less HRV during both the race and non-race debates than did European Americans. These researchers did not find reliable reactivity differences between African Americans and European Americans on any other measure (Dorr et al., 2007).

Vella and Friedman (2009) evaluated the role of anger inhibition and hostility, and the moderating situational influences of harassment and evaluation, in predicting cardiovascular reactivity and recovery to mental arithmetic. To accomplish the research goals, 48 male undergraduate students took part in 3-minute tasks. The baseline task included the participants seated quietly. In the stressor the participant engaged in a mental arithmetic task in which the subject performed a serial subtraction. Participants were

either placed in a harassment or non-harassment condition to test the effect of harassment on cardiovascular reactivity. Participants in the non-harassment condition were permitted to complete the task without commentary. In the harassment condition, participants were exposed to verbal harassment through audiotape recorded statements played over an intercom.

The results of the study found that race predicted DBP in that individuals of Asian or African American ethnicities displayed larger increases in DBP to mental arithmetic stress relative to European American participants (Vella & Friedman, 2009). A significant interaction was found between hostility and anger inhibition in predicting DBP reactivity, as well as a marginal interaction between these variables for SBP reactivity (Vella & Friedman, 2009). Results also revealed that anger inhibition moderated the effects of hostility on DBP responders to math arithmetic stress, whereby hostile men scoring high on anger inhibition displayed the most task reactivity, which was significantly greater than hostile men scoring low on anger inhibition.

For the recovery analysis, the researchers found two significant 3-way (harassment x evaluation x anger inhibition) and (harassment x evaluation x hostility) interactions on SBP recovery during the evaluation period (Vella & Friedman, 2009). The researchers also found the interaction between evaluation and anger inhibition to be significant among harassed participants, whereas this interaction in the absence of harassment was not significant (Vella & Friedman, 2009).

Neumann, Waldstein, Sollers, Thayer, and Sorkin (2004) investigated the role of dispositional hostility in cardiovascular reactivity and the relation of dispositional hostility during an anger recall-task. The role of distraction to post-task recovery was also

assessed, examining the link between hostility and cardiovascular reactivity and disease. Eighty healthy women, ages 18-30, participated in the study to evaluate dispositional hostility and cardiovascular reactivity patterns in women (Neumann et al., 2004). The psychosocial measures used were the Cook-Medley Hostility (Ho) Scale, the S-Anger subscale of the STAXI, the Revised Impact of Events Scale (IES) to assess state rumination. The dependent variables assessed were SBP and DBP, HR, Cardiac pre-ejection period (PEP), stroke volume (SV), left ventricular ejection time (LVET), cardiac output (CO), and total peripheral resistance (TPR) (Neumann et al., 2004).

The procedure involved the participant engaging in a 15-minute baseline period, a 3-minute anger recall task, and a 10-minute recovery period. In the anger recall task, participants were asked to recall and talk about a recent event, occurring within the last year, which continued to make the individual angry, frustrated, irritated, or upset (Neumann et al., 2004). In the recovery period, the individual was randomly assigned to either a distraction technique (i.e. reading a neutral article about the possibility of life in outer space) or a standard recovery period (i.e. no reading or implemented distractions). The S-Anger subscale was administered prior to the task. Following recovery, participants provided a retrospective self-report of state anger (S-Anger subscale) and state rumination pertaining to the task and recovery periods. The participants then completed the Ho Scale and manipulation check items created for the study (Neumann et al., 2004).

The results revealed that hostility independently predicted slower SBP and PEP post-stress recovery (Neumann et al., 2004). Additionally, the distraction condition independently predicted faster HR recovery, and hostility did not potentiate

cardiovascular reactivity or state anger during the anger recall task (Neumann et al., 2004). The Neumann and colleagues (2004) study found beneficial effects of distraction in expediting cardiovascular recovery which was likely due to reduced rumination and anger.

The aforementioned studies provided information supporting the theory that a person's experience of anger can have an effect on their cardiovascular system, specifically demonstrating increased BP with the experience of anger (cardiovascular reactivity). The reviewed studies provide information about the effectiveness of experimental manipulations in causing these cardiovascular responses, especially using mental arithmetic with harassment (Dorr et al., 2007; Neumann et al., 2006; Vella & Friedman, 2009). This is the technique that will be utilized in the current study during the stressor. The information provided in the previously reviewed studies show conflicting information regarding whether anger produces changes in SBP, DBP, or both. Furthermore, the physiological mechanism behind the cardiovascular responses evidenced in the previous studies was not explored.

Additionally, the previous studies revealed the beneficial effects of cardiovascular recovery after a stressor (Neumann et al., 2004). The current study builds on these results and attempts to show similar effects of enhanced BP recovery using relaxation techniques. Previous studies have shown that practicing relaxation techniques can have a number of beneficial effects on one's psychological and physiological health. The next section will review select studies that investigated the effectiveness of relaxation techniques at reducing BP.

General Relaxation Studies

Bruning and Frew (1987) studied the physiological effects of three interventions (management skills training, exercise, and meditation) on pulse rate, DBP, SBP, and galvanic skin response (the physiological stress indicators) over a 6-month period. There was no specific stressor studied in this experiment, the researchers focused on the effectiveness of relaxation techniques at reducing BP. The study included 65 participants, ages 23 to 60. The physiological measures were taken every 2 weeks during the 6-month period of the experiment. The participants in all groups except the control group engaged in 8 to 10 hours of training per week. Thirteen weeks into the experiment, the treatment groups were split and reassigned to another intervention and approximately 10 weeks later the groups were rearranged again. The results revealed that the stress reduction interventions led to decreases in pulse rate and SBP, supporting the researchers' hypothesis of the stress reduction strategies producing improvements in the physiological indicators (Bruning & Frew, 1987). None of the relaxation strategies were found to be superior to the others on reducing cardiovascular responses to stress alone or in combination.

Agras, Horne, and Taylor (1982) studied the BP lowering effects of expectation of relaxation training in patients with essential hypertension. The subjects in the group assigned to immediate BP lowering instructions were told that relaxation would produce immediate effects and that these effects would persist and increase with continued practice; hence they expected immediate effects (Agras et al., 1982). Subjects in the group assigned to the delayed BP lowering instructions were told that relaxation effects would be delayed over the course of the first three training sessions and that they might

even experience a slight rise in BP. Both groups were shown graphs supposedly representing data showing the patterns of BP lowering effects to be expected and which were described in the instructions. All subjects received three relaxation training sessions, each lasting for 20 minutes; all of which were completed in one day. The relaxation technique used in this study was progressive muscle relaxation (tensing briefly and then relaxing muscles). As in the previous study, SBP was found to decrease. Changes in SBP were evidenced in the group that expected immediate change in their BP as opposed to the group that was told to expect a delay in the effects of the relaxation training. DBP changes were not statistically significant (Agras et al., 1982).

Using multidimensional behavioral program developed for the treatment of essential hypertension vs. medication only controls, Lehnert, Kaluza, Vetter, Losse, and Dorst (1987) conducted a study consisting of self-monitoring of BP, health education, relaxation techniques (Jacobson's progressive muscle relaxation technique with autogenic training) and social skills training. The participants engaged in the treatment activities in addition to taking prescribed anti-hypertensive medications. The results revealed that BP levels for the two groups became normotensive after the 6-week training program. The percentage of patients taking antihypertensive medication in the treatment group was 60.2% prior to treatment and 44.4% after treatment. For the control group there were 68% and 73.8% respectively. This data was consistent at 6-month and 23-month follow assessments (Lehnert et al., 1987). The focus of this study was to evaluate the effectiveness of the behavioral program as a whole. As a result, it is difficult to determine which dimension resulted in the observed BP reduction. It can only be concluded that the combination of the relaxation training (Jacobson's progressive muscle relaxation

technique with autogenic training) and social skills training produced the reduction in BP. The researchers themselves stated, "We cannot deduce a relative hierarchy of the single training elements employed; the effective and long-lasting lowering of BP, the change in health related behaviors, and the frequency of practicing relaxation techniques instead underline the importance of a multimodal approach per se" (Lehnert et al., 1987; p. 428).

Parker, Gilbert, and Thoreson (1978) compared the effectiveness of two common relaxation techniques (progressive relaxation training (PRT) and meditation training (MT)) on autonomic arousal. The participants in the study included 30 alcoholics enrolled in a substance abuse program in a Veterans Administration hospital. The three experimental groups met for 3 weeks during which a state anxiety measure, BP, HR, and spontaneous galvanic skin responses were measured. The results indicated that both progressive relaxation training and meditation training produced a reduction in BP from baseline to after 3 weeks of training. SBP was significantly lower for MT than PRT and the control (sitting quietly) group (Parker et al., 1978). The Quiet Rest Control (QR) group's SBP was significantly higher at the end of the final training session (Parker et al., 1978). For both the PRT and MT mean DBPs were significantly lower at the end of the final training session. The QR mean was significantly higher at the end of the final training session. For HR, the three groups had lower HRs at the end of the final training session, with no differences between the groups found (Parker et al., 1978). The researchers also found that the spontaneous GSRs collected at the beginning of the first session and the end of the final session revealed no significant results (Parker et al., 1978). In summary, PRT and MT do not affect self-reported anxiety or HR over a 3-week

training period, but they do result in decreased SBP and DBP arousal (Parker et al., 1978).

The previous studies assessing the efficacy of various relaxation techniques on BP support the effectiveness of relaxation techniques, including progressive muscle relaxation and meditation, for the treatment of hypertension. Given the available literature, it is difficult to deduce the effectiveness of one relaxation technique over another. Research studies tend to assess relaxation techniques as a unit and do not specifically test the procedures and specific skill sets utilized in particular relaxation techniques to produce BP lowering effects. However, there is mounting evidence for the BP lowering effects of two specific relaxation techniques, transcendental meditation and diaphragmatic breathing. These techniques likely yield BP lowering effects by producing a generalized relaxation response which counters the cardiovascular arousal witnessed in the fight-or-flight response discussed earlier.

Transcendental Meditation

Transcendental meditation (TM) was developed by Mahesh Yogi in 1968. During TM a person repeats a mantra or repetitious sound, used as a focus of attention, and the person is taught to repeat the sound mentally in an effortless way. The task-irrelevant thought processes, such as unrelated thoughts, ideas, images, preoccupations, worries, or sensations (also known as "mental chatter"), are not usually dealt with satisfactorily in most common forms of relaxation techniques, whereas meditation approaches commonly contain explicit instructions for dealing with them.

Parati and Steptoe (2004) conducted a meta-analysis of the studies that had been conducted prior to 2004 assessing the effectiveness of TM in the treatment of

hypertension. The authors noted that most of the studies had methodological weaknesses that prevented a conclusive decision about the usefulness of TM as a treatment, i.e. small sample size, high drop-out rates, inadequate characterization of study participants, a lack of appropriate controls, and most importantly the researchers may have been affiliated with a TM organization which is a potential bias (Parati & Steptoe, 2004). Further, the authors discussed previous TM studies' methodological weaknesses in the assessment of BP which has great intra-individual and inter-individual variability and examined studies that had problems in establishing accurate baseline levels of BP against which the effects of treatment can be evaluated (Parati & Steptoe, 2004). An overview of the results provided by the meta-analysis explained that the BP reduction that was evidenced was likely due to the slowed breathing practiced in TM.

Wallace, Silver, Mills, Dillbeck, and Wagoner (1983) studied students of the Maharishi International University in Fairfield, Iowa, and found that long-term meditators had a statistically significant lower BP as compared to the norms provided by the U.S. Department of Health and Welfare publication of "Vital Statistics". There was also a significant difference in the SBP of long-term meditators (people who have practiced TM for longer than 5 years) and short-term meditators (people who have practiced for less than 5 years).

Paul-Labrador, Polk, Dwyer, Velasquez, Nidich, Rainforth, Schneider and Merz (2006) conducted a randomized, placebo-controlled clinical trial of 16 weeks of TM or active control treatment (health education), matched for frequency, and time at an academic medical center. The participants were diagnosed with stable coronary heart disease. The group of subjects who participated in TM showed beneficial changes (as

measured by a statistically significant mean change) in their SBP; in addition, to a reduction in other risk factors for heart disease, including insulin resistance and increased HRV, as compared to the health education group (Paul-Labrador et al., 2006).

The aforementioned studies that assessed TM as a treatment for hypertensive patients have provided evidence for its usefulness, especially in lowering SBP. These studies provide valuable information regarding the effectiveness of TM in the reduction of BP, but their findings are inconsistent as to the mechanism behind the reduction in BP after periods of relaxation via TM. The effectiveness of DB appears to be more clearly understood than TM.

Diaphragmatic Breathing

DB also known as abdominal breathing or belly breathing is the act of breathing deep into your lungs by flexing your diaphragm rather than breathing shallowly by flexing your rib cage (Diaphragmatic Breathing, 2009). Correctly performing DB can be marked by the expansion of the abdomen rather than the chest when breathing. It is generally considered a healthier and fuller way to ingest oxygen (Diaphragmatic Breathing, 2009).

Lee, Lee, Lee, Cornelissen, Otsuka, and Halberg (2003) studied the effect of DB on BP and HR at different times of the day and week. The DB method employed was Charles Stroebel's quieting technique of DB, used at 2 hour intervals during the active span of the day (Lee et al., 2003). The major weakness of this study was that it was a case study, observing one 16-year old. As a result, it is difficult to generalize this information; however, SBP and HR decreased. The response to DB was smallest in the morning and largest in the afternoon. There was also a decrease in SBP on the weekend.

Grossman, Grossman, Schein, Zimlichman, and Gavish (2001) evaluated the effectiveness of slow and regular breathing, not necessarily DB to lower BP. They used a method referred to as, Breathe with Interactive Music (BIM) (Grossman et al., 2001). Thirty-three subjects (23 males and 10 females) with essential hypertension, aged 25 to 75 years, recruited from an out-patient clinic, participated in this study (Grossman et al., 2001). The subjects used BIM for 10 minutes a day (in the evening) for 8 weeks. The BIM device consisted of a belt-type respiration sensor, a computerized control unit and headphones that composed real-time sound patterns with temporal structure similar to the actual breathing pattern but with prolonged expiration (in the sound pattern) (Grossman et al., 2001). The breathing pattern modification occurs as the user voluntarily follows the sound pattern with his/her breathing movements. The control group did not use the BIM device but listened to a Walkman playing a 10-minute recorded cassette of quiet synthesized music similar to that of the BIM, but with a non-identifiable rhythm (Grossman et al., 2001). This study did not measure psychological factors but focused on the effectiveness of the relaxation technique on BP. The results of this study showed a clinically and statistically significant reduction in DBP and clinically significant results that approached statistical significance ($p = 0.07$) for SBP for the treatment group as compared to the control group. This study provided more evidence for the effectiveness of a controlled breathing technique at BP reduction.

Overall, DB appeared to be a more reliable and consistent method of reducing BP. The previous studies support the hypothesis of the current study that DB will demonstrate a greater reduction in BP than MR.

Rationale

The extensive research previously discussed has elucidated certain anger coping styles, exaggerated cardiovascular reactivity, and prolonged cardiovascular recovery as cardiovascular risk factors in the development of hypertension. The pathophysiological pathway by which stress can lead to hypertension has also been revealed, namely prolonged autonomic nervous system activation. Additionally, the reviewed prevalence statistics provided information regarding the deleterious consequences of hypertension in the United States. As a result, it is imperative that more information regarding the adverse health consequences of negative emotions (specifically anger) be further studied, in addition to possible resolutions to the problem (i.e. relaxation techniques).

It has been reviewed that hypertension is usually treated with dietary changes and anti-hypertensive medications. The literature has also revealed the effectiveness of relaxation techniques in producing a general relaxation response. If relaxation techniques can produce a response in the body that counters the response induced by stress, relaxation would be an effective treatment in the reduction of hypertension. Given this information, the present study tested several hypotheses.

Hypotheses

The hypotheses for the present study are as follows:

1. Relaxation techniques can be effective at reducing BP during a recovery period from stress as will be evidenced by a statistically significant reduction in BP scores from the end of a stressor to recovery period.

2. DB will be shown to be superior to MR and a standard control (SC) in reducing BP during a recovery period. DB will show a greater decrease in BP readings from stressor to recovery period than both MR and the SC condition.
3. There will be a significant positive correlation between trait anger coping style and the cardiovascular variables (baseline SBP, baseline DBP, math task SBP, math task DBP, SBP reactivity, DBP reactivity, SBP recovery at 2, 5, 10, and DBP recovery 2, 5, 10 minutes). Higher scores on the Anger In and Anger Out subscales of the STAXI are interpreted as maladaptive anger coping styles.
4. Anger coping style will moderate BP responses at rest (baseline), during the stressor (math task), and during the recovery periods. This finding will be demonstrated by significant regression coefficients using Anger In and Anger Out subscale scores as predictor variables. For the recovery BP regression models, recovery condition assignment was also added as predictor variables. Interactions and main effects of the independent variables (recovery condition assignment and anger coping style for recovery BP) on the dependent variables were assessed.
5. Individuals with greater BP change scores following the math task (cardiovascular reactivity) will have higher resting BP means suggesting that greater physiological responses to stress are associated with general hyper-arousal that may lead to hypertension.

CHAPTER III

METHODOLOGY

Participants

The participants in this study included students enrolled at Eastern Virginia Medical School (EVMS) in Norfolk, Virginia. To be included in this study, participants needed to be generally healthy (approximately 50% women). Participants were recruited via email advertisement and distributed flyers approved by EVMS's IRB (See Appendix F). All participants were required to be English speakers between 20 and 35 years of age. Individuals were excluded from the study if they had a current psychiatric diagnosis or were being treated for psychiatric conditions. Participants were excluded if they had a current or previous diagnosis of a neurological disorder (i.e. multiple sclerosis), had a history of multiple head injuries, traumatic brain injury, or multiple losses of consciousness. An individual was also excluded from participating in the study if they suffered a stroke or cerebrovascular incident or disease, kidney or liver disease, hypertension, active cancer/treatment within the past year, electronic assistive devices that may interfere with natural heart rhythm, and participants were excluded if they were using tobacco products or smoking. There were 24 people excluded from participating in the study due to taking various medications, hormone therapy, head injuries, pregnancy, and heart disease.

Measures

Spielberger State-Trait Anger Expression Inventory (STAXI). The Spielberger State-Trait Anger Expression Inventory (STAXI) was used to measure trait anger coping style.

The STAXI assesses components of anger as an emotive state and disposition, including modes of anger expression (Spielberger, 1988). It was developed with the goal of distinguishing the components of anger in the context of both normal and abnormal personality and with the goal of examining the contribution of anger to the development or exacerbation of medical conditions such as hypertension, coronary heart disease, and cancer (Spielberger, 1988). There are seven subscales, labeled Trait Anger, Angry Temperament, Angry Reaction, Anger-In, Anger-Out, Anger Control, and Total Anger Expression. Only Anger In and Anger Out were assessed in the present study. The Anger In subscale is an 8 item scale that measures the frequency with which the respondent holds in or suppresses his or her anger. The Anger In variable was calculated by adding items: #13 + 15 + 16 + 20 + 23 + 26 + 27 + 31. The Anger Out subscale is also an 8 item scale but measures the frequency with which the respondent expresses their anger to other people or objects. The Anger Out variable was calculated by adding items: 12 + 17 + 19 + 22 + 24 + 29 + 32 + 33. All items are rated on a four point scale and are assigned a score between 1 (Almost Never) and 4 (Almost Always). The STAXI has good internal consistency (alpha coefficients = .65-.92) in populations of varying age and gender, and acceptable retest reliability (.61-.89), across multiple samples (Spielberger, 1988). The STAXI was also the recommended scale from Schum's (2003) meta-analysis as the best measure to assess the relationship of anger and cardiovascular factors.

Physiological Measures

Tanita scale. A Tanita digital scale was used to measure the participants' weight, body mass index (BMI), basal metabolic rate, fat free mass, percent body fat, and total body water.

Welch Allyn Automatic Blood Pressure Monitor. A Welch Allyn Spot Vital Signs Model [4200B; Serial # 200712247] BP monitor was used to measure the participants' BP.

Variables

The design of this study is an experimental mixed design with between-subjects variables (recovery condition and trait anger coping style) and a within-subjects variable (time: baseline, task, and recovery period at 2 minutes, 5 minutes, and 10 minutes).

Dependent Variables. The dependent variable is BP (SBP and DBP). Cardiovascular reactivity was calculated as the difference between mean BP during the math task (the stressor) and mean baseline BP during the rest period (Kelsey, Ornduff, & Alpert, 2007). Reliability of cardiovascular reactivity measures for BP are moderate, ranging from r = .46 for systolic BP reactivity and r = .37 for diastolic BP reactivity (Kelsey et al., 2007). Kelsey and associates evaluated the internal consistency reliability of cardiovascular reactivity and found the reliability of cardiovascular reactivity within tasks, as assessed by Cronbach's α coefficient of internal consistency, ranged from α = .83 to .96 for 4-minute to 5-minute math tasks (Kelsey et al., 2007). Cardiovascular Recovery was computed by averaging BP readings at 2 minutes (BP1 + BP 2/ 2), 5 minutes (BP1+BP2+BP3+BP4+BP5/5), and 10 minutes (BP1+BP2+BP3+BP4+BP5+BP6+BP7+BP8+BP9+BP10/10) following the math task.

Independent Variables. There are three main independent variables in this study (anger coping style, time [baseline, task], and recovery condition). The recovery conditions assigned in this study were DB, MR, and SC. The participants were randomly assigned to one of the three conditions. Cardiovascular recovery was assessed at 2

minutes, 5 minutes, and 10 minutes following the stressor and anger coping style was measured using the Spielberger State-Trait Anger Expression Inventory (STAXI), both described earlier.

Procedure

The study participants were screened during an initial phone interview. The phone interview obtained information regarding the exclusionary criteria (discussed above). If the individual was deemed suitable to participate in the study, they were invited to the laboratory for the testing session. Prior to participating in the study, the participants were instructed to abstain from drinking caffeine and exercising for 12 hours, using alcoholic beverages for 24 hours, or taking any medications that would affect their BP for 24 hours. Upon arrival to the laboratory, the participants signed an informed consent form and it was explained that participation in the study was voluntary. The participant was able to withdraw from the study at any time. Several pre-screen questions were asked before the study began. The questions included: 1. When was the last time you had caffeine? 2. When was the last time you had tobacco products? 3. When was the last time you exercised? 4. When was the last time you had any alcohol, cold or allergy medications or supplements? If these questions were answered in a fashion consistent with the requirements to participate in the study, the testing session ensued. If the potential participant was determined not to be appropriate for the study at that time, the experimenter rescheduled the session and explained the reasons for the reschedule.

The testing session occurred in the Psychophysiologic Evaluation and Treatment Laboratory at EVMS. The participant sat upright in a leather recliner. Once the study began, the participant was asked to complete questionnaires that addressed their

demographic information and medical history. Next, the participant's height and weight was obtained. The participant was asked to remove their shoes and socks and stand against the wall looking straight ahead with their chin up and back flat. The participant stepped on the Tanita scale and their weight, BMI, and other physiological measures were obtained. Next, the participant's BP was measured.

A BP cuff of the appropriate size for the participant's arm was attached to the participant's non-dominant arm. During the baseline phase, the participant was asked to sit with his/her feet flat on the floor, without moving around very much for a couple of minutes and rest. The baseline period lasted for 10 minutes followed by the Math Task Stressor Phase (with harassment). This method of mental arithmetic with harassment is similar to the technique first established by Hokanson and Shelter in 1961, and has since been used in a variety of studies assessing anger relevant traits and cardiovascular reactivity and recovery (Dorr et al., 2007; Lai & Linden, 1992; Vella & Friedman, 2009: Neumann et al., 2006). The experimenter stated, *"I am now going to give you a number and ask you to subtract 7 from this number out loud as many times as possible in six minutes. Please complete this as quickly and accurately as possible. I will continue to monitor your heart rate and BP, so just try to ignore the BP cuff inflating and beeping during the task. Do you have any questions before we begin?"* The participant was allowed time to ask any questions. The goal of this portion of the study was to have the participant experience stress. In order to assess the participant's subjective affect, if the participant was proceeding through the task with apparent ease, i.e. at a rate of one correct answer per two seconds for three answers in a row, the session was paused. The time the experimenter stopped the session was recorded, and the participant was asked to

subtract 13 from the initial 4-digit number and the time was started again. On the other hand, if the participant was having extreme difficulty subtracting by 7's, i.e. giving more than 3 incorrect responses, the experimenter paused the session, recorded the time they stopped, and asked the participant to subtract 3 from the initial 4-digit number and the time was re-started from that point. This ability matching was done to attempt to equate the level of difficulty of the task across subjects.

At the beginning of the Math Task Stressor, the experimenter asked the participant to subtract from 9,617. Participants performed a mental arithmetic task while being harassed by the experimenter. After 1 minute, the experimenter said, *"Ok, come on, you need to go faster than that."* At 2 minutes, the experimenter said, *"Ok stop. Now begin subtracting from the number 7,485."*At 3 minutes, the experimenter stated, *"You really need to go faster. This should be simple."* At 4 minutes, the experimenter said, *"Ok stop. Now begin subtracting from the number 5,843."* At 5 minutes, the experimenter stated, *"Keep going faster. You should be getting more of these."* At 6 minutes the experimenter says, *"Ok stop, time is up."* The participant's BP was evaluated at the beginning of the Math Task, at one and a half minutes, at 3 minutes, and at four and a half minutes. The stressor portion of the experiment lasted for a total of 6 minutes.

Additional questionnaires were administered that were not included in the present study and the recovery period began. There were three possible recovery conditions that were randomly assigned (SC, DB, or MR). Under the SC Condition, the participant was asked to *"Please sit quietly without moving around too much until you are instructed to stop."* During the DB Condition, the participant was instructed to, *"Place one hand on your chest and the other on your abdomen. When you take a deep breath in, the hand on*

the abdomen should rise higher than the one on the chest. This ensures that the diaphragm is pulling air deeply into the lungs. After exhaling through the mouth, take a slow deep breath in through your nose for a count of 5 and pause for a second (as long as you are able, not exceeding 5). Then slowly exhale through your mouth, blowing the air out of your mouth for a count of 7. As all the air is released, gently contract your stomach to completely expel the remaining air from your lungs. Repeat the cycle until you are instructed to stop." The participant listened to a recording with the count to inhale, pause, and exhale throughout this 10 minute recovery condition. During the MR Condition, the participant was instructed to, *"Do the following until you are instructed to stop: Settle down in a comfortable posture with your eyes closed. Say "Peace" silently in your consciousness. Then begin repeating it. Start slowly, speaking to yourself silently as distinctly as possible. You will naturally and gradually increase the speed of repetitions. Stop as needed. As you recite your mantra, you may find that you relax into the sound. Repeat your mantra silently until you are instructed to stop."* The participant's BP was monitored during the recovery period at the beginning (0 minutes), and every 60 seconds for a total of 10 minutes.

After the recovery phase of the study, the participants were asked to fill out additional questionnaires including the STAXI. The data received in this experiment was part of a larger study and only the STAXI was utilized in the present study. The participant was then debriefed. The participant was provided $25 for their participation. They were asked to sign a receipt and a copy was kept for the records of the researchers.

Data reduction

The two cardiovascular measures assessed in the present study were SBP and DBP. The baseline mean was the average of the last four readings only. This was done to rule out possible reactivity associated with acclimating to the beginning of the testing session (Chafin et al., 2004). Further, discarding the first 5 minutes of the baseline period allows adaptation to occur and thus provides a more stable estimate of the resting BP levels (Manuck et al., 1989).

Cardiovascular reactivity was computed using the difference between the mean of baseline BP readings (defined above) and the mean of the stressor (math task) BP readings. When cardiovascular recovery was used as a dependent variable, the averages of the BP readings during the recovery period at 2 minutes, 5 minutes, and 10 minutes following the math task were used. This was done because measuring how much recovery has occurred at fixed time points following the stressor can assess residual arousal at a specific time at the end of the stressor (Christenfeld et al., 2000).

CHAPTER IV

RESULTS

Preliminary Analysis

The selected sample size (89 participants) was large enough for a significance level of $p < 0.05$ with 95% power of test and a medium Cohen's d effect size of .30 (Cohen, 1988; Jorgensen et al., 1996; Schum et al., 2003). The power analyses were run using a stand alone power analysis program, G*Power (Faul, Erdfelder, Lang, & Buchner, 2007). To test for a significant increase in BP from baseline to math task (the stressor) dependent means t-tests were conducted. The results revealed that exposure to the serial math task with harassment (the stressor) caused a significant increase from baseline to math task in SBP, $t(87) = -17.44$, $p < .001$ and DBP, $t(87) = -19.41$, $p < .001$.

Sample Demographics

Descriptive statistics (e.g., mean, standard deviation for continuous variables, frequency, and percentages for categorical variables) were computed for each demographic variable (See Table 2). The racial makeup of the sample was representative of the population of EVMS. The EVMS student body is made up of 47.2 % White, 6.6 % Black, 18.6% Asian, Hispanic 4.2%, Native American 0.5%, 12.4% Indian/Pakistani, 10.6 % other/unknown (EVMS 2008-2009 Fact Book, 2010).

Variance among the treatment groups were analyzed for the baseline period by performing separate one-way ANOVAs for DBP and SBP to test for group equivalence. The group means were not found to be significantly equal for DBP $F(2, 88) = 0.55$, *ns* or for SBP, $F(2, 88) = 0.58$, *ns* (See Table 2 for group means).

Table 2
Descriptive Statistics of Sample Demographics

	N	%	Minimum	Maximum	Mean	SD
Cardiovascular Measures						
Baseline SBP			86.50	150.00	111.49	9.80
Baseline DBP			53.25	96.00	68.14	6.60
Math Task SBP			88.25	162.75	126.62	13.99
Math Task DBP			59.75	105.50	79.84	8.84
Recovery SBP @ 2 min			86.50	151.50	114.95	10.92
Recovery SBP @ 5 min			86.60	142.00	112.75	9.61
Recovery SBP @ 10 min			87.67	137.67	110.72	9.27
Recovery DBP @ 2 min			52.50	98.50	70.30	7.56
Recovery DBP @ 5 min			52.00	95.20	68.69	7.18
Recovery DBP @ 10 min			50.67	93.00	67.23	7.26
Demographics						
Gender						
Males	41	46.1				
Females	48	53.9				
Age (years)			21	34	25.20	2.88
Race/Ethnicity						
Caucasian	55	61.8				
Black, AA	9	10.1				
Asian	16	18.0				
Latino	2	2.2				
Native American	1	1.1				
Bi- or Multicultural	5	5.6				
Other	1	1.1				
Trait Anger						
Anger In	89		9	30	15.80	3.88
Anger Out	89		8	23	13.00	2.98
Body Mass Index (kg/m^2)			16.4	31.9	23.16	3.37
Waist (inches)			21.0	39.50	29.00	3.83
Hips (inches)			26.0	44.0	34.48	3.60
Waist/Hip Ratio (inches)			0.68	1.02	0.84	0.08
Arm (cm)			15.0	34.00	25.30	3.76
Height (inches)			59.5	83.00	66.84	4.22
Weight (pounds)			82.4	250.80	148.45	32.34

Note. Total $N = 89$.

Pearson's correlations were also conducted to test for potential co-variation between the demographic variables (i.e. BMI, age, gender, etc.). BMI was found to be a covariate for the BP measures. Based on these statistics, BMI was included as a covariate in the statistical analyses for cardiovascular reactivity and recovery. None of the other demographic variables consistently correlated with the cardiovascular measures (See Table 3). Additionally, Pearson correlations of demographic variables were conducted with the trait anger variables (Anger In and Anger Out), considered to be continuous variables (See Table 4).

Testing of Hypotheses

To test the first hypothesis: "Relaxation techniques can be effective at reducing BP during a recovery period from stress as will be evidenced by a statistically significant reduction in BP scores from stressor to recovery period", ANCOVAs were conducted using BMI, average baseline BP, and BP Reactivity (the change score of math task BP minus baseline BP) as covariates. BMI was included as a covariate based on preliminary analyses showing a significant relation between BMI and the BP variables. BMI was also found to be significantly related to BP in similar studies (Starner & Peters, 2004). The respective baseline and reactivity BP scores were included as covariates to control for possible differences in baseline BP readings and math task BP readings that may affect the relationship between recovery BP scores and the recovery assignment. The dependent variables used in the ANCOVAs were the average SBP and DBP means during the recovery period at 2 minutes, 5 minutes, and 10 minutes with each ANCOVA ran separately for each dependent variable.

Table 3

Pearson's Correlations of Cardiovascular and Body Type Variables

Cardiovascular Measure (mm Hg)	Body Mass Index (kg/m^2)	Weight (pounds)	Height (inches)	Waist (inches)	Hips (inches)	Waist/Hip Ratio (inches)
Avg. Baseline SBP (N)	.40** (88)	.46** (89)	.34** (89)	.47** (89)	.17 (89)	.48** (89)
Avg. Baseline DBP (N)	.31** (88)	.15 (89)	-.07 (89)	.28** (89)	.16 (89)	.20 (89)
Math Task SBP (N)	.45** (87)	.48** (88)	.32** (88)	.52** (88)	-.26* (88)	.47** (88)
Math Task DBP (N)	.35** (87)	.25* (88)	.04 (88)	.39** (88)	.26* (88)	.27* (88)
SBP Reactivity (N)	.29** (87)	.28** (88)	.15 (88)	.33** (88)	.25** (88)	.21 (88)
DBP Reactivity (N)	.18 (87)	.21 (88)	.15 (88)	.28** (88)	.21 (88)	.19 (88)
Recovery SBP (N)	.41** (87)	.43** (88)	.28** (88)	.45** (88)	.16 (88)	.46* (88)
Recovery DBP (N)	.28** (87)	.12 (88)	-.09 (88)	.24* (88)	.21 (88)	.10 (88)

Note. $*p < 0.05$, $**p < 0.01$

Table 4

Correlations of Cardiovascular Measures and Demographic Information

Cardiovascular Measures (mm Hg)	Anger In	Anger Out	Age (years)	Race	Gender
Avg Baseline SBP (N)	-.15 (89)	-.01 (89)	.13 (89)	-.16 (89)	-.64** (89)
Avg Baseline DBP (N)	-.02 (89)	.12 (89)	.14 (89)	.12 (89)	-.16 (89)
Avg Math Task SBP (N)	-.14 (88)	-.00 (88)	.12 (88)	-.15 (88)	-.47** (88)
Avg Math Task DBP (N)	-.06 (88)	.02 (88)	.10 (88)	.07 (88)	-.15 (88)
SBP Reactivity (N)	-.06 (88)	.00 (88)	.04 (88)	-.12 (88)	-.10 (88)
DBP Reactivity (N)	-.08 (88)	-.11 (88)	-.01 (88)	-.05 (88)	-.12 (88)
Avg Recovery SBP (N)	-.16 (88)	-.05 (88)	.18 (88)	-.09 (88)	-.53** (88)
Avg Recovery DBP (N)	-.07 (88)	.03 (88)	.14 (88)	.17 (88)	-.05 (88)

Note. Age is a continuous variable correlated with Pearson's correlations, r. Race is a categorical variable correlated with biserial correlation coefficient, r_b and entered into SPSS as 1= Caucasian, White; 2= African American, Black; 3 = Asian; 4 = Latino or Hispanic; 5= Native American; 6 = Bi- or multiracial; 7 = Other. Gender is a categorical variable and was correlated with Point-biserial coefficient correlations r_{pb} (entered into SPSS as 1 = Male, 2 = Female. Anger Scales are continuous variables and were correlated with Pearson's correlations. $^{**}p < 0.01$.

The results for the first hypothesis indicated that for recovery SBP, the relaxation techniques were not significantly related to BP at 2 minutes, $F(2, 78) = 0.71$, *ns*, partial $\eta^2 = .02$; at 5 minutes, $F(2, 78) = 0.48$, *ns*, partial $\eta^2 = .01$; or at 10 minutes, $F(2, 80) = .83$, *ns*, partial $\eta^2 = .02$ (See Tables 5-7 & Figure 1). However, recovery DBP produced significant results and demonstrated that relaxation techniques can be effective at reducing DBP at 2 minutes, $F(2, 78) = 7.27$, $p = .001$, partial $\eta^2 = .16$; 5 minutes, $F(2, 78) = 15.46$, $p < .001$, partial $\eta^2 = .28$; and 10 minutes $F(2, 80) = 17.89$, $p < .001$, partial $\eta^2 = .31$. (See Tables 8-10 & Figure 2). BMI did not produce significant results; however, respective baseline BP and BP reactivity were significant covariates for all equations. Significant mean differences for DBP were revealed for DB only.

Table 5

Analysis of Covariance for SBP at 2 Minutes by Recovery Condition

Source	*SS*	*df*	*MS*	*F*	partial η^2
Body Mass Index (cv)	4.03	1	4.03	0.19	.00
Baseline SBP (cv)	5945.69	1	5945.69	275.84***	.78
SBP Reactivity (cv)	466.22	1	466.22	21.63***	.22
Recovery Condition	30.54	2	15.27	0.71	.02
Error	1681.28	78	21.56		

Note. cv = covariate. *** $p < .001$.

Table 6

Analysis of Covariance for SBP at 5 minutes by Recovery Condition

Source	*SS*	*df*	*MS*	*F*	partial η^2
Body Mass Index (cv)	3.45	1	3.45	0.31	.00
Baseline SBP (cv)	5140.46	1	5140.46	463.95***	.86
SBP Reactivity (cv)	248.23	1	248.23	22.40***	.22
Recovery Condition	10.61	2	5.30	0.48	.01
Error	864.23	78	11.08		

Note. cv = covariate. *** $p < .001$.

Table 7

Analysis of Covariance for SBP at 10 minutes by Recovery Condition

Source	*SS*	*df*	*MS*	*F*	partial η^2
Body Mass Index (cv)	1.43	1	1.14	0.11	.00
Baseline SBP (cv)	4799.58	1	4799.58	448.91***	.85
SBP Reactivity (cv)	215.59	1	215.59	20.16***	.20
Recovery Condition	17.81	2	8.90	0.83	.02
Error	855.33	80	10.69		

Note. cv = covariate. *** $p < .001$.

Table 8

Analysis of Covariance for DBP at 2 minutes by Recovery Condition

Source	*SS*	*df*	*MS*	*F*	partial η^2
Body Mass Index (cv)	6.50	1	6.50	0.64	.01
Baseline DBP (cv)	3462.70	1	3462.70	340.40***	.81
DBP Reactivity (cv)	178.47	1	178.47	17.55***	.18
Recovery Condition	147.76	2	73.88	7.37***	.16
Error	793.22	78	10.17		

Note. cv = covariate. *** $p < .001$.

Table 9

Analysis of Covariance for DBP at 5 minutes by Recovery Condition

Source	*SS*	*df*	*MS*	*F*	partial η^2
Body Mass Index (cv)	5.40	1	5.40	0.80	.01
Baseline DBP (cv)	3319.49	1	3319.45	491.92***	.87
DBP Reactivity (cv)	98.17	1	98.17	14.55***	.16
Recovery Condition	208.60	2	104.30	15.46***	.28
Error	526.34	78	6.75		

Note. cv = covariate. *** $p < .001$.

Table 10

Analysis of Covariance for DBP at 10 minutes by Recovery Condition

Source	*SS*	*df*	*MS*	*F*	partial η^2
Body Mass Index (cv)	18.05	1	18.05	2.05	.03
Baseline DBP (cv)	3285.43	1	3285.43	372.88***	.82
DBP Reactivity (cv)	67.45	1	67.45	7.66**	.09
Recovery Condition	315.28	2	157.64	17.89***	.31
Error	704.88	80	8.81		

Note. cv = covariate. *** $p < .001$, ** $p < .01$.

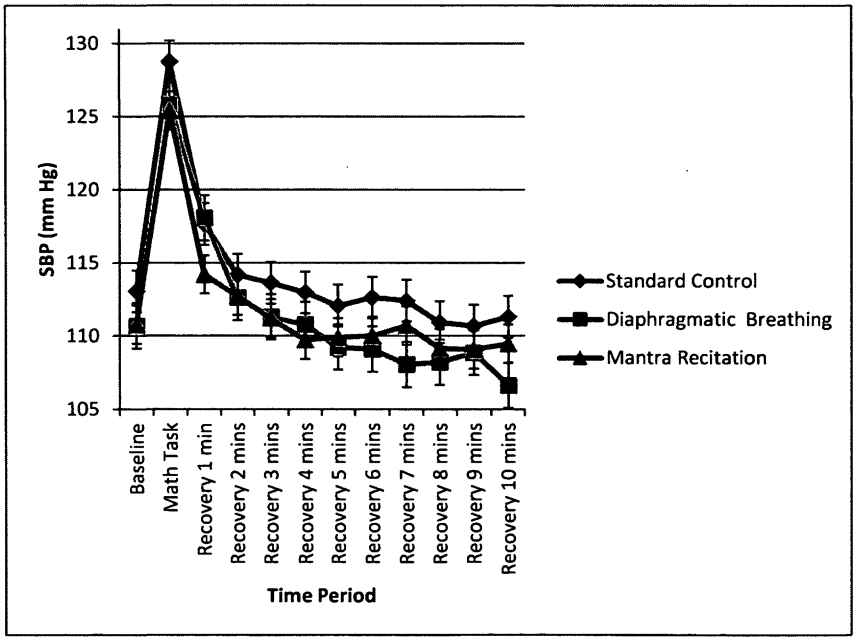

Figure 1. Systolic blood pressure (mm Hg) across time. Mean SBP by time (baseline to the 10th minute of the recovery condition). Error bars represent standard error of the means.

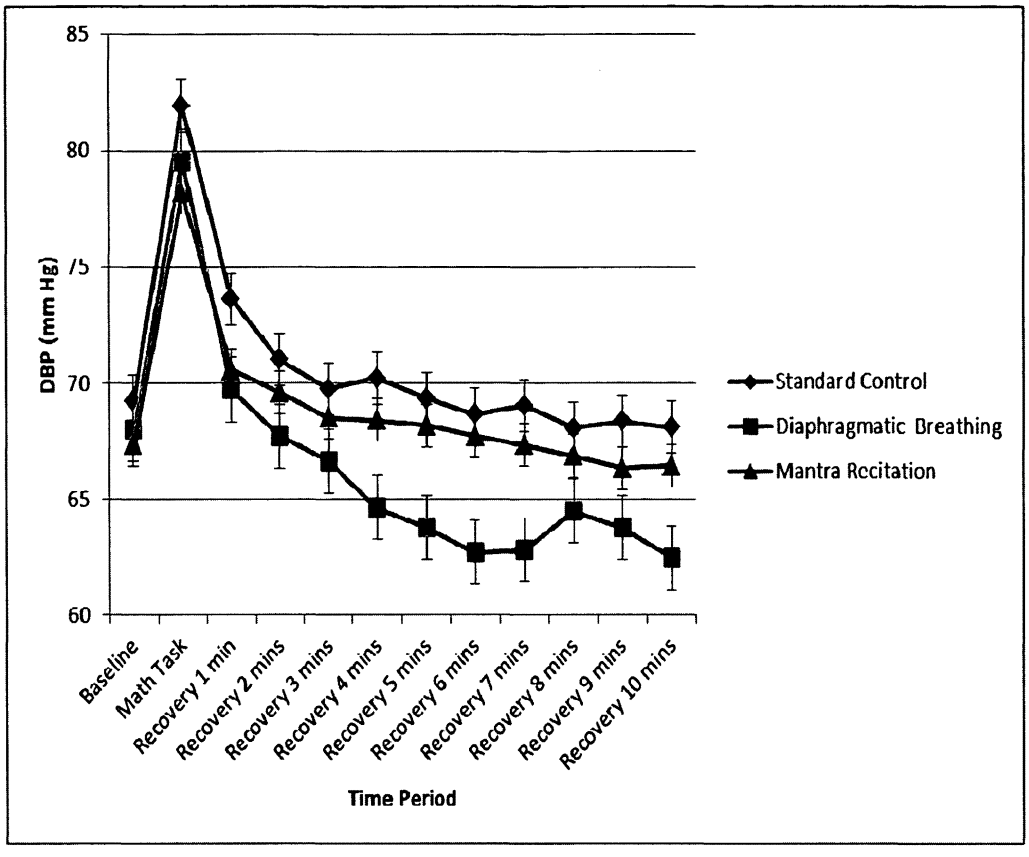

Figure 2. Diastolic blood pressure (mm Hg) across time. Mean DBP by Time (baseline to the 10th minute of the recovery condition. Error bars represent standard error of the means.

To test the second hypothesis: "DB will be shown to be superior to MR and the SC in reducing BP during a recovery period. DB will show a greater decrease in BP readings from stressor to recovery period than both MR and the SC," apriori planned contrasts were conducted. The contrasts were planned based on theoretical hypothesis and previous research studies which showed the effectiveness of DB exercises at BP reduction (Grossman et al., 2001; Lee et al., 2003).This hypothesis was supported, DB was shown to be superior to MR and the SC in reducing DBP, F (1, 85) = 6.11, $p < .05$, SE = .76, 95% CI [65.77, 68.77], partial η^2 = 0.07. For recovery DBP, the contrast

comparing the control group to the DB and MR condition did not produce significant results, F (1, 85) = 2.86, *ns, SE* = .76, 95% CI [65.77, 68.77], partial η^2 = .03, and the contrast comparing MR to SC and DB groups was not significant, F (1, 85) = .56, *ns, SE* = .76, 95% CI [65.77, 68.77], partial η^2 = .01 (See Table 12 & Figures 4, 5, & 8).

The contrasts comparing the recovery conditions using SBP did not support the hypothesis. The contrast comparing DB to the SC and MR conditions was not significant, F (1, 85) = .81, *ns, SE* = .99, 95% CI [108.77, 112.72], partial η^2 = .01. Additionally, the SC condition was not significantly different from the DB and MR conditions, F (1, 85) = .84, *ns, SE* = .99, 95% CI [108.77, 112.72], partial η^2 = .01, and the MR condition was not significantly different from the SC and DB condition for SBP, F (1, 85) = .00, *ns, SE* = .99, 95% CI [108.77, 112.72], partial η^2 = .00 (See Table 11 & Figures 3, 6, & 7).

Table 11
Mean Systolic Blood Pressure (mm Hg) by Recovery Conditions (with Standard Deviations in Parentheses)

	Baseline	Math Task	Recovery		
			2 min	5 min	10 min
SC	113.06 (10.83)	128.77 (11.22)	115.70 (9.43)	113.56 (9.08)	112.05 (8.79)
DB	110.69 (8.78)	125.83 (15.26)	115.80 (12.58)	113.22 (10.37)	109.49 (10.06)
MR	110.77 (9.87)	125.40 (15.18)	113.47 (10.77)	111.55 (9.58)	110.70 (9.02)

Note. SC = Standard Control; DB = Diaphragmatic Breathing; MR = Mantra Recitation.

Table 12
Mean Diastolic Blood Pressure (mm Hg) by Recovery Conditions (with Standard Deviations in Parentheses)

	Baseline	Math Task	Recovery		
			2 min	5 min	10 min
SC	69.12 (8.02)	81.91 (9.01)	72.14 (7.99)	70.31 (7.79)	69.10 (7.78)
DB	67.99 (4.30)	79.54 (7.66)	68.70 (6.57)	66.70(5.77)	64.64 (5.65)
MR	67.33 (7.05)	78.20 (9.64)	70.30 (7.56)	69.05 (7.55)	68.07 (7.66)

Note. SC = Standard Control; DB = Diaphragmatic Breathing; MR = Mantra Recitation.

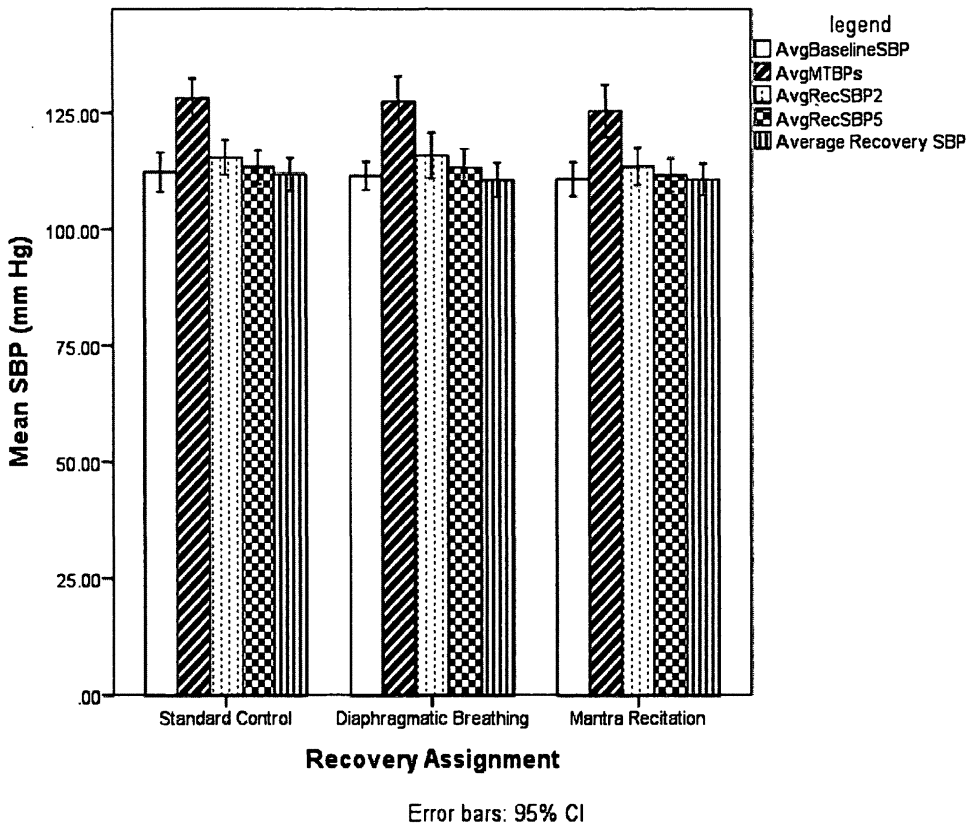

Figure 3. Bar graph of systolic blood pressure by recovery assignment. Average SBP by Recovery assignment. AvgBaselineSBP = Average Baseline SBP; AvgMTBPs = Average Math Task SBP; AvgRecSBP2 = Average Recovery SBP at 2 minutes; AvgRecSBP5 = Average Recovery SBP at 5 minutes; Average Recovery SBP = Average Recovery SBP at 10 minutes. Error bars represent 95% confidence interval.

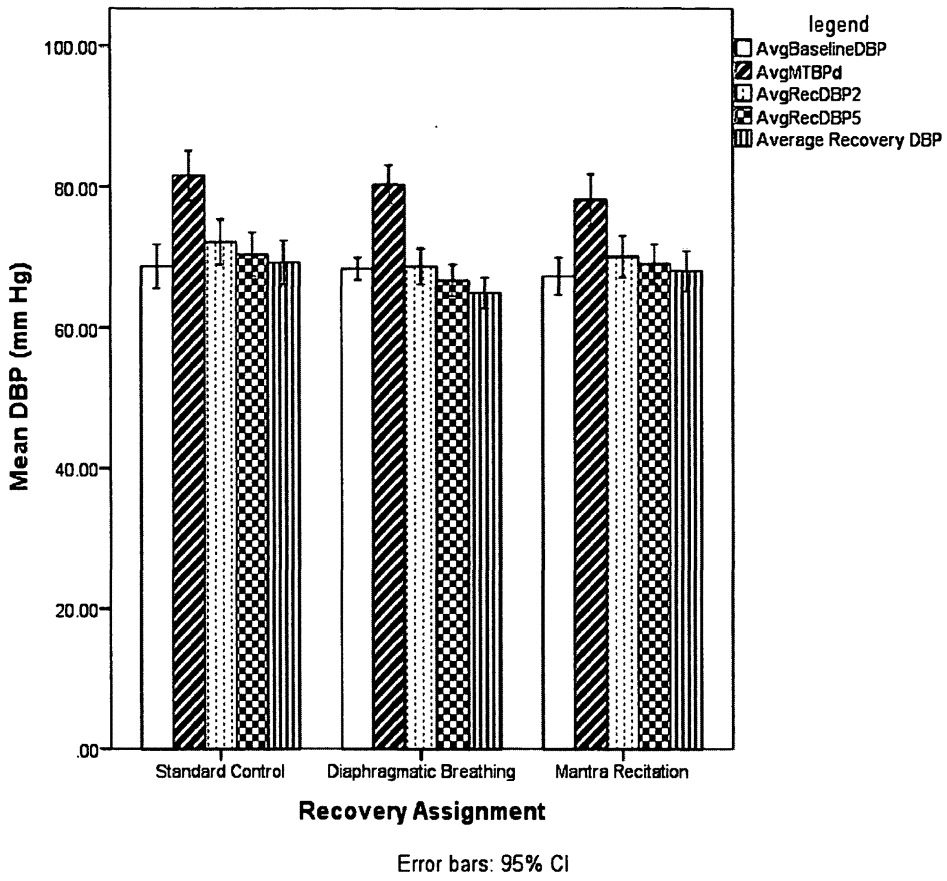

Figure 4. Bar graph of diastolic blood pressure by recovery assignment. Average DBP by Recovery assignment. AvgBaselineDBP = Average Baseline SBP; AvgMTBPd = Average Math Task DBP; AvgRecDBP2 = Average Recovery DBP at 2 minutes; AvgRecDBP5 = Average Recovery DBP at 5 minutes; Average Recovery DBP = Average Recovery DBP at 10 minutes. Error bars represent 95% confidence interval.

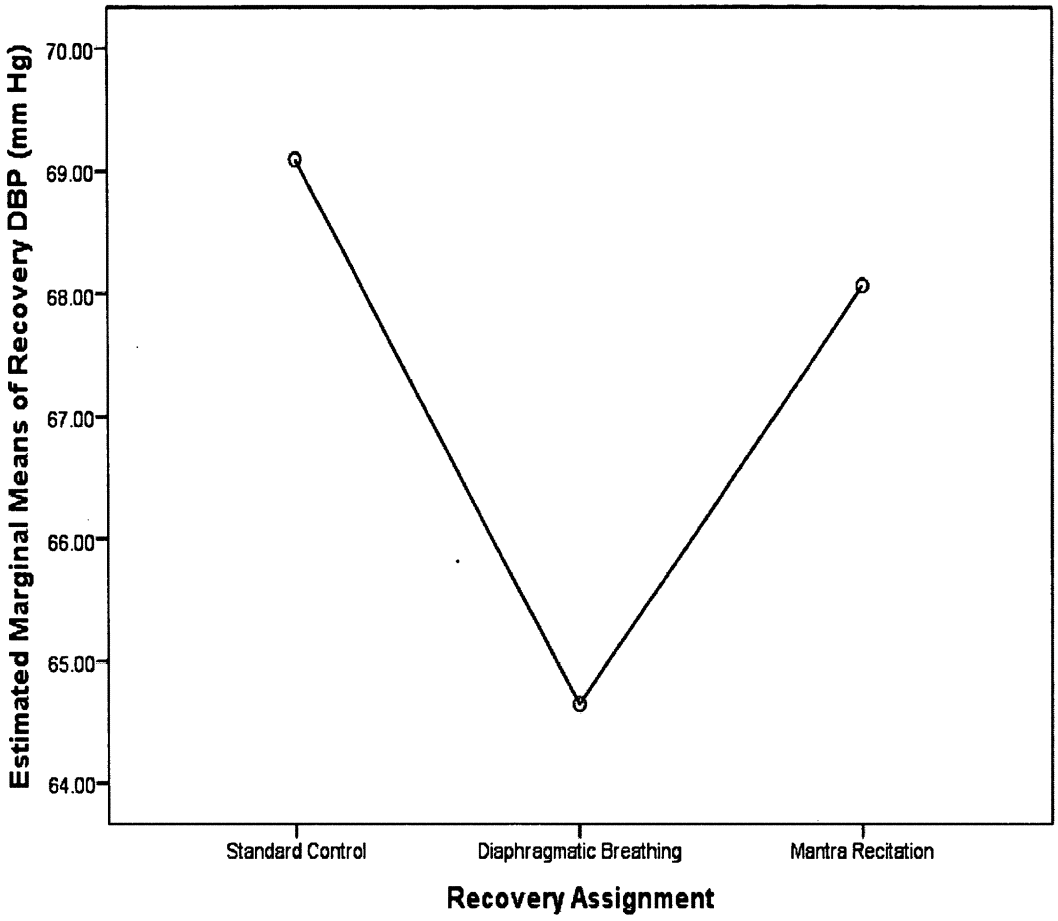

Figure 5. Estimated marginal means of recovery Diastolic blood pressure at 10 Minutes from Significant Contrast. Estimated marginal means are predicted means based on the equation, not actual DBP means. Diaphragmatic Breathing resulted in the quickest return to baseline BP, based on change scores (Recovery-Baseline).

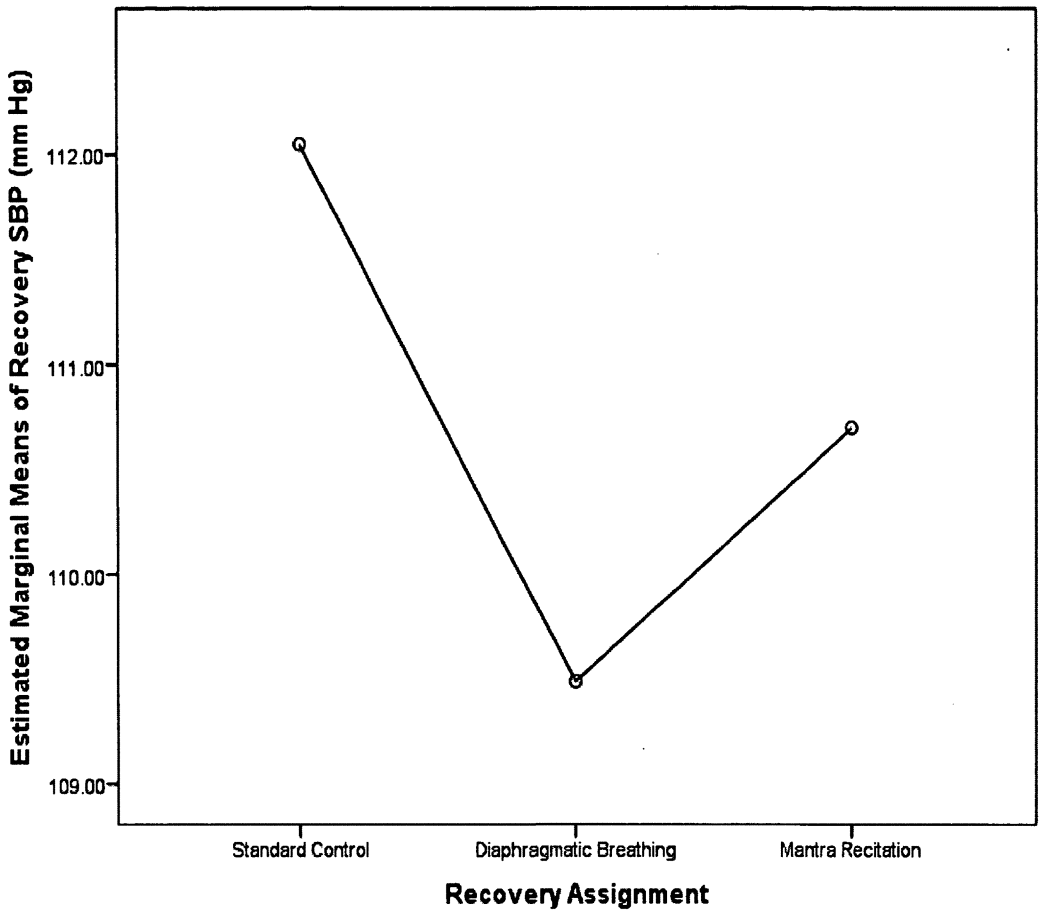

Figure 6. Estimated marginal means of recovery systolic blood pressure at 10 minutes. Estimated marginal means are predicted means based on the equation, not actual SBP means. Diaphragmatic Breathing resulted in the quickest return to baseline BP, based on change scores (Recovery-Baseline).

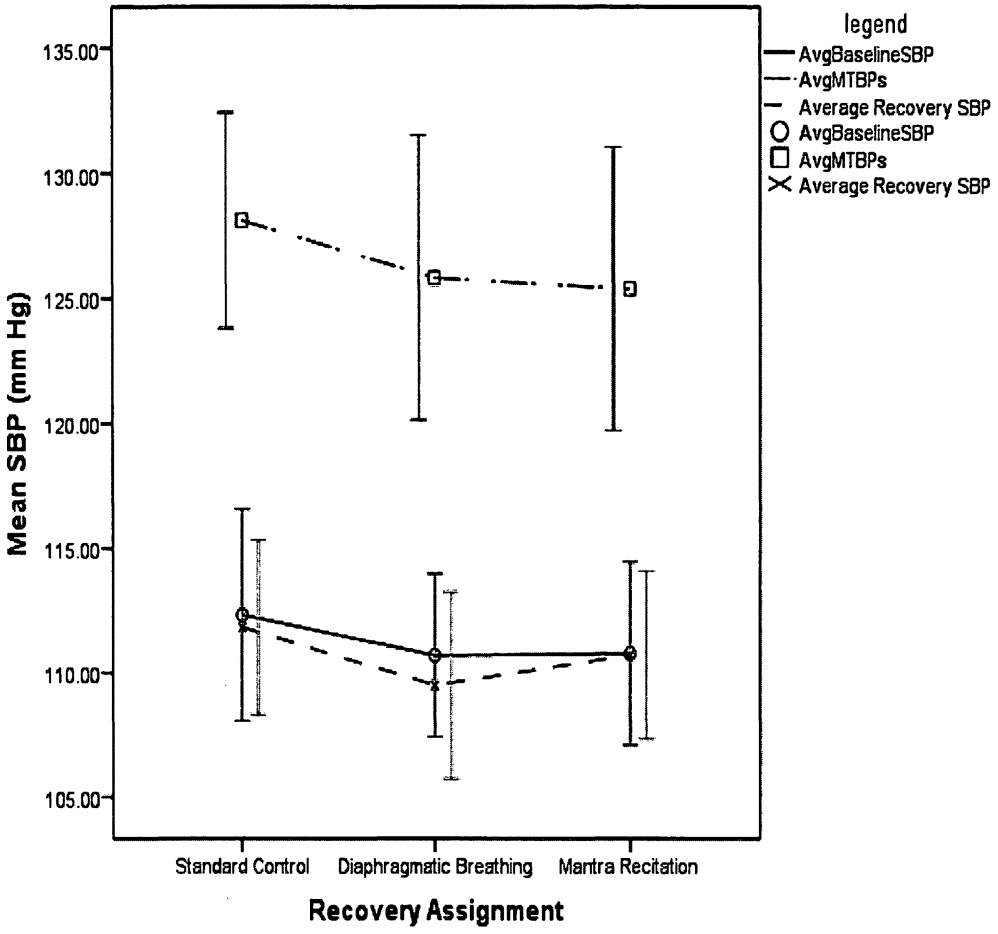

Figure 7. Systolic blood pressure by recovery condition at 10 minutes. Average baseline SBP is the average of the last four SBP measures during the baseline period. AvgMTBP is the average of the SBP readings during the math task stressor. Average recovery SBP is the average of the SBP readings across the 10 minute recovery condition. Error bars represent 95% confidence intervals.

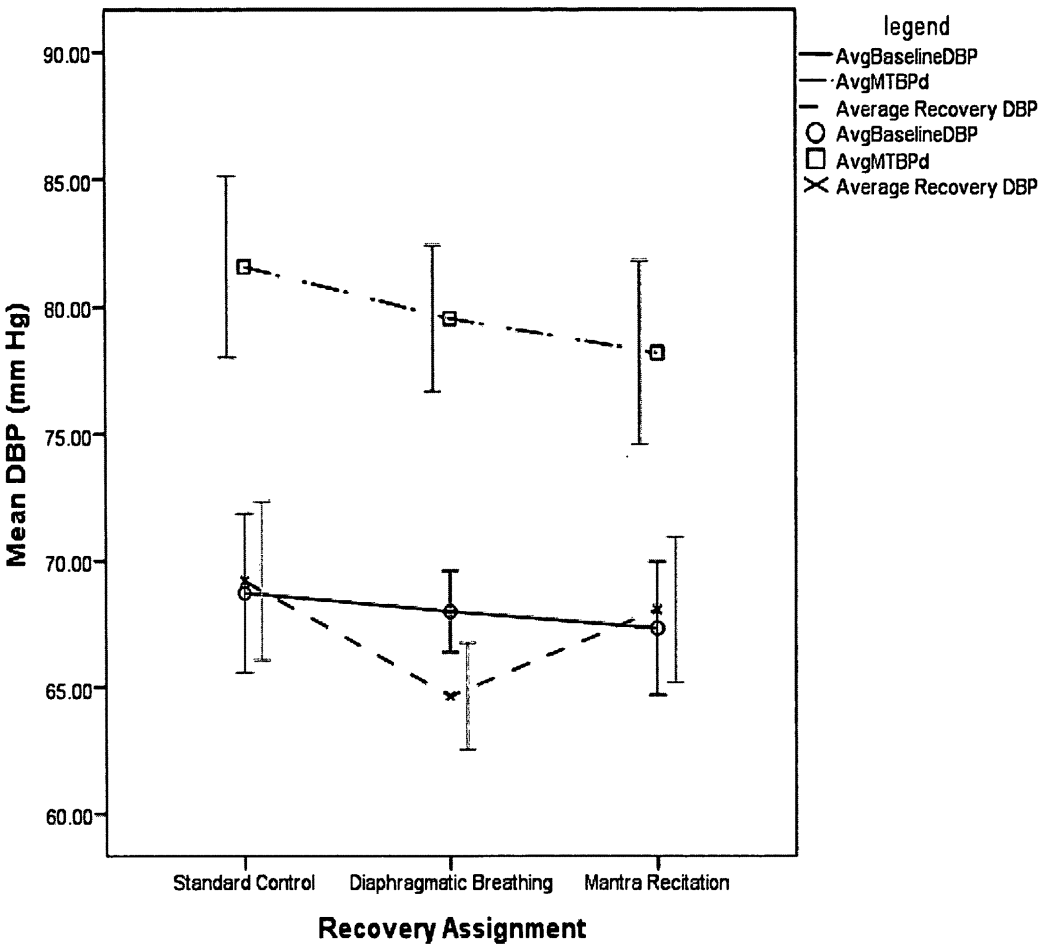

Figure 8. Diastolic blood pressure by recovery condition at 10 minutes. Average BaselineDBP (AvgBaselineDBP) is the average of the last four DBP measures during the baseline period. AvgMTBP is the average of the DBP readings during the math task stressor. Average recovery DBP is the average of the DBP readings across the10 minute recovery condition. Error bars represent 95% confidence intervals.

The third hypothesis is "There will be a significant positive correlation between trait anger coping style and the cardiovascular variables (baseline SBP, baseline DBP, math task SBP, math task DBP, SBP reactivity, DBP reactivity, SBP recovery at 2, 5, 10, and DBP recovery 2, 5, 10 minutes). Higher scores on the Anger In and Anger Out subscales of the STAXI are interpreted as maladaptive anger coping styles". This hypothesis was not supported. Anger In and Anger Out were not significantly related to any cardiovascular measure (baseline SBP, baseline DBP, math task SBP, math task DBP, SBP reactivity, DBP reactivity, SBP recovery at 2, 5, 10, and DBP recovery 2, 5, 10 minutes) (See Table 13).

Table 13

Correlations of Cardiovascular Measures and Trait Anger Coping Style

Cardiovascular Measure (mm Hg)	Anger In	Anger Out
Baseline SBP	-.15	-.01
Baseline DBP	-.02	.12
Math Task SBP	-.14	.00
Math Task DBP	-.06	.02
SBP Reactivity	-.06	.00
DBP Reactivity	-.08	-.11
Recovery SBP @ 2 min	-.14	-.02
Recovery SBP @ 5 min	-.15	-.01
Recovery SBP @ 10 min	-.16	-.05
Recovery DBP @ 2 min	-.11	.02
Recovery DBP @ 5 min	-.08	.02
Recovery DBP @ 10 min	-.07	.03

Note. These correlations are not statistically significant.

The fourth hypothesis is "Anger coping style will moderate BP responses at rest (baseline), during the stressor (math task), and during the recovery periods. This finding will be demonstrated by significant regression coefficients using Anger In and Anger Out subscale scores as predictor variables. For the recovery BP regression models, recovery condition assignment was also added as predictor variables. Interactions and main effects of the independent variables (recovery condition assignment and anger coping style for recovery BP) on the dependent variables were assessed".

For the models with baseline BP readings and math task BP, multiple regression analyses were performed to examine the effects of trait anger coping style on BP. SBP Reactivity and DBP Reactivity and the recovery variables were also assessed as additional statistical information on the relation of anger coping style with cardiovascular variables. To conduct these analyses: Anger In and Anger out subscale scores were each regressed separately on baseline mean SBP and DBP, stressor (or math task) SBP and DBP, and SBP and DBP change scores (assessing cardiovascular reactivity) resulting in 12 separate regression models. BMI was included as a covariate. Results revealed that trait anger coping style did not significantly predict, and was not related to BP at baseline, during a stressor, cardiovascular reactivity, or during recovery from the stressor (See Table 14). BMI was a significant covariate for all equations.

To further assess the recovery dependent variables, additional separate hierarchical multiple regression models (separate models for each anger coping style) were calculated to evaluate relaxation technique assignment and either Anger In or Anger Out subscale scores on 2-min, 5-min, and 10-min SBP or DBP averages for the recovery period. The models were calculated as follows: ([Step 1: Covariates: Body Mass Index

(BMI), respective Baseline BP, and respective BP Reactivity; ([Step 2: Main Effect (Anger In and Relaxation Technique group assignment); Step 3: (Interaction Term) Anger In*Relaxation Technique Group on 2-min, 5-min, 10-min SBP; 2-min, 5-min, 10-min DBP means (Dependent Variables)] and ([Step 1: Covariates: Body Mass Index, respective Baseline BP, and respective BP Reactivity; [Step 2: Main Effect: Anger Out and Relaxation Technique group; [Step 3: Interaction Term: Anger Out*Relaxation Technique Group; on 2-min, 5-min, 10-min SBP and 2-min, 5-min, 10 min DBP]. The first step held BMI constant for SBP and DBP baseline readings and SBP and DBP task reactivity for BP recovery readings.

The results for the recovery BP regressions revealed that the models used to predict cardiovascular measures using the various covariates and trait anger coping style and relaxation techniques as independent variables were accurate (indicated by significant *F* tests) (See Tables 15-26). However, there were no significant main effects or interaction terms for recovery assignment or trait anger coping style. In combination, the results of the regression analyses revealed that trait anger coping style does not moderate BP responses.

Table 14

Multiple Regression Analyses for Trait Anger Coping Style Predicting Baseline, Stressor, and Cardiovascular Reactivity

Criterion	β	*p*	*R*	R^2
Anger In				
Baseline SBP (mm Hg)	-.14	.17	.42	.18
Baseline DBP (mm Hg)	-.01	.95	.31	.09
Math Task SBP (mm Hg)	-.05	.19	.47	.22
Math Task DBP (mm Hg)	-.05	.60	.35	.12
SBP Reactivity (mm Hg)	-.05	.61	.29	.08
DBP Reactivity (mm Hg)	-.08	.48	.20	.04
Anger Out				
Baseline SBP (mm Hg)	.03	.75	.40	.16
Baseline DBP (mm Hg)	.15	.15	.34	.12
Math Task SBP (mm Hg)	.05	.63	.45	.20
Math Task DBP (mm Hg)	.05	.60	.35	.12
SBP Reactivity (mm Hg)	.03	.76	.29	.08
DBP Reactivity (mm Hg)	-.09	.40	.20	.04

Note. There were no significant relationships found between trait anger coping style and BP. Body Mass Index (BMI) used as covariate and there were no significant equations.

Table 15

Hierarchical Regression of Anger In and Recovery Assignment on Recovery SBP at 2 minutes

Variable	*B*	*SE*	β	*R*	R^2
Step 1				.91	.83
Constant	1.30	6.14			
BMI	0.01	0.17	.00		
Baseline SBP	0.97	.06	.84***		
SBP Reactivity	0.32	.07	.23***		
Step 2				.91	.83
Constant	-0.64	7.11			
BMI	0.01	0.17	.00		
Baseline SBP	0.98	0.06	.85***		
SBP Reactivity	0.32	0.07	.23***		
Anger In	0.91	0.14	.03		
Recovery Assignment	-0.08	0.63	-.01		
Step 3				.91	.83
Constant	-3.84	8.24			
BMI	0.00	0.17	.00		
Baseline SBP	0.98	0.06	.85***		
SBP Reactivity	0.32	0.07	.24***		
Anger In	0.32	0.33	.11		
Recovery Assignment	1.91	2.63	.14		
Anger In x Recovery	-0.13	0.17	.17		

Note. ***$p < .001$. For Step 3: $F(6, 77) = 63.21$, $p < .001$.

Table 16

Hierarchical Regression of Anger In and Recovery Assignment on Recovery SBP at 5 Minutes

Variable	*B*	*SE*	β	*R*	R^2
Step 1				.94	.89
Constant	7.86	4.39			
BMI	0.03	0.12	.01		
Baseline SBP	0.90	0.04	.89***		
SBP Reactivity	0.23	0.05	.19***		
Step 2				.94	.89
Constant	6.58	5.09	.01		
BMI	0.03	0.12	.89		
Baseline SBP	0.91	0.04	.19***		
SBP Reactivity	0.23	0.05	.03***		
Anger In	0.06	0.10	-.01		
Recovery Assignment	-0.09	0.45	-.01		
Step 3				.94	.89
Constant	6.29	5.91			
BMI	0.30	0.12	.01		
Baseline SBP	0.91	0.04	.89***		
SBP Reactivity	0.23	0.05	.19***		
Anger In	0.08	0.23	.03		
Recovery Assignment	0.09	1.89	.01		
Anger In x Recovery	-0.01	0.12	-.02		

Note. ***$p < .001$.For Step 3: $F(6, 77) = 101.68$, $p < .001$.

Table 17

Hierarchical Regression of Anger In and Recovery Assignment on Recovery SBP at 10 Minutes

Variable	*B*	*SE*	β	*R*	R^2
Step 1				.94	.88
Constant	12.42	4.19			
BMI	0.09	0.12	.03		
Baseline SBP	0.84	0.04	.88***		
SBP Reactivity	0.20	0.05	.18***		
Step 2				.94	.88
Constant	12.81	4.78			
BMI	0.09	0.12	.03		
Baseline SBP	0.84	0.04	.87***		
SBP Reactivity	0.20	0.05	.18***		
Anger In	-0.04	0.09	-.02		
Recovery Assignment	0.18	0.45	.02		
Step 3				.94	.88
Constant	12.38	5.67			
BMI	0.09	0.12	.03		
Baseline SBP	0.84	0.04	.87***		
SBP Reactivity	0.20	0.05	.18***		
Anger In	-0.01	0.23	-.00		
Recovery Assignment	0.44	1.86	.04		
Anger In x Recovery	-0.02	0.12	-.03		

Note. ***$p < .001$. For Step 3 $F(6, 79) = 98.81$, $p < .001$.

Table 18

Hierarchical Regression of Anger In and Recovery Assignment on Recovery DBP at 2 Minutes

Variable	*B*	*SE*	β	*R*	R^2
Step 1				.90	.81
Constant	-2.86	4.29			
BMI	0.03	0.12	.01		
Baseline DBP	1.02	0.06	.87***		
DBP Reactivity	0.25	0.07	.19**		
Step 2				.90	.81
Constant	-0.56	4.77			
BMI	0.03	0.12	.01		
Baseline DBP	1.02	0.06	.87***		
DBP Reactivity	0.25	0.07	.18**		
Anger In	-0.13	0.10	-.06		
Recovery Assignment	-0.04	0.47	-.01		
Step 3				.90	.81
Constant	-4.52	5.91			
BMI	0.02	0.12	.01		
Baseline DBP	1.02	0.06	.87***		
DBP Reactivity	0.25	0.07	.18**		
Anger In	0.12	0.24	.06		
Recovery Assignment	2.09	1.94	.23		
Anger In x Recovery	-0.14	0.12	-.27		

Note. $^{**}p < .01$, $^{***}p < .001$. For Step 3: $F(6, 77) = 55.58$, $p < .001$.

Table 19

Hierarchical Regression of Anger In and Recovery Assignment on Recovery DBP at 5 Minutes

Variable	*B*	*SE*	β	*R*	R^2
Step 1				.91	.83
Constant	-2.50	3.79			
BMI	0.07	0.10	.03		
Baseline DBP	0.99	0.05	.89***		
DBP Reactivity	0.18	0.06	.14**		
Step 2				.91	.83
Constant	-1.55	4.23			
BMI	0.06	0.11	.03		
Baseline DBP	0.99	0.05	.89***		
DBP Reactivity	0.18	0.06	.14**		
Anger In	-0.08	0.09	-.04		
Recovery Assignment	0.21	0.42	.02		
Step 3				.92	.83
Constant	-5.45	5.23			
BMI	0.05	0.11	.02		
Baseline DBP	1.00	0.05	.90***		
DBP Reactivity	0.18	0.06	.14**		
Anger In	0.16	0.21	.08		
Recovery Assignment	2.31	1.72	.26		
Anger In x Recovery	-0.14	0.11	-.28		

Note. $^{**}p < .01$, $^{***}p < .001$. For Step 3: $F(6, 77) = 66.16$, $p < .001$.

Table 20

Hierarchical Regression of Anger In and Recovery Assignment on Recovery DBP at 10 Minutes

Variable	*B*	*SE*	β	*R*	R^2
Step 1				.88	.78
Constant	-1.47	4.33			
BMI	0.04	0.12	.02		
Baseline DBP	0.97	0.06	.87***		
DBP Reactivity	0.14	0.07	.11*		
Step 2				.88	.78
Constant	-0.50	4.78			
BMI	0.03	0.12	.01		
Baseline DBP	0.98	0.06	.87***		
DBP Reactivity	0.15	0.07	.11*		
Anger In	-0.10	0.10	-.05		
Recovery Assignment	0.29	0.49	.03		
Step 3				.89	.78
Constant	-3.72	6.02			
BMI	0.02	0.12	.01		
Baseline DBP	0.98	0.06	.87***		
DBP Reactivity	0.15	0.07	.11*		
Anger In	0.10	0.25	.06		
Recovery Assignment	2.00	2.00	.22		
Anger In x Recovery	-0.11	0.13	-.23		

Note. $*p < .05$, $*** p < .001$. For Step 3: $F(6, 79) = 47.49$, $p < .001$.

Table 21

Hierarchical Regression of Anger Out and Recovery Assignment on Recovery SBP at 2 Minutes

Variable	*B*	*SE*	β	*R*	R^2
Step 1				.91	.83
Constant	1.29	6.14			
BMI	0.01	0.17	.00		
Baseline SBP	0.97	0.06	.84***		
SBP Reactivity	0.32	0.07	.23***		
Step 2				.91	.83
Constant	1.13	6.87			
BMI	0.02	0.17	.01		
Baseline SBP	0.97	0.06	.84***		
SBP Reactivity	0.32	0.07	.23***		
Anger Out	0.22	0.17	.01		
Recovery Assignment	-0.73	0.64	-.01		
Step 3				.91	.83
Constant	-3.16	8.62			
BMI	0.01	0.17	.00		
Baseline SBP	0.97	0.06	.84***		
SBP Reactivity	0.32	0.07	.23***		
Anger Out	0.39	0.48	.11		
Recovery Assignment	2.45	3.11	.18		
Anger Out x Recovery	-0.20	0.24	-.22		

Note. ***$p < .01$. For Step 3: $F(6, 77) = 62.89$, $p < .001$

Table 22

Hierarchical Regression of Anger Out and Recovery Assignment on Recovery SBP at 5 Minutes

Variable	*B*	*SE*	β	*R*	R^2
Step 1				.94	.89
Constant	7.85	4.39			
BMI	0.03	0.12	.01		
Baseline SBP	0.90	0.04	.89***		
SBP Reactivity	0.23	0.05	.19***		
Step 2				.94	.89
Constant	6.79	4.89			
BMI	0.05	0.12	.02		
Baseline SBP	0.90	0.04	.89***		
SBP Reactivity	0.23	0.05	.19***		
Anger Out	0.08	0.12	.03		
Recovery Assignment	-0.10	0.45	-.01		
Step 3				.94	.89
Constant	5.37	6.16			
BMI	0.04	0.12	.02		
Baseline SBP	0.90	0.04	.89***		
SBP Reactivity	0.23	0.05	.19***		
Anger Out	0.21	0.34	.06		
Recovery Assignment	0.74	2.22	.06		
Anger Out x Recovery	-0.07	0.17	-.08		

Note. ***$p < .001$. For Step 3: $F(6, 77) = 101.97$, $p < .001$

Table 23

Hierarchical Regression of Anger Out and Recovery Assignment on Recovery SBP at 10 Minutes

Variable	*B*	*SE*	β	*R*	R^2
Step 1				.94	.88
Constant	12.42	4.19			
BMI	0.09	0.12	.03		
Baseline SBP	0.84	0.04	.88***		
SBP Reactivity	0.20	0.05	.17***		
Step 2				.94	.88
Constant	12.90	4.69			
BMI	0.08	0.12	.03		
Baseline SBP	0.84	0.04	.88***		
SBP Reactivity	0.20	0.05	.18***		
Anger Out	-0.06	0.12	-.02		
Recovery Assignment	0.19	0.45	.02		
Step 3				.94	.88
Constant	12.65	5.98			
BMI	0.08	0.12	.03		
Baseline SBP	0.84	0.04	.88***		
SBP Reactivity	0.20	0.05	.18***		
Anger Out	-0.04	0.34	-.01		
Recovery Assignment	0.33	2.20	.03		
Anger Out x Recovery	-0.01	0.17	-.02		

Note. ***$p < .001$. For Step 3: $F(6,79) = 98.92$, $p < .001$

Table 24

Hierarchical Regression of Anger Out and Recovery Assignment on Recovery DBP at 2 Minutes

Variable	*B*	*SE*	β	*R*	R^2
Step 1				.90	.81
Constant	-2.86	4.29			
BMI	0.03	0.12	.01		
Baseline DBP	1.02	0.06	.87***		
DBP Reactivity	0.25	0.07	.19***		
Step 2				.90	.81
Constant	-1.74	4.74			
BMI	0.02	0.12	.01		
Baseline DBP	1.02	0.06	.88***		
DBP Reactivity	0.25	0.07	.19***		
Anger Out	-0.08	0.13	-.03		
Recovery Assignment	-0.04	0.48	-.00		
Step 3				.90	.81
Constant	-5.82	6.23			
BMI	0.01	0.12	.00		
Baseline DBP	1.03	0.06	.88***		
DBP Reactivity	0.25	0.07	.19***		
Anger Out	0.25	0.35	.10		
Recovery Assignment	2.24	2.30	.24		
Anger Out x Recovery	-0.18	0.18	-.29		

Note. $^{**}p < .01$. For Step 3: $F(6, 77) = 54.35$, $p < .001$.

Table 25

Hierarchical Regression of Anger Out and Recovery Assignment on Recovery DBP at 5 Minutes

Variable	*B*	*SE*	B	*R*	R^2
Step 1				.91	.83
Constant	-2.50	3.79			
BMI	0.07	0.10	.03		
Baseline DBP	0.99	0.05	.89***		
DBP Reactivity	0.18	0.06	.14**		
Step 2				.91	.84
Constant	-1.57	4.17			
BMI	0.05	0.11	.02		
Baseline DBP	1.00	0.05	.90***		
DBP Reactivity	0.18	0.06	.14**		
Anger Out	-0.12	0.11	-.05		
Recovery Assignment	0.23	0.42	.03		
Step 3				.91	.84
Constant	-4.75	5.48			
BMI	0.04	0.11	.02		
Baseline DBP	1.00	0.05	.90***		
DBP Reactivity	0.18	0.06	.14**		
Anger Out	0.14	0.31	.06		
Recovery Assignment	1.99	2.02	.23		
Anger Out x Recovery	-0.14	0.16	-.23		

Note. $^{**}p < .01$, $^{***}p < .001$. For Step 3: $F(6, 77) = 65.56$, $p < .001$.

Table 26

Hierarchical Regression of Anger Out and Recovery Assignment on Recovery DBP at 10 Minutes

Variable	*B*	*SE*	β	*R*	R^2
Step 1				.88	.78
Constant	-1.47	4.33			
BMI	0.04	0.12	.02		
Baseline DBP	0.97	0.06	.87***		
DBP Reactivity	0.14	0.07	.11*		
Step 2				.88	.78
Constant	-0.83	4.76			
BMI	0.01	0.13	.00		
Baseline DBP	0.98	0.06	.88***		
DBP Reactivity	0.15	0.07	.11*		
Anger Out	-0.11	0.13	-.05		
Recovery Assignment	0.30	0.49	.03		
Step 3				.88	.78
Constant	-3.21	6.34			
BMI	0.01	0.13	.00		
Baseline DBP	0.99	0.06	.88***		
DBP Reactivity	0.15	0.07	.11*		
Anger Out	0.08	0.36	.04		
Recovery Assignment	1.62	2.36	.18		
Anger Out x Recovery	-0.10	0.18	-.17		

Note. *$p < .05$, *** $p < .001$. For Step 3: $F(4, 82) = 2.00$, *ns*.

The fifth hypothesis: "Individuals with greater BP change scores after the math task (cardiovascular reactivity) will have higher resting BP means (average baseline BP) suggesting that greater physiological responses to stress are associated with general hyper-arousal that may lead to hypertension." This final hypothesis was assessed using Pearson's correlations. The fifth hypothesis was not supported. Baseline BP was not significantly and positively related to BP reactivity for baseline SBP and SBP reactivity $r = .20$, $p = .06$ (approached significance), for baseline SBP and DBP reactivity $r = .13$, *ns*, for baseline DBP and DBP reactivity $r = .03$, *ns*, or baseline DBP and SBP reactivity $r = .16$, *ns*. The present study did not demonstrate that greater physiological responses to stress were associated with higher baseline BP, but a trend that approached significance for SBP was revealed suggesting that higher SBP reactors may have higher resting SBP.

Additional Analyses

The association of cardiovascular reactivity and recovery variables was further investigated. The analyses revealed that individuals with high baseline DBP were more likely to also have high baseline SBP, $r = .74$, $p < .001$. Additionally, individuals who showed higher SBP reactivity were also more likely to exhibit greater DBP reactivity, $r = .81$, $p < .001$. The additional analyses also found a significant positive relation among individuals with higher BP reactivity comparing average baseline SBP and average recovery SBP at 10 minutes, $r = .40$, $p < .001$, indicating individuals with higher SBP at rest also had higher SBP recovery readings, and average recovery SBP at 10 minutes and DBP reactivity, $r = .30$, $p < .05$, indicating a relation between DBP reactivity and SBP recovery BP readings. Additionally, higher DBP during the stressor was found to be positively correlated with higher SBP reactivity ($r = .64$, $p < .001$) and DBP reactivity (r

= .66, $p < .001$). Average SBP during the stressor is positively correlated to SBP reactivity ($r = .73$, $p < .001$) and DBP reactivity ($r = .56$, $p < .001$). These results indicate that the hemodynamics of BP dysregulation is a complex and interconnected physiological mechanism.

CHAPTER V

DISCUSSION/CONCLUSIONS

The present study investigated the effectiveness of relaxation techniques at reducing BP after exposure to a stressor with harassment. The results revealed that relaxation techniques can be effective at reducing BP, especially DB on DBP. Additionally, DB demonstrated superior effectiveness at reducing BP in comparison to MR and SC. This study also assessed the role of trait anger coping style on the BP-relaxation technique relation and there were no significant results revealed for the influence of trait anger coping style on BP or trait anger coping style in combination with relaxation techniques on BP.

Anger Coping Style

The lack of significant results for anger coping style is contrary to the findings of previous studies that demonstrated the deleterious effects of maladaptive anger coping styles on one's cardiovascular functioning (Faber & Burns, 1996; Lai & Linden, 1992; Neumann et al., 2004). However, there are also studies, like the current study, that were unable to produce significant effects of BP and anger coping style. For example, Friedman and associates (2001) were unsuccessful in finding relations between anger and BP. Interestingly, Friedman and associates used the same anger scale as the present study. The lack of significant results associated with anger and BP may be explained by the fact that the participants in this study may not have been significantly stressed or angered by the math task stressor with harassment. The participants in this study may have experienced this exercise as a psychological challenge. Kamarck and Lovallo (2003)

defined psychological challenge as situations that are motivationally relevant (i.e. posing negative or positive consequences of importance to the individual) and that require adaptive (cognitive or motoric) responding (Kamarck & Lovallo, 2003). Additionally, the participants demonstrated a restricted range of scoring on the Anger out subscale. The possible range was 8 to 32 and the sample in the current study exhibited scores between 8 and 23, with a range of 8.89 (See Table 2). This is likely an artifact of using advanced graduate students who are known to remain calm during times of stress and may be less likely to respond to anger with angry outbursts.

The studies that have found associations between anger coping style and cardiovascular measures show conflicting results. Some studies show associations with DBP (Lai & Linden, 1992; Neumann et al., 2006; Schwartz et al., 1981), whereas others show anger coping style producing significant results with SBP (Schum et al., 2001). Schum and associates (2001) reviewed multiple studies that found no significant correlations for anger and DBP. Additionally, a number of studies that found significant results for anger variables and cardiovascular responses used anger instigation techniques/tasks or other ways of evaluating individual experiences of anger, as opposed to self-report measures alone. The lack of significant associations between anger coping style and BP may have been a consequence of using a sample of graduate students with a restricted range of responding to the trait anger assessment and in general using a self-report measure which may be confounded by social desirability.

Cardiovascular Reactivity and Recovery

The current study also attempted to show a correlation between resting BP and cardiovascular reactivity in a pursuit to associate cardiovascular hyper-arousal with

higher resting BP readings. There were no significant positive correlations found for cardiovascular reactivity and resting (or baseline) BP. This study did not demonstrate that hyperarousal was associated with high overall BP readings. This result was contrary to reactivity literature that showed an association between cardiovascular reactivity and higher baseline BP (Carroll et al., 2011). Carroll and associates (2011) theorized that large-magnitude cardiovascular reactions to psychological stress played a role in the development of cardiovascular pathology in general and HBP in particular (Carroll et al., 2011; Manuck, et al., 1990). The present study was unable to produce results to support this reactivity hypothesis; however, the sample in the Carroll and associates (2011) study had a mean age of 41.10 (SD = 0.43) years. The current study's mean age was 25.20 (SD = 2.88). The physiological response observed in the Carroll and associates (2011) study may be a phenotype that is not expressed until individuals are older.

There was a significant positive relation among individuals with higher BP reactivity and average baseline SBP in the current study. This result indicated that there was an association between cardiovascular reactivity and SBP at rest. This association was also observed with recovery SBP and baseline SBP, indicating that the amount of time an individual takes to recover from a stressor was associated with generally higher SBP. The significant relation found between recovery SBP and DBP reactivity showed a relation between the time it took individuals to recover from stress and their amount of cardiovascular reactivity. SBP reactivity and DBP reactivity were also strongly and positively related. Average SBP during the stressor was found to be positively and significantly correlated to both SBP reactivity and DBP reactivity. These results reveal the intricate associations of cardiovascular responses during stress and during a recovery

period following a stressor. The physiological responses involve multiple interactions with various mediators and when any one mediator is increased or decreased there are compensatory changes in the other mediators that depend on time course and level of change of each of the mediators (McEwen, 2008). An example of this process was described earlier in the physiological reactions during the fight or flight response, i.e. the production of cortisol and other stress hormones, sympathetic nervous system activity, reduced parasympathetic nervous system activity, insulin and blood glucose production, and elevated BP. The physiological changes that also occur during the relaxation response are just as intricate and complex.

Relaxation Techniques

In an attempt to produce information regarding methods of recovering from a stressor and reducing HBP, this study demonstrated that relaxation techniques are effective at reducing BP. Specifically, DB was found to be significantly more effective at reducing DBP than a SC and MR. In comparing two forms of relaxation techniques, this study was able to elucidate the beneficial effects of deep (diaphragmatic) breathing in reducing BP which likely resulted from a generalized relaxation response in the body. This generalized relaxation response is the goal of relaxation techniques and likely countered the cardiovascular arousal witnessed in the fight-or-flight response which is a consequence of an individual's experience of stress. By practicing relaxation techniques, the purpose is to reduce sympathetic nervous system arousal including the constriction of blood vessels (which would cause an increase in BP), reduce heart rate, and the release of certain hormones, for example epinephrine, norepinephrine, and cortisol which function

to mobilize fuels, including lipids and other body fats; basically to counter the stress response.

The results of the present study add to the literature supporting the effect of stress on cardiovascular recovery, reactivity, and in turn CVD. This study showed that the participants' BP (SBP and DBP) increased significantly from baseline to the stressor. However, relaxation techniques did not reveal a significant reduction in SBP. Given the significant reduction in DBP witnessed in the current study, there appeared to be evidence that DBP was more malleable to the effects of relaxation techniques than SBP. The study of hemodynamics may explain why DB reduced DBP. Hemodynamics is an important part of cardiovascular physiology dealing with the forces the heart develops to circulate blood through the cardiovascular system (Sramek, 2000).

DBP is the pressure in the blood vessels when the heart is at rest and the vasculature decreases during each beat to its lowest level, the diastolic level (Sramek, 2000). Stress caused the vasoconstriction of the cardiac muscles including the blood vessels, which is the reason the participants experienced increases in BP during the math task stressor. Conversely, when a person is engaged in relaxation techniques, their body experiences a generalized relaxation response which causes the blood vessels to relax and results in a reduction in BP (as was evidenced with DB and DBP in the present study). The participants' BP rose as a result of increased vascular resistance. Relaxation techniques countered this resistance via the generalized relaxation response that caused the blood vessels to relax or reduce the constriction on the blood vessels. DBP produced significant results because the blood vessels were less constricted, while the heart was not

pumping during the DB condition. SBP was reduced but not significantly. Given more time engaging in DB, SBP may have shown a significant reduction as well.

The present study's finding of the effectiveness of relaxation techniques at reducing BP gives clarification to the body of literature on relaxation techniques that reveal conflicting results of the effects of relaxation techniques on BP. For example, Parker and associates (1978) found relaxation techniques to be effective at reducing BP. These researchers found that both progressive relaxation training and meditation training produced a reduction in BP from baseline, after 3 weeks of the study. The Parker and associates (1978) study found meditation training to produce superior effects to progressive muscle relaxation. Additionally, Lee and associates (2003) found that SBP and HR decreased with the practice of DB. Grossman and associates (2001) found a significant reduction in DBP and results that approached significance for SBP using a controlled breathing technique. These studies in combination with the current study provide evidence for the effectiveness of relaxation techniques at reducing BP and that DBP may respond more quickly to the practice of relaxation techniques than SBP.

Strengths and Limitations

This study had several strengths and limitations. The strengths of the study included the study's design which revealed BP fluctuations during the various conditions in the experiment. The math task with harassment used as the stressor was effective at increasing BP from baseline to the stressor, which helped to show the effectiveness of relaxation techniques as a tool aiding in BP reduction during cardiovascular recovery after the stressor. The current study also strictly controlled for confounding variables. Participants were not enrolled in the study if they had been taking medications that could

affect their BP and were restricted from caffeine consumption and engaging in exercise, which could have affected the cardiovascular measures as well. An additional strength of this study was that significant results demonstrating the effectiveness of relaxation techniques in reducing BP, indicating that the interventions utilized were successful. Furthermore, the fact that males and females were assessed was an additional strength as some studies only included one gender.

The limitations included the fact that measures of impedance to assess the mechanism described in the previous section (vasoconstriction of blood vessels) were not utilized including peripheral vascular resistance to measure the vascular resistance to the flow of blood in the peripheral arterial vessels (the function of internal vessel diameter) and cardiac output which measures the amount Power of blood pumped by the left ventricle. Measures of myocardial contractility used in the Sinha and associates study (1992), for example pre-ejection period (PEP) (the time interval from the beginning of electrical stimulation of the ventricles to the opening of the aortic valve) and left ventricular ejection time (LVET) (the time interval from opening to closing of the aortic valve) would have provided a more robust explanation of the process evidenced in this study. Another limitation was the power of the regression equations was likely limited given the smaller sample size. Additionally, there was a relatively small proportion of ethnic minorities who participated in the study which limits the generalizability of the study's results. Of the 89 participants in the study, only 34 self-identified as ethnic minorities (38.2%). Further, given the relatively young and healthy sample utilized, the results may need to be generalized with caution to persons diagnosed with CVD and/or

older adults. These two populations are of clinical importance given that ethnic minorities and older adults constitute a large percentage of individuals diagnosed with HBP.

The present study did not demonstrate a relation between anger coping style and BP, which may be a result of using a self-report measure. Due to social desirability, some individuals may not be as truthful about their method of coping with anger. An additional limitation was the lack of manipulation checks used in the study to ensure the participants were engaged in, and experienced the desired effects, of the experimental conditions. A manipulation check was needed to ensure the participants were stressed during the math task stressor. There was also a need for an assessment to monitor whether the participants were actually engaged in DB and MR.

Conclusions and Clinical Implications

In conclusion, a major finding of the present study was that relaxation training was effective at reducing BP after a stressor. Specifically, DB was found to be effective at reducing DBP after a stressor. DB showed a 5 mm Hg reduction in DBP in comparison to the SC. This is a statistically significant reduction but the clinical significance of this reduction is not as clearly determined, especially when considering the definition of clinical significance is "a conclusion that an intervention has an effect of practical meaning to patients and health care providers; even though an intervention is found to have a statistically significant effect, this effect might not be clinically significant" (McGraw-Hill Concise Dictionary of Modern Medicine, 2002). If you describe a person with a normal BP reading of 120/80, a five point increase in DBP (DBP = 85) would put the individual in the prehypertension range and means they are at increased risk for

developing hypertension and other CVDs. Given this information, this is a clinically significant result.

The results of the present study also indicated that higher SBP and DBP reactors are at higher risk of developing CVD because they were more likely to exhibit elevated BP readings during a stressor and at baseline. This information can be useful when treating individuals with hypertension. Relaxation techniques should be considered as an adjunctive treatment of HBP along with hypertensive medication, if not in lieu of such medication as relaxation techniques are cost effective and do not have the side effects associated with medication treatment.

Future directions

Given the societal impact of CVD and that relaxation training demonstrated effectiveness at reducing BP, relaxation techniques should continue to be researched as an adjunctive and eventually a primary treatment of hypertension. This study should be replicated using other physiological measures, i.e. cortisol as the dependent variable. This study can be replicated with the addition of measures of impedance (myocardial contractility (PEP and LVET) and peripheral vascular resistance) to assess the contributing factors of BP changes during the task and recovery conditions. Additionally, high versus low cardiovascular reactors on BP recovery should be assessed. This study should be replicated using a sample of older adults and ethnic minorities. This research can reduce the mortality rate caused by complications of hypertension and could benefit individuals who may experience adverse side effects from antihypertensive medications and/or may not be able to afford the medications usually prescribed to treat hypertension. After all, it costs nothing to slow down and take time to breathe deeply.

REFERENCES

Agras, W. S., Horne, M., Taylor, C. B. (1982). Expectation and the blood-pressure lowering effects of relaxation. *Psychosomatic Medicine, 44*(4), 389-395.

American Heart Association (2007). *African Americans and Cardiovascular Diseases-Statistics.* Retrieved May 30, 2007, from http://www.americanheart.org/downloadable/heart/1168611462350AFAM07.pdf

American Heart Association (2007). *Heart Facts 2007: All Americans/African Americans.* Retrieved May 30, 2007, from http://www.americanheart.org/downloadable/heart/1176927558476AllA AfAm%20HeartFacts07_lores.pdf

American Heart Association (2008). *High blood pressure statistics.* Retrieved July 21, 2009, from http://www.americanheart.org/presenter.jhtml?identifier=4621.

American Heart Association. (2009). *Heart Disease and Stroke Statistics – 2009 Update.* Dallas, Texas: American Heart Association; 2009.

Anderson, N. B., Lane, J. D., Taguchi, F., & Williams, R. B. (1989). Patterns of cardiovascular responses to stress as a function of race and parental hypertension in men. *HealthPsychology, 8*(5), 525-540.

Anderson, N. B., Lane, J. D, Taguchi, F., Williams, R. B., & Houseworth, S. J. (1989). Race, parental history or hypertension, and patterns of cardiovascular reactivity in women. *Psychophysiology, 26*(1), 39-47.

Armstead, C. A., Lawler, K. A., Gorden, G., Cross, J., & Gibbons, J. (1989). Relationship of racial stressors to blood pressure responses and anger expression in black college students. *Health Psychology, 8*(5), 541-556.

Barnes, V. A., Davis, H. C., Murzynowski, J. B., & Treiber, F. A. (2004). Impact of meditation on resting and ambulatory blood pressure and heart rate in youth. *PsychosomaticMedicine, 66,* 909-914.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*, 1173-1182.

Bishop, G. D., Pek, J., & Ngau, F. (2005). Ethnicity, sex, trait anger, and nocturnal blood pressure. *Psychophysiology, 42,* 290-297.

Bleil. M. A., Gianaros, P. J., Jennings, J. R., Flory, J. D. & Manuck, S. B. (2008). Trait negative affect: Toward an integrated model of understanding psychological risk for impairment in cardiac autonomic function. *Psychosomatic Medicine, 70*, 328 -337.

Brondolo, E., Brady ver Halen, N., Pencille, M., Beatty, D., & Contrada, R. J. (2009). Copingwith racism: a selective review of the literature and a theoretical and methodological critique. *Journal of Behavioral Medicine, 32*, 64-88.

Brosschot, J. F., & Thayer, J. F. (2003). Heart rate response is longer after negative emotions than after positive emotions. *International Journal of Psychophysiology, 50*, 181-187.

Bruehl, S., Chung, O. K., & Burns, J. W. (2006). Anger expression and pain: An overview of findings and possible mechanism. *Journal of Behavioral Medicine, 29*(6), 593-606.

Bruning, N. S., & Frew, D. R. (1987). Effects of exercise, relaxation, and management skills training on physiological stress indicators: A field experiment. *Journal of Applied Psychology, 72*(4), 515-521.

Carroll, D., Phillips, A. C., Der, G., Hunt, K., & Benseval, M. (2011). Blood pressure reactions to acute mental stress and future blood pressure status: Data from the 12-year follow-up of the West Scotland study. *Psychosomatic Medicine, 73*, 737-742.

Centers for Disease Control and Prevention. (2011). *High blood pressure facts*. Retrieved April 28, 2012 from www.cdc.gov/bloodpressure/facts.htm.

Centers for Disease Control and Prevention. (2012). Centers for Disease Control and Prevention. CDC Features - May is High Blood Pressure Education Month. Retrieved June 5, 2012 from http://www.cdc.gov/Features/HighBloodPressure/

Chafin, S., Roy, M., Gerin, W., & Christenfeld, N. (2004). Music can facilitate blood pressure recovery from stress. *British Journal of Health Psychology, 9*, 393-403.

Cheung, B. M. Y., Lo, J. L. F., Fong, D. Y. T., Chan, M. Y., Wong, S. H. T., Wong, V. C. W., Lam, K. S. L., Lau, C. P., & Karlberg, J. P. E. (2005). Randomized controlled trial of qigong in the treatment of mild essential hypertension. *Journal of Human Hypertension, 19,* 697-704.

Chobanian, A. V., Bakris, G. L., Black, H. R., Cushman, W. C., Green, L. A., Izzo, J. L., et al. (2003). Seventh report of the joint national committee on prevention,

detection, evaluation, and treatment of high blood pressure. *Hypertension, 42,* 1206-1252.

Christenfeld, N., Glynn, L. M., & Gerin, W. (2000). On the reliable assessment of cardiovascular recovery: An application of curve-fitting techniques. *Psychophysiology, 37,* 543-550.

Clinical significance. (2002) *McGraw-Hill concise dictionary of modern medicine.* Retrieved May 20, 2012 from http://medical dictionary.thefreedictionary.com/clinical+significance.

Cohen, J., Cohen, P., West, G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences: 3rd Edition.* Mahwah, New Jersey: Lawrence Erlbaum Associates, Publisher.

Davich, V. N. (1998). *The best guide to meditation.* New York: Renaissance Media, Inc.

Davidson, K., MacGregor, M. W., Stuhr, J., & Gidron, Y. (1999). Increasing constructive anger verbal behavior decreases resting blood pressure: A secondary analysis of a randomized controlled hostility intervention. *International Journal of Behavioral Medicine, 6*(3), 268-278.

Diaphragmatic breathing (2009, October 26). In *Wikipedia, The Free Encyclopedia.* Retrieved December 7, 2009 from http://en.wikipedia.org/wiki/Diaphragmatic_breathing.

Dorr, N., Brosschot, J. F., Sollers, J. J., & Thayer, J. F. (2007). Damned if you do, damned if you don't: The differential effect of expression and inhibition of anger on cardiovascular recovery in Black and White males. *International Journal of Psychophysiology, 66,* 125-134.

Drummond, P., & Quah, S. H. (2001). The effect of expressing anger on cardiovascular reactivity and facial blood flow in Chinese and Caucasians. *Psychophysiology, 38*, 190-196.

Durel, L. A., Carver, C. S., Spitzer, S. B., Llabre, M. M., Weintraub, J. K., Saab, P. G., & Schneiderman, N. (1989). Associations of blood pressure with self-report measures of anger and hostility among black and white men and women. *Health Psychology, 8*(5), 557-575.

Engebretson, T. O., & Matthews, K. A., Scheier, M. F. (1989). Relations between anger expression and cardiovascular reactivity: Reconciling inconsistent findings through a matching hypothesis. *Journal of Personality and Social Psychology, 57*(3), 513-521.

Engebetson, T.O. & Stoney, C.M. (1995). Anger expression and lipid concentrations. *International Journal of Behavioral Medicine, 2*(4), 281-298.

English, E. H., & Baker, T. B. (1983). Relaxation training and cardiovascular response to experimental stressors. *Health Psychology, 2*(3), 239-259.

Faber. S. D., & Burns, J. W. (1996). Anger management style, degree of expressed anger, and gender influence cardiovascular recovery from interpersonal harassment. *Journal of Behavioral Medicine, 19*, 31-53.

Fang, C. Y., & Myers, H. F. (2001). The effects of racial stressors and hostility on cardiovascular reactivity in African American and Caucasian men. *Health Psychology, 20*(1), 64-70.

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. Behavior Research Methods, *39*, 175-191.

Faulker, B., & Kushner, H. (1989). Race differences in stress-induced reactivity in young adults. *Health Psychology, 8*(5), 613-627.

Finney, M. L., Stoney, C. M., & Engebretson, T. O. (2002). Hostility and anger expression in African Americans and European American men is associated with cardiovascular and lipid reactivity. *Psychophysiology, 39*, 340-349.

Friedman, R., Schwartz, J. E., Schnall, P. L., Landsbergis, P. A., Pieper, C., Gerin, W., & Pickering, T. G. (2001). Psychological variables in hypertension: Relationship to casual or ambulatory blood pressure in men. *Psychosomatic Medicine, 63,* 19-31.

Gandelman, G. (2006, July 20). *Atherosclerosis health article.* Retrieved May 30, 2010, from http://www.healthline.com/adamcontent/atherosclerosis?utm_term=atherosclerosis&utm.

Geller, J., Cockell, S. J., & Goldner, E. M. (2000). Inhibited expression of negative emotions and interpersonal orientation in anorexia nervosa. *Internal Journal of Eating Disorders, 28*(1), 8-19.

Gianfranco, P. & Steptoe, A. (2004). Stress reduction and blood pressure control in hypertension: a role from transcendental meditation? *Journal of Hypertension, 22*, 2057- 2060.

Goldstein, H. S., Edelberg, R., Meier, C. F., & Davis, L. (1988). Relationship to resting blood pressure and heart rate to experienced anger and expressed anger. *PsychosomaticMedicine, 50,* 321-329.

Goldstein, I. B., Shapiro, D., & Thananopavaran, C. (1984). Home relaxation techniques for essential hypertension. *Psychosomatic Medicine*, *46*(5), 398-414.

Grossman, E., Grossman, A., Schein, M. H., Zimlichman, R., & Gavish, B. (2001) Breathing control lowers blood pressure. *Journal of Human Hypertension, 15,* 253-269.

Holmes, D. S., Solomon, S., Cappo, B. M., & Greenberg, J. L. (1983). Effects of transcendental meditation versus resting on physiological and subjective arousal. *Journal of Personalityand Social Psychology, 44*(6), *1245-1252.*

Jayadeveappa, R., Johnson, J. C., Bloom, B. S., Nidich, S., Desai, S. Chhatre, S., Raziano, D. B., & Schneider, R. (2007). Effectiveness of transcendental meditation of functional capacity and quality of life of African Americans with congestive heart failure: A randomized control study. *Ethnicity & Disease, 17,* 72 -77.

Johnson, E. J. & Broman, C.L. (1987). The relationship of anger expression to health problems among African American Americans in a national survey. *Journal of Behavioral Medicine, 10*(2), 103-116.

Johnson, E. H. & Spielberger, C. D. (1992). Assessment of the experience, expression and control of anger in hypertension research. In Johnson, E. H., Gentry, W. D., & Julius, S. (Eds.), *Personality, elevated blood pressure, and essential hypertension* (3-24). Washington, D. C.: Hemisphere Publishing Corporation.

Jorgensen, R. S., Johnson, B. T., Kolodziej, M. E., & Schreer, G. E. (1996). Elevated blood pressure and personality: A meta-analytic review. *Psychological Bulletin, 120*(2), 293-320.

Kamarck, T. W., Debski, T. T., Manuck, S. B. (2000). Enhancing the laboratory-to-life generalizabiltiy of cardiovascular reactivity using multiple occasions of measurement. *Psychophysiology, 37,* 533-542.

Kamarck, T. W., & Lovallo, W. R. (2003). Cardiovascular reactivity to psychological challenge: Conceptual and measurement considerations. *Psychosomatic Medicine, 65*, 9 – 21.

Keller, D. (2007). Diaphragmatic Breath. In *Refining the Breath: The Yogic Practice of Pranayama 4th Ed., 30*-39. Retrieved November 10, 2008 from http://www.doyoga.com/articles_07/web_site_articles/breath/diaphragmatic_breath.pdf.

Kelsey, R. M., Ornduff, S. R., Alpert, B. S. (2007). Reliability of cardiovascular reactivity to stress: Internal consistency. *Psychophysiology, 44,* 216-225.

Lai, J. Y., & Linden, W. (1992). Gender, anger expression style, and opportunity for anger release determine cardiovascular reaction to and recovery from anger provocation. *Psychosomatic Medicine, 54*, 297-310.

Lambert, K. & Kinsley, C. H. (2005). *Clinical neuroscience: The neurobiological foundations of mental health.* New York, NY: Worth Publishers.

Lee, J. S., Lee, M. S., Lee, J. Y., Cornelissen, G., Otsuka, K., & Halberg, F. (2003). Effects of diaphragmatic breathing on ambulatory blood pressure and heart rate. *Biomedicine & Pharmacotherapy, 57*, 87-91.

Lehnert, H., Kaluza, K., Vetter, H., Losse, H., & Dorst, K. (1987). Long-term effects of a complex behavioral treatment of essential hypertension. *Psychosomatic Medicine, 49,* 422-430.

Linden, W., Earle, T. L., Gerin, W., Christenfeld, N. (1997). Physiological stress reactivity and recovery: Conceptual siblings separated at birth? *Journal of Psychosomatic Research, 42*(2), 117-135.

Manuck, S. B., Kasprowicz, A. L., Monroe, S. M. Larking, K. T., & Kaplan, J. R. (1989). Psychophyiologic reactivity as a dimension of individual differences. In Schneiderman, N., Weiss, S. M., & Kaufmann, P. G. (1989). *Handbook of research methods in cardiovascular behavioral medicine.* New York: Plenum Press.

Manuck, S. B., Kasprowicz, A. I., & Muldoon, M. F. (1990). Behaviorally-evoked cardiovascular reactivity and hypertension: conceptual issues and potential associations. Annals of Behavioral Medicine *12*, 17–29.

McEwen, B. S. (2008). Central effects of stress hormones in health disease: Understanding the protective and damaging effects of stress and stress mediators. *European Journal of Pharmacology, 583*, 174-185.

McGrady, A., &. Roberts, G. (1992). Racial differences in the relaxation response of hypertensives. *Psychosomatic Medicine, 54*, 71-78.

McNeilly, M. D., Robinson, E. L., Anderson, N. B., Pieper, C. F., Shah, A., Toth, P. S., et al. (1995). Effects of racist provocation and social support on cardiovascular reactivity in African American women. *International Journal of Behavioral Medicine, 2*(4), 321-338.

MedlinePlus. (2010). *Hypertension*. MedlinePlus Medical Encyclopedia. Retrieved on February 14, 2010 from www.nlm.nih.gov/medlineplus/print/ency/article/000468.htm.

Merrium-Webster, Incorporated. (2009). Anger. *Merrium-Webster Online Dictionary*. Retrieved May 1, 2009 from http://www.m-w.com.

Neumann, S. A., Brown, J. R. P., Waldstein, S. R., & Katzel, L. I. (2006). A walking program's attenuation of cardiovascular reactivity in older adults with silent myocardial ischemia. *Journal of Aging and Physical Activity, 14*, 115-128.

Neumann, S.A., Waldstein, S.R., Sollers, J.J. III, Thayer, J.F., & Sorkin, J.D. (2004). Hostility and distraction have differential influences on cardiovascular recovery from anger recall in women. *Health Psychology, 23*(6), 631-640.

Parati, G., & Steptoe, A. (2004). Stress reduction and blood pressure control in hypertension: A role in transcendental meditation? *Journal of Hypertension, 22,* 2057-2060.

Parker, J. C., Gibert, G. S., & Thoreson, R. W. (1978). Reduction of autonomic arousal in alcoholics: A comparison of relaxation and meditation techniques. *Journal of Consulting and Clinical Psychology, 46*(5), 879-886.

Paul-Labrador, M., Polk, D., Dwyer, J. H., Velasquez, I., Nidich, S., Rainforth, M., et al. (2006). Effects of a randomized controlled trial of transcendental meditation on components ofthe metabolic syndrome in subjects with coronary heart disease. *Archives of Internal Medicine, 166,* 1218-1224.

Ostchega, Y., Yoon, S. S., Hughes, J., Louis, T. (2008). Hypertension awareness, treatment, and control -- continued disparities in adults: United States, 2005

-2006. *NCHS data brief.* Hyattsville, MD: National Center for Health Statistics.

Rapp, D. A., Brenes, G. A., Feldman, S. R., Fleischer Jr., A. B., Graham, G. F., Dailey, M., et al.. (2004). Anger and acne: Implications for quality of life, patient satisfaction and clinical care [Abstract]. *British Journal of Dermatology, 151*(1), 183-189.

Redmond, D. P., Gaylor, M. S., McDonald, R. H., & Shapiro, A. P. (1974). Blood pressure and heart-rate response to verbal instruction and relaxation in hypertension. *Psychosomatic Medicine, 36*(4), 285-297.

Richards, J. C., Alvarenga, M., Hof, A. (2000). Serum lipids and their relationships with hostility and angry affect and behaviors in men. *Health Psychology, 19*(4), 393 398.

Roberts, G., & McGrady, A. (1996). Racial and gender effects on the relaxation response: Implications for the development of hypertension. *Biofeedback and Self-Regulation, 21*(1), 51-62.

Schachter, J. (1957). Pain, fear, and anger in hypertensives and normotensives: A psychophysiological study. Psychosomatic Medicine, *19*, 17-29.

Schein, M. H., Gavish, B., Herz, M., Rosner-Kahana, D., Naveh, P., Knishkowy, B., et al. (2001). Treating hypertension with a device that slows and regularizes breathing: a randomized, double-blind controlled study. *Journal of Human Hypertension, 15,* 271-278.

Schum, J. L., Jorgensen, R. S., Verhaeghen, P., Sauro, M., & Thibodeau, R. (2003). Trait anger, anger expression, and ambulatory blood pressure: A meta-analytic review. *Journal of Behavioral Medicine, 26*(5), 395-415.

Schwartz, G. E., Weinberger, D. A., Singer, J. A. (1981). Cardiovascular differences of happiness, sadness, anger, and fear following imagery and exercise. *Psychosomatic Medicine, 43*(4), 343-364.

Seer, P. (1979). Psychological control of essential hypertension: Review of the literature and methodological critique. *Psychological Bulletin, 86*(5), 1015-1043.

Siegman, A. W. (1994). From type A to hostility to anger: Reflections on the history of coronary-prone behavior. In Siegman, A. W. & Smith, T. W. (Eds.). *Anger, hostility, and the heart.* (pp. 1-21). Hillsdale, NJ: Lawrence Erlbaum Associates, Publishers.

Shapiro, D., Goldstein, I. B., Jamner, L. D. (1995). Effects of anger/hostility, defensiveness, gender, and family history of hypertension on cardiovascular reactivity. *Psychophysiology, 32*, 425-435.

Sinha, R., Lovallo, W. R., & Parsons, O. A. (1992). Cardiovascular differentiation of emotions. *Psychosomatic Medicine, 54,* 422–435.

Spielberger, C. D. (1988). *Manual for the State-Trait Anger Expression Inventory (STAXI).* Odessa, FL: Psychological Assessment Resources.

Spielberger, C. D., Krasner, S. S., and Solomon, E. P. (1988). The experience expression, and control of anger. In Janisse, M.P. (Ed). *Individual differences, stress, and health psychology* (pp. 89-108). New York, New York: Springer Verlag.

Spielberger, C. D., Reheiser, E. C., & Sydeman, S. J. (1995). Measuring the experience, expression, and control of anger. In Kassinove, H (Ed). *Anger disorders: Definition, diagnosis, and treatment* (pp. 49-68). Washington, D.C.: Taylor & Francis.

Sramek, B. B. (2000). Hemodynamics for seniors. *International Hemodynamic Society.* Retrieved June 8, 2012 from http://www.hemodynamicsociety.org/hseniors.html.

Starner, T. M, & Peters, R. M. (2004). Anger expression and blood pressure in adolescents. *The Journal of School Nursing, 20*(6), 335-342.

Stewart, J. C., & France, C. R. (2001). Cardiovascular recovery from stress predicts longitudinal changes in blood pressure. *Biological Psychology, 58*, 105-120.

Stewart, J. C., Janicki, D. L., & Kamarck, T. W. (2006). Cardiovascular reactivity to and recovery from psychological challenge as predictors of 3-year change in blood pressure. *Health Psychology, 25*(1), 111-118.

Suinn, R.M. (2001). The terrible twos-anger and anxiety: Hazardous to your health. *American Psychologist, 56*(1), 27-36.

Suls, J., & Bunde, J. (2005). Anger, anxiety, and depression as risk factors for cardiovascular disease: The problems and implications of overlapping affective dispositions. *Psychological Bulletin, 131*(2), 260-300.

Trivedi, R., Sherwood, A., Strauman, T. J., & Blumenthal, J. A. (2008). Laboratory-based blood pressure recovery is a predictor of ambulatory blood pressure. *Biological Psychology, 77,* 317-323.

Van De Graaff, K. (1998). *Human anatomy* (5th ed.). Boston: McGraw-Hill.

Vander, A., Sherman, J., Luciano, D. (1998). *Human physiology: The mechanisms of body function.* New York: McGraw-Hill.

Vella, E. J. & Friedman, B. H. (2009). Hostility and anger in: Cardiovascular reactivity and recovery to mental arithmetic stress. *International Journal of Psychophysiology, 72*, 253-259.

Venable, V. L., Carlson, C. R., & Wilson, J. (2001). The role of anger and depression in recurrent headache. Headache*: The Journal of Head and Face Pain*, 41(1), 21-30.

Vitaliano, P. P., Russo, J., Paulsen, V. M., & Bailey, S. L. (1995). Cardiovascular recovery from laboratory stress: Biopsychosocial concomitants in older adults. *Journal of Psychosomatic Research, 39*(3), 361-377.

Wallace, R. K., Silver, J., Mills, P. J., Dillbeck, M. C., & Wagoner, D. E. (). Systolic blood pressure and long-term practice of the Transcendental Meditation and TM Sidhi program: Effects of TM on systolic blood pressure. *Psychosomatic Medicine, 45*(1), 41-46.

APPENDIX A

PHONE SCREEN

Name: _____

To be read to subject before screening questions: *We are calling from Eastern Virginia Medical School regarding the research study that you expressed interest in from the email advertisement sent to you on _____. The study proposes to establish a better understanding of effects of stress on psychological and physical health. This information is being asked to determine your eligibility in our research study and will not be used other than for the purposes of this research study. If you decide not to participate or for some reason are ineligible to participate this information will be destroyed. Does this sound like something you would like to participate in?* (If no, thank the person for their time. If yes, ask the following screening questions. If the potential subject is determined ineligible for any of the screening criteria below, please provide the explanation for their ineligibility presented in italics below each screening question.)

1. How old are you?_____
If not between the ages of 20-35:
[I am sorry at this time we are looking for subjects between the ages of 20-35, but thank you for your time and interest.]

2. How would you describe your ethnic background?_____

3. Do you currently smoke or use tobacco? Y N
If yes:
[I am sorry, we are unable to have people who use tobacco because smoking can affect measures that we are collecting, but we thank you for your time and interest.]

4. Do you have any assistive devices such as a pacemaker? Y N
[I am sorry, we are unable to have people with any electrical assistive devices such as yours, because it can send a false signal when measuring heart rate, but we thank you for your time and interest]

5. Do you have hypertension/high blood pressure (≥140/90 mm Hg)? Y N
If yes:
[I am sorry, we are unable to have people who have hypertension because it can affect measures that we are collecting, but we thank you for your time and interest.]

6. Have you ever been diagnosed with cancer? Y N
   Within the last year? Y N

   Were you under any treatment for cancer? Y N
   Within the last year? Y N

If treatment has been received within the past year:
[Treatments for cancer can often interfere with the measures we are taking, so we are unable to have you participate in the study, but thank you for your time and interest.]

7. Have you ever been diagnosed with a neurological illness (i.e., Multiple Sclerosis)?
Y N
If yes:
[Certain illnesses and diseases may interfere with nerve conduction in some of the measures we are collecting, so we are unable to have you participate in the study, but thank you for your time and interest.]

8. Have you ever been diagnosed with Kidney or Liver Disease? Y N
Do you receive or require dialysis? Y N

If the person requires dialysis:
[Certain illnesses and diseases may interfere with nerve conduction in some of the measures we are collecting, so we are unable to have you participate in the study, but thank you for your time and interest.]

9. Have you ever had a stroke or TIA (Transient ischemic attack, or "mini stroke")?
Y N Date:_____
If yes:
[I am sorry, we are unable to have people who have had a stroke previously because a stroke can affect measures that we are collecting, but we thank you for your time and interest.]

10. Have you ever been diagnosed with any type of cardiovascular or heart disease? Y N
If yes:
[I am sorry, we are unable to have people who have heart disease because it can affect measures that we are collecting, but we thank you for your time and interest.]

11. Do you have any tumors of the brain? Y N
If yes:
[I am sorry, we are unable to have people who have a brain tumor because its presence can affect measures that we are collecting, but we thank you for your time and interest.]

12. How long have you been speaking English? _____
If English is not used in everyday speaking and reading for at least 10 years:
[I am sorry, we are unable to have people who have been speaking English for less than 10 years due to the complexity of the questionnaires, but we thank you for your time and interest.]

13. On average about how many alcoholic beverages do you consume in a week?
(One beverage is equal to a 12oz. beer, 5 oz. Wine, or 1.5 oz of hard liquor) _____

If more than 21 alcoholic beverages are consumed per week:
[I am sorry, we are unable to have people who drink more than 21 alcoholic beverages per week, because it could affect measures that we are collecting, but we thank you for your time and interest.]

14. Do you use any illicit drugs? Y N
    If yes, which type? _____
    If yes, how often? _____

If any illicit drugs are used more than once a month:
[I am sorry, we are unable to have people participate who use illicit drugs with a greater frequency than once a month, because it could affect measures that we are collecting, but we thank you for your time and interest.]

15. Are you currently taking any medication? Y N
    Which ones? _____

    Are you taking any Cough/Cold/Allergy Medications (Ex: Tylenol Cold, Actifed, Benadryl, Sudafed, Robitussin)? Y N

    Are you taking any Weight Loss Aids (Ex: Dexatrim, Orlistat, Adipex, Ephedra, Metabolife)? Y N

    Are you taking any Prescription Muscle Relaxants (Ex: Flexoril, Skelaxin)?
    Y N

    Are you taking any Sleeping Pills (Ex: Restoril, Sonata, Ambien)? Y N

    Are you taking any Narcotic Pain Pills (Ex: Percocet, Oxycontin, Ultram)? Y
    N

If yes to any of the specific medication questions:
[Some medications can interfere with the measures we are taking, so we are unable to have you participate in the study, but thank you for your time and interest.]

16. Have you ever been diagnosed with a psychiatric illness? Y N
    Which one(s)? _____
    When? _____
    Were you given any medications for treatment? Y N
    Which ones? _____

If diagnosed or in treatment for certain mood disorders (e.g. depression, psychotic disorders, Schizophrenia, bipolar disorder):

[Unfortunately we are unable to accept people who suffer from these diseases due to the possible interference in the measures we collect during the study, but we thank you for your time and interest.]

17. Have you ever had a concussion or head injury?
How many? _____
When? _____

If yes:
[I am sorry, we are unable to have people who have had a concussion or head injury previously because it can affect measures that we are collecting, but we thank you for your time and interest.]

18. Do you have any prior experience with standardized relaxation training or meditation? Y N

If yes:
[I am sorry, we are unable to have people who have had prior experience with standardized relaxation training, but we thank you for your time and interest.]

Is subject eligible? Y N

Yes: *Well, Mr./Ms. _____ it appears you would be a good candidate for this study. Would you be able to come in for your visit on_____ at_____ ? The visit takes about 90 to 120 minutes to complete, so please wear comfortable clothes. You will come into our office and be given informed consent, which details the study. After reviewing the consent, you can determine if the study is right for you. You will then fill out several questionnaires and complete several tests.*

In preparation for the study, we ask that you refrain from caffeine for four hours, and abstain from over-the-counter medications such as Sudafed for 24 hours, as these may affect assessment.

Can I have your contact information?
Phone #: _____ (cell) _____ (work) _____ (home)
(Circle the one that is the participant's preferred number.)

Email address: _____

Home address:_____
_____
_____
_____

If you have any questions prior to your visit, please feel free to call the lab at _____.

Criteria:

a) Report no clinical history of severe and chronic diseases affecting general health (treatment for cancer [within the past year], hypertension (typical blood pressure < 140/90 mm Hg), current smoking or tobacco use) neurological disorders [e.g., multiple sclerosis], chronic kidney or liver disease (end stage renal disease requiring dialysis), stroke or cerebrovascular events or disease, or brain tumors)

b) Be fluent in English

c) Report consumption of fewer than 21 alcoholic beverages per week

d) Deny current substance dependence or use of an illicit substance on more than a monthly basis

e) Deny use of certain medications (e.g., cough/cold/allergy medications, weight loss aids, prescription muscle relaxants, sleeping pills, and/or narcotic pain pills)

f) Report no diagnosis or treatment for certain mood disorders (e.g. depression, psychotic disorders, Schizophrenia, bipolar disorder)

g) Deny a history of multiple head injuries, traumatic brain injury or multiple losses of consciousness

h) Deny a history of experience with standardized relaxation training techniques

APPENDIX B

BIOLOGICAL MEASURES FORM

Height: _____'_____"

Weight of Clothes: _____ lbs. (winter clothes 4 lbs; summer clothes 2 lbs)

Hours/Week Exercise on Average: _____ hours

Years Exercising at this Level: _____ years

Male Female

Athletic Standard

Age: _____ years

Weight: _____ lbs.

Body Mass Index: _____

Body Fat % _____

Basal Metabolic Rate: _____

Fat Free Mass: _____

Total Body Water: _____

Waist: _____ inches

Hips: _____ inches

APPENDIX C

DEMOGRAPHICS AND MEDICAL HISTORY FORM

Please answer the following questions to the best of your ability and remember you do not have to answer any question you do not want to.

1a. Sex:

(1) Male
(2) Female

2a. Age: _____

3a. Marital Status:

(1) Married or living with partner
(2) Widowed
(3) Divorced or annulled
(4) Separated
(5) Single; never married

4a. Racial categories:

(1) Caucasian, white
(2) African American, black
(3) Asian
(4) Latino or Hispanic
(5) Native American
(6) Bi- or multiracial, *please specify:* _____
(6) Other, *please specify:* _____

5. Number of biological parents with medical history of hypertension:

(0)
(1)
(2)

6. Do you have or have you had any of the following? (PLEASE CHECK ONE)

	YES (1)	**NO (0)**	**DON'T KNOW (-3)**
Heart or Blood Vessels			
1. High blood pressure (hypertension)	( )	( )	( )
2. Heart attack (myocardial infarction,	( )	( )	( )

coronary occlusion or coronary thrombosis)			
3. Angina (chest pain brought on by exertion)	( )	( )	( )
4. Heart bypass surgery	( )	( )	( )
5. Heart balloon angioplasty, stent	( )	( )	( )
6. Congestive heart failure	( )	( )	( )
7. Valve problems (heart murmur or leaky valve)	( )	( )	( )
8. Heart pacemaker	( )	( )	( )
9. Rheumatic fever with involvement of heart	( )	( )	( )
10. Abnormal heart rhythm (e.g. atrial fibrillation)	( )	( )	( )
11. Other (Specify: _____)	( )	( )	( )
Brain			
12. Stroke (cerebrovascular accident)	( )	( )	( )
13. TIA or transient ischemic attack (brief stroke that completely resolved within 24 hours)	( )	( )	( )
14. Convulsions, fits or seizures, epilepsy	( )	( )	( )
15. Head injury or concussion or spinal cord injury, how many?	( )	( )	( )
16. Loss of consciousness, how long?	( )	( )	( )
17. Parkinson's disease, Multiple Sclerosis or Muscular Dystrophy	( )	( )	( )
18. Other (Specify:_____)	( )	( )	( )

Other Diseases	YES (1)	NO (0)	DON'T KNOW (-3)
19. Diabetes (high blood or urine sugar)	( )	( )	( )
20. Kidney problems (nephritis, kidney infection, kidney stones)	( )	( )	( )
21. Surgery (type: _____)	( )	( )	( )
22. Cancer (site:_____)	( )	( )	( )
23. Asthma	( )	( )	( )
24. Other lung problems (TB, emphysema, pleurisy, chronic bronchitis, or other problems)	( )	( )	( )
25. Thyroid condition	( )	( )	( )
26. Liver disease (hepatitis, cirrhosis or other problems)	( )	( )	( )
27. Arthritis	( )	( )	( )
28. Hives, hay fever, or other allergies	( )	( )	( )
29. Other major disease (specify: _____)	( )	( )	( )
30. Have you ever been hospitalized?	( )	( )	( )

Please indicate whether the following are true:	YES (1)	NO (0)
31. Full use of arms, hands, and fingers	( )	( )
32. Full use of legs and feet	( )	( )
33. Good eyesight (when wearing glasses or contact lens)	( )	( )

APPENDIX D

SCRIPTS FOR RECOVERY EXERCISES

1. Standard (Control) Recovery

   "Please rest quietly without moving around too much until you are instructed to stop."

   [Research team member will time this for 10 minutes.]

2. Diaphragmatic Breathing

   "Place one hand on your chest and the other on your abdomen. When you take a deep breath in, the hand on the abdomen should rise higher than the one on the chest. This ensures that the diaphragm is pulling air deeply into the lungs."

   "After exhaling through the mouth, take a slow deep breathe in through your nose for a count of 5 and pause for a second (or as long as you are able, not exceeding 5)."

   "Slowly exhale through your mouth blowing the air out of your mouth for a count of 7. As all the air is released, gently contract your stomach to completely expel the remaining air from your lungs."

Adapted from Davis, Eshelman, & McKay (2000).

[You should see the subject adequately demonstrating this technique with the abdomen contracting during the exhalation and expanding during the inhalation before continuing with recovery.]

"Repeat the cycle until you are instructed to stop."

[After reading these instructions, double-click the icon on desktop labeled "Diaphragmatic Breathing" (a Windows Media Player audio file located in the N: drive, "Post-Stress Recovery Study" folder, "Working Protocol" folder.). This will open up in Windows Media Player automatically. Make sure that the "Repeat" feature is selected. Time this task for 10 minutes and press the "Stop" button when the exercise is over.]

3. Mantra Recitation

"Do the following until you are instructed to stop:

Settle down in a comfortable posture with your eyes closed.

Say "Peace" silently in your consciousness. Then begin repeating it. Start slowly, speaking to yourself silently as distinctly as possible. You will naturally and gradually increase the speed of repetitions.

Stop as needed. As you recite your mantra, you may find that you relax into the sound.

Repeat your mantra silently until you are instructed to stop."

[Observe the participant performing mantra recitation for 10 minutes once instructions are given and participant has begun.]

Adapted from Davich (1998).

APPENDIX E

SPIELBERGER STATE-TRAIT ANGER EXPRESSION INVENTORY (STAXI)

Part I

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 generally feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

	Almost Never	Sometimes	Often	Almost Always
Generally,				
1. I am quick tempered.....	1	2	3	4
2. I have a fiery temper.....	1	2	3	4
3. I am a hotheaded person.....	1	2	3	4
4. I get angry when I'm slowed by others' mistakes.....	1	2	3	4
5. I feel annoyed when I am not given recognition for doing good work.....	1	2	3	4
6. I fly off the handle.....	1	2	3	4
7. When I get mad, I say nasty things..	1	2	3	4
8. It makes me furious when I am criticized in front of others.....	1	2	3	4
9. When I get frustrated, I feel like hitting someone.....	1	2	3	4
10. I feel infuriated when I do a good job and get a poor evaluation.....	1	2	3	4

Part II

Everyone feels angry or furious from time to time, but people differ in the ways that they react when they are angry. A number of statements are listed below which people use to describe their reactions when they feel angry or furious. Read each statement and then circle the number to the right which indicated how often you generally react or behave in the manner described when you are feeling angry or furious. Remember that there are no right or wrong answers. Do not spend too much time on any one statement.

	Almost Never	Sometimes	Often	Almost Always
Generally,				
11. I control my temper.....	1	2	3	4
12. I express my anger.....	1	2	3	4
13. I keep things in.....	1	2	3	4
14. I am patient with others.....	1	2	3	4
15. I pout or sulk.....	1	2	3	4
16. I withdraw from people.....	1	2	3	4
17. I make sarcastic remarks to others...	1	2	3	4
18. I keep my cool.....	1	2	3	4
19. I do thinks like slam doors.....	1	2	3	4
20. I boil inside, but I don't show it.....	1	2	3	4
21. I control my behavior.....	1	2	3	4
22. I argue with others.....	1	2	3	4

	Almost Never	Sometimes	Often	Almost Always
23. I tend to harbor grudges that I don't tell anyone about.....	1	2	3	4
24. I strike out at whatever infuriates.....	1	2	3	4
25. I can stop myself from losing my temper	1	2	3	4
26. I am secretly quite critical of others...	1	2	3	4
27. I am angrier than I am willing to admit..	1	2	3	4
28. I calm down faster than most other people	1	2	3	4
29. I say nasty things.....	1	2	3	4
30. I try to be tolerant and understanding...	1	2	3	4
31. I'm irritated a great deal more than people are aware of.....	1	2	3	4
32. I lose my temper.....	1	2	3	4
33. If someone annoys me, I'm apt to tell him or her how I feel.....	1	2	3	4
34. I control my angry feelings.....	1	2	3	4

APPENDIX F

FLYER

EVMS Students NEEDED!
RESEARCH STUDY

Volunteers who are **ages 20-35** are being recruited to participate in a research study examining the effects of stress on health.

Participants will be compensated.

Please call **757-446-5712** for more information or to schedule an appointment.

Psychophysiologic Evaluation and Treatment Laboratory (PETL)
Department of Psychiatry and Behavioral Sciences
Eastern Virginia Medical School
825 Fairfax Avenue, 734 Hofheimer Hall
Norfolk, VA 23507

This advertisement has been approved by an Eastern Virginia Medical School Institutional Review Board.
(IRB #09-01-EX-0014)

VITA

Virginia Consortium
Administrative Office
VBHEC/Suite 239
1881 University Drive
Virginia Beach, VA 23453

Catherine J. Mills was born in Brooklyn, New York. She graduated from Hillcrest High School with honors with a Pre-Medical diploma. Catherine then earned a B.A. degree in Biology and Psychology from the University of North Carolina at Greensboro. She was awarded her M.A. in Clinical Psychology in 2008 from North Carolina Central University. She expects to receive her Psy.D. from the Virginia Consortium Program in Clinical Psychology in 2012.