

THE EFFECTS OF ACUTE AEROBIC EXERCISE
ON CARDIOVASCULAR REACTIVITY IN RESPONSE
TO PSYCHOLOGICAL STRESS IN TRAINED CYCLISTS

by

Susi Palmer

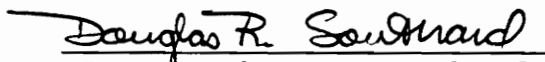
Thesis submitted to the faculty of the
Virginia Polytechnic Institute and State University
in partial fulfillment of the requirements for the degree of

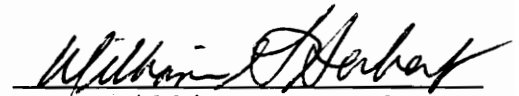
MASTER OF SCIENCE IN EDUCATION

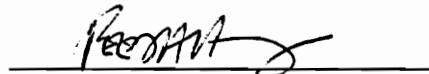
in

Health and Physical Education

APPROVED:


Dr. Douglas R. Southard


Dr. William G. Herbert


Dr. Reed H. Humphrey

February, 1992

Blacksburg, Virginia

c.2

LD
5655
V855
1992
P356
c.2

**Effects of Acute Aerobic Exercise on Cardiovascular
Reactivity in response to
Psychological Stressors in Trained Cyclists**

by

Susi Palmer

Chairperson: Dr. Douglas R. Southard

Department of Health and Physical Education

(ABSTRACT)

The purpose of this study was to examine the effects of acute aerobic exercise on cardiovascular reactivity to psychological stress. Twenty-four trained male cyclists (aged 19-26) from local cycling teams and clubs in the Blacksburg, Virginia area participated in this exercise study. These subjects cycled a minimum of 4 times per week, 30 minutes per session and were randomly assigned to either the Cold Pressor (CP) or Stroop Color Word (SCW) tasks. All subjects participated in exercise and control sessions. The exercise session consisted of 30 minutes at 70% VO_2 max and the control session involved several health assessments with no exercise.

There was no significant difference in blood pressure (BP) or heart rate (HR) change scores (peak-baseline) for treatment or interaction effects. Baseline and recovery systolic blood pressure (SBP), however, showed significant differences for the main effect of treatment (exercise vs. control) ($p < .05$); interaction effects had a tendency

toward significance ($p=.09$) for baseline SBP. These differences in baseline prompted a re-analysis to statistically explore a possible reduction in anticipatory response to psychological stressors. No treatment or interaction effects were found in this secondary analysis.

The results of this study provide only limited support for the hypothesis that acute aerobic exercise reduces BP response to subsequent non-exercise stressors. The effects, if present at all, appear to be stronger in the baseline or anticipatory period. Future research designs should be capable of specifically examining effects in the anticipatory period. These results also provide little support for a selective effect of aerobic exercise on BP reactivity based upon the type of stressor presented.

ACKNOWLEDGEMENTS

This project could not have been accomplished without the help of many people. I would like to thank my committee members, Drs. Doug Southard, Bill Herbert, Reed Humphrey and Jay Williams for supporting me along the way. I owe Dr. Southard a special thanks for his words of wisdom and encouragement (locally and long-distance!). Also, for having an open ear and showing kindness at the times when I needed it most.

Thanks to Susan Hemesath and Jaime Esteban for their confidence and advice and to Al Hansen for his generous help with my figures. Thanks to Gina Brumfield, Tammy Lease and Susan King for what seemed like endless hours in the lab with their help with data collection.

Thanks also to my family for having me home again while finishing and somehow allowing me the freedom to feel frustrated and be grouchy during the final stages. Thanks and I love you, mom, dad and Patti.

I especially want to thank my fiance, Andrew. I could not have done it without him and his confidence in me. His ideas and relentless help with writing and data collection, as well as his availability to laugh and cry with, have truly showed me what unconditional love is.

Most of all, without the strength and love of the Lord Jesus Christ, I could never have done this project. Because of Him, there is true meaning and joy in living.

TABLE OF CONTENTS

	<u>Page</u>
Acknowledgements	iv
List of Tables	vi
List of Figures	vii
I. INTRODUCTION.....	1
Statement of the Problem.....	4
Research Hypotheses.....	5
Significance of the Study.....	5
Delimitations.....	7
Limitations.....	8
Definitions and Symbols.....	8
Basic Assumptions.....	9
Summary.....	10
II. REVIEW OF THE LITERATURE.....	12
Introduction.....	12
Aerobic Exercise and CVR.....	13
Benefits of aerobic exercise.....	13
Chronic Exercise and CHD.....	14
Modulating Effects of Aerobic exercise on CVR.....	18
Cross-sectional studies.....	18
Training studies.....	19
Acute Exercise studies.....	21
Mechanisms of Influence.....	24
Summary.....	25
III. JOURNAL MANUSCRIPT.....	28
Abstract.....	29
Introduction.....	31
Summary.....	34
Purpose.....	34
Methods.....	35
Statistical Procedures.....	39
Results.....	40
Discussion.....	44
References.....	55
IV. SUMMARY AND RESEARCH RECOMMENDATIONS.....	60
Research Implications.....	63
Recommendations for Future Research.....	68
BIBLIOGRAPHY.....	71
APPENDIX A: Methodology.....	77
APPENDIX B: Informed Consent.....	86
APPENDIX C: Subject Data Forms.....	91
APPENDIX D: Research Protocols.....	99
APPENDIX E: Statistical Design.....	104
APPENDIX F: Statistical Analyses.....	107
APPENDIX G: Raw Data Tables.....	123
VITA	127

LIST OF TABLES

<u>Table</u>	<u>Page</u>
1. Subject Characteristics.....	50
2. Table of Main Effect of Treatment.....	51

LIST OF FIGURES

<u>Figure</u>	<u>Page</u>
1. Systolic Blood Pressure Reactivity.....	52
2. Diastolic Blood Pressure Reactivity.....	53
3. Heart Rate Reactivity.....	54

CHAPTER I

Introduction

Exercise and health have attained overwhelming popularity during the past decade. The notion of sports and physical activity, however, is not a new phenomenon as people of previous centuries have participated in sports and all kinds of activity. Historically, sporting activities were recreation oriented. These days, the motivation behind exercise is different. Today, people engage in physical activity and exercise to lose weight, to look younger, and to manage stress. Increasingly, people also are turning to exercise for therapy and rehabilitation.

Cardiovascular disease (CVD) is the number one killer in America. The awareness that exercise may help reduce some of the risks for acquiring CVD is a major reason that exercise has become an integral part of today's lifestyle. The physiologic and psychological benefits of exercise are diverse. These include cholesterol lowering effects, improved functional capacity, social interaction and mood alterations, all adding to an improved quality of life for the participant.

Other factors related to CVD are pharmacological, behavioral and psychological influences. Cardiovascular

reactivity (CVR), defined as the physiological change in heart rate (HR) and blood pressure (BP) occurring in response to stress, has been hypothesized to be associated with the development of this disease. Studies have linked CVR and exercise to promote the idea of exercise as a possible non-pharmacological intervention to halt the progression of CVD. Additionally, exercise may even help prevent or retard its progression by reducing CVR. A higher likelihood for the development of CVD are those with high CVR (Dimsdale, Alpert, & Schneiderman, 1986). Ebbeson, Prkachin, & Mill (1989) showed that acute exercise may provide a "protective window" effect to stress reactivity following exercise. In other words, hemodynamic measures such as HR and BP may be modulated following aerobic exercise especially for those who undergo excessive CVR during mental stress. Therefore, it could possibly be said that excessive CVR during mental stress may be a risk factor for CVD.

The notion that physical exercise *per se* reduces the risk for CVD has been the reason that research in this field has been challenged. At the epidemiological level, interest in mortality and morbidity due to CVD are a main focus. Several cross-sectional studies show that differences in aerobic fitness predict mortality better than current activity status (Bruce, Hossack, deRouen, & Hofer, 1983;

Mundel, Erikssen, & Roddahl, 1987; Solboldski, Kolesar, Kornitzer, de Backer, Mikes, Dramaix, Degre, & Denolin, 1988). Unfortunately, many variables are involved in the development and progression of CVD, thereby making such research an even greater challenge.

Various psychosocial mechanisms have been studied in order to help determine the effect of exercise on CVR (Rejeski, Gregg, Thompson, & Berry, 1990; and Hemesath, 1990). In addition, psychological stressors such as the Cold Pressor (CP) and Stroop Color Word tasks (SCW) have been implicated as valuable tools when estimating the presence of CVR (Ebbeson, 1989; Keys, Taylor, Blackburn, Brozek, Anderson, & Simonson, 1971; Rejeski, Gregg, Thompson, & Berry, 1990). Therefore, psychological tasks seem to be a promising avenue for the determination of CVR and the impact of such changes on cardiovascular health.

Most studies in this area have focused on the effects of chronic training on CVR. These studies have yielded mixed results, mostly due to complications in methodology (deGeus, vanDoornen, deVisser, & Orlebeke, 1990; Duda, Sedlock, Melby, & Thaman, 1988). Additionally, most of these studies are cross-sectional. Less common, however, is research exploring the effects on CVR utilizing acute aerobic exercise bouts. These studies explore the effects of acute aerobic exercise on CVR for homogenous populations

(Ebbeson, 1989, Hemesath, 1990 and Rejeski, 1990). Even though mostly positive results exist in acute exercise studies, this is still a new area and more research is needed. The mechanisms by which these processes occur are unclear as well. If exercise is to be used as therapeutic intervention for CVD, it is imperative that the mechanisms underlying CVR be understood.

Statement of the Problem

Acute aerobic exercise has been suggested to have a "protective window" effect on CVR to psychological stress. That is, responses in parameters such as HR, BP, and the like can be attenuated through an acute bout of aerobic exercise. In addition, the majority of research pertains to chronic training studies; few conclusive studies involving acute bouts of aerobic exercise exist. To more thoroughly determine mechanisms involved, stressors eliciting different mediated origins are needed. With the exception of Ebbeson et al., 1990, previous studies have not included this (Rejeski, 1990, Hemesath, 1990). Also, exercise intensities and/or durations have been too short or too long for normal populations. Therefore, the purpose of this study is to study the effects of an acute aerobic exercise bout (30 minutes at 70% VO_2 max) vs. a control session (40 minutes health assessment measurements) on CVR in response to two different psychological stressors, the CP task or the SCW

task on trained cyclists. Specifically, this study has two purposes:

- 1) To examine the effects of acute aerobic exercise on systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) following the Cold Pressor or Stroop Color Word psychological stress tasks.
- 2) To extend Rejeski's findings by determining if a similar reduction in SBP, DBP, and HR will occur in both the CP and SCW tasks. (Rejeski used the SCW task only).

Research Hypotheses

- Ho1: There is no difference in CVR (HR, SBP, and DBP) to the laboratory stressors presented 1 hr following a 30-minute exercise session as compared to 1 hr following the control session.
- Ho2: There is no difference in the effect of 30-minutes of aerobic exercise on CVR to the CP task as compared to the SCW task.

Significance of the study

Exercise has been associated with the reduction of CVR in response to non-exercise stressors. Much of this research is currently available; however, a handful remains inconclusive. The implications are important to CHD patients and need to be confirmed. Methodological problems may

be the blame for the nonsignificant findings. For example, some designs have been non-random, biases have been made in subjective selection, or specific characteristics, ie., Type A's or hypertensives (Lake, Suarez, & Tocci, 1985; Sherwood, Light, & Blumenthal, 1989). Some have even used subjective ratings for exercise levels rather than using maximal exercise tests (Blumenthal et al., 1988). Aerobic power and physical activity, which compete to be the most important parameter of CVR reduction, still have not been properly addressed. Lately, some researchers have opposed the idea of using heterogeneous groups.

Most studies have focused on chronic exercise training rather than the immediate effects of acute exercise bouts. The need for using acute exercise is obvious from the documented research showing the CV changes immediately following an acute bout of exercise. These changes have been shown to have lasting effects for up to 24 hours (Ebbeson, 1989) which chronic exercise can possibly mask. Few studies consider the effects of acute aerobic exercise. In addition, previous studies lack the use of a consistent stressor, ie., SCW or CP to fully determine the effects of exercise on CVR. Therefore, acute aerobic exercise will also be used in order to confirm promising results as seen by the very few existing studies. Additionally, this study will utilize both the Cold Pressor (CP) and Stroop Color

Word (SCW) tasks. The CP task was implicated as a valuable test in estimating the probability of CVD development via adequate CVR responses (Ebbeson, 1989; Martinez, 1990). The SCW task has also been found to be effective in eliciting cardiovascular and neuroendocrine responses (Rejeski, 1990; Bittker, Buschbaum, Williams, & Wynne, 1975). The CP task will be used to observe peripheral alpha-receptor mediation while the SCW task affects central beta-receptor activity. The methodological inconsistencies encountered in other studies will be addressed by using both a physical (CP) and mental (SCW) psychological stressor which isolates their respective mechanism in a homogeneous subject pool.

Delimitations

The following delimitations were set by the investigator:

1. The subjects were males of the same approximate age ranging from 19-26 years.
2. The study used trained males as defined by ACSM fitness guidelines (bike at least four times per week, at least 30 minutes per session).
3. The subjects were free of any known cardiovascular disease, diabetes, Raynaud's disease and were non-smokers.

4. Change in CVR was measured as the difference between peak response and baseline measurement for each dependent variable: HR, SBP and DBP.
5. Subjects were to adhere to specific stipulations prior to each session regarding food, exercise, alcohol and analgesic restrictions.
6. Subjects were tested at approximately the same time of day for each session.
7. Both the SCW and CP tasks were used in this study.

Limitations

The following factors may have affected the outcome of the study:

1. Subjects may have been at different phases in their training program.
2. Some of the subjects may have had previous experience to the VO_2 max test and thus have attained a learning effect to it.
3. The subjects may have had differences in levels of competitiveness.
4. There may have been differences in levels of fitness training among the subjects.

Definitions and Symbols

Several terms and symbols used in this study may require some clarification as follows:

1. Acute Aerobic Exercise : one bout of aerobic exercise used in this study in order to study immediate physiological responses.
2. Cardiovascular Reactivity: magnitude of change in any function related to the cardiovascular system (specifically, HR, SBP and DBP) that is brought on by a stressor (Ebbeson et.al., 1989).
3. Cold Pressor Task (CP): a physical psychological stressor in which the subject immerses his hand into cold water (1-3 degrees Celsius) for one minute in order to measure a vasoconstrictive response (Keys et.al., 1971).
4. Stroop Color Word Task (SCW): a mental psychosocial stressor in which subjects perform for the measurement of hemodynamic responses. The subjects respond to names of colors that are written in opposing colors which flash on a computer screen at a given interval while an audio tape plays another color. The subject responds to the color of each word as many times correctly as possible (Rejeski et.al., 1990).

Basic Assumptions

The investigator made the following basic assumptions:

1. Subjects exerted a maximal effort on the bicycle VO_2 max Test.

2. Subjects complied to exercise adherence requirements prior to each session.
3. HR, SBP, and DBP were fairly stable from day to day.

Summary

The risk of acquiring CAD is often associated with the prevalence of risk factors. Some of these include being a male, smoking, hyperlipidemia, hypertension, or positive family history. Based upon the documented role of exercise in reducing some of these risk factors, it is increasingly clear that lack of exercise is an important risk factor as well. Given such, promoting the idea of a pro-active lifestyle to combat the incidence of CVD as well as its progression is relevant.

Additionally, the way one responds to non-exercise stressors, such as mental stress, could have an influence on coronary artery disease. That is, one who shows an exaggerated CVR response to such anxiety could have an elevated risk for the development of CVD. Those who have a higher fitness level appear to have decreased CVR response to stress of this kind. While there are certain methodological problems that need to be addressed, it is hopeful that the present study will find conclusive information regarding tempering effects of exercise on CVR.

This study assessed cardiovascular reactivity in response to the Cold Pressor or Stroop Color Word psychosocial tasks in trained males following an acute bout of aerobic exercise versus a health assessment session. In addition, the purpose behind using two stressors is to help determine further the mechanisms involved.

CHAPTER II

Review of the Literature

Introduction

The inability to cope with the everyday stress associated with both the workplace and the home may be a contributing factor in the development of CHD. Psychological stress may be defined when a variety of psychosocial stimuli disturb normal physiologic processes. The response of the cardiovascular system to psychological stress is termed cardiovascular reactivity (CVR). Excessive CVR may be deemed as a marker for a psychogenic risk factor in CHD. In other words, prolonged exposure to psychological stress may result in chronically elevated blood pressure measurements (especially diastolic blood pressure (DBP)) (Obrist, 1981). When the cardiovascular system is harmed in this manner, severe pathophysiologic consequences can result. When factors such as hypertension disrupt the integrity of the vessel wall, physical obstruction (ie., atherosclerosis) can occur. It is this link between CVR and CHD that mark the importance of detecting non-pharmacological intervention for the reduction and/or prevention of CHD.

It is well documented that many people suffer from excessive levels of stress and that regular exercise has been suggested to reduce the harmful effects of CVR to

repeated exposure to psychological and daily stressors. (Blumenthal & McCubbin, 1987; Surwit, 1986). Paffenbarger, Hyde, Wing & Hsieh, (1986) suggest that the protective results of aerobic exercise might reflect intermediate effects on the stress-related processes involved in CVR development and progression. In turn, this may help reduce CHD risk. Though the exact mechanism remains unclear, biological pathology seems the most promising. Certainly, with all the physiological benefits of exercise, it is obvious that exercise plays an important role in the reduction of CHD.

The tendency exists for reduced CVR due to exercise. The exact mechanism(s) remain unclear, though there appears to be a link between the type of CVR, type of stressor used and their respective receptor and pathway of mediation. It is the purpose of this study to use acute aerobic exercise to strengthen the notion that through lessened CVR to psychological stress, it may be possible to reduce CHD and/or retard its progression.

AEROBIC EXERCISE AND CVD

Understanding the relationship between CVR, CHD and exercise is important when considering the overall picture of exercise and CHD.

Benefits of aerobic exercise. The physiological changes associated with physical exercise are impressive and

quite diverse. These include weight reduction, increases in lean body mass and decreases in percent body fat, increased feelings of self-esteem and cognitive functioning, and stress management. Exercise is even prescribed as an interventive therapy for special populations such as those with diabetes, osteoporosis, and arthritis. A special population that has gained much attention lately however, is that of CHD. In addition to the mechanisms above, exercise may moderate excessive CVR to environmental stimuli, thereby reducing one's risk of developing hypertension and CHD. (Dimsdale, Alpert & Schneiderman, 1986).

Chronic exercise and CHD. While the number of deaths related to CHD, strokes and associated cardiovascular problems has declined since the 1960's (Goldman & Cook, 1984), the incidence is still quite high and continues to be the number one killer in the United States today. Promoting knowledge and reduction of risk factors seems to be an avenue for controlling the incidence of CHD. The occurrence of risk factors in certain individuals is inevitable due to the life process, family history, gender, and other uncontrollable variables. Reducing controllable risk factors is of utmost importance. Ideally, the control and/or elimination of risk factors is the best treatment. Even when CHD has been established, exercise can help combat these risk factors as well as prevent them. Exercise helps

reduce blood lipids (by increasing HDL cholesterol), weight and blood pressure, increase the efficiency of the heart, and even serve as a coping mechanism to daily life stressors. This would lead one to suspect that physical inactivity might be is a risk factor. Additional evidence seems to indicate that the longer these risk factors exist, the greater the impact on one's physical health (Goldman & Cook, 1984).

Numerous studies involving the beneficial effects of exercise on animals exist. Physical training not only resulted in higher HDL and lower LDL and VLDL cholesterol and triglyceride levels, but showed a lower incidence of coronary atherosclerosis in monkeys (Kramsch, 1981). Increases in the diameter of coronary arteries and proliferation of coronary collateral development was shown as well (Wyatt and Mitchell, 1978, Neill and Oxendine, 1979). Further studies on humans are needed before similar conclusions can be drawn. These results leave promising avenues for such research, however.

Other beneficial effects that chronic exercise has on CHD are well established. For example, physical activity and CHD risk have a significant inverse relationship to each other (Powell, 1987). The likelihood of survival from a myocardial infarction increases as a result of regular exercise (Morris, 1973). Overall, one sees reduced

morbidity and mortality among patients who participate in regular exercise training (May, 1982, Barnard, 1983, Kavanagh, 1979). A study by Kannell & Sorlie (1979) suggested that inactive men were significantly more likely to develop CHD than their active counterparts, independent of other risk factors. Likewise, a study by Morris et al., (1973) found a 50% reduction in CHD in active vs. inactive men over an 8-year period. Paffenbarger, Wing & Hyde (1978) reported those who expended 2,000+ KCal/week (defined here as an active lifestyle) had half the risk of CHD compared to those less active.

Less obvious acute responses help promote CHD risk factor reductions. Rosing (1970) and Williams (1980) found increases in fibrinolysis and fibrinolytic capacity in response to venous occlusion. This is important as it may help alleviate coronary thrombosis. Additional benefits include enhanced myocardial electrical stability and decreases in coronary vasospasm. This is thought to be secondary to decreased adrenergic stimulation (Maseri, 1978). During cardiac ischemia, the risk of ventricular fibrillation is reduced as physical training results in increases in cardiac parasympathetic tone (Kenney, 1985, Billman, Schwartz & Stone, 1984). The decreases in total peripheral resistance (TPR) seen with regular exercise should not be overlooked. Though the exact mechanisms

remain unclear, the increase in vascularity along with alterations in metabolic function work together to increase cardiac output which is an important benefit in healthy and diseased populations because it increases the efficiency of the heart (Barnard, 1983). Certainly, with all the physiological benefits of chronic exercise, it is obvious that habitual exercise plays an important role in the reduction of CHD.

Occupational stress may lead to high reactivity which could relate to CHD. Perhaps exercise can lessen these risks. "High strain" situations on the job may predispose individuals to a higher incidence of CHD, myocardial infarction (Karasek, 1982) and mortality (Karasek et al., 1981) where high strain was defined as conditions of high demand and low control. When public speaking was used as the psychological stressor (Freed, Thomas, Lynch & Friedman, 1989) HR, SBP and DBP were significantly greater while the patients were speaking than while they were resting quietly. More importantly, ventricular and atrial arrhythmias were more frequent while talking. Incidence of ventricular arrhythmias has been recognized as a significant risk factor for sudden cardiac death (SCD), especially immediately post-MI (Ruberman, 1984). CVR in response to these stressors may be lessened by acute aerobic activity. The beneficial

effects of aerobic activity on cardiac function and peripheral resistance have been clearly documented.

Modulating effects of AE on CVR

Cross-sectional studies. Differences in fitness levels has been suggested to contribute to differences in CVR to psychological stressors. Some of the research shows agreement that higher fit individuals showed faster autonomic recovery to stress as well as lessened HR and DBP during cognitive/mental stressors (Light, Obrist, James & Strogatz, 1987, Lake, Suarez, Schneiderman & Tocci, 1985, and Ward, Carmelli, & McElroy, 1989), while others revealed differences in recovery time only (Hollander & Seraganian, 1984, Keller & Seraganian, 1984, Sinyor et al., 1983, and deGeus, van Dornen, deVisser & Orlebkko, 1990). deGeus et al., (1990) suggested that these differences in HR may be due to cardiac vagal/sympathetic activity; ie., increased vagal tone may exist in those with higher fitness levels, thereby possibly explaining this improved functioning of the parasympathetic nervous system.

While these results may be inconsistent, deGeus strongly suggests that it may not only be fitness level that is responsible, but an underlying genetic component, that being aerobic power which may influence reactivity to stress. For example, substantial differences in aerobic fitness were found in spite of the fact that these

individuals reported low levels of habitual activity. Subjective ratings may deter some of these findings. Even though differences in CVR at rest and exercise may be influenced by factors such as predetermined (genetic) aerobic fitness capacities, cross-sectional type studies are worth considering as there are positive results indicating that higher fitness levels may help reduce CVR. Using an indicator of anaerobic threshold (ie., ventilatory equivalent) may be help to separate the effects of fitness from training. More conclusive research is needed in order to fully understand the impact that difference fitness levels has on CVR.

Training studies. Numerous results have been found in support of the use of aerobic exercise as method of reducing CVR, and sympathoadrenal responses during and after mental stress. Significant results along these lines were found in a study (Blumenthal, Fredrikson, Kuhn, Ulmer, Walsh-Riddle & Applebaum, 1990) of 36 healthy Type A males in which random assignment put the subjects in either an aerobic exercise training group (AE) or strength and flexibility (SF) group. Results indicated that the AE group (who underwent 12 weeks of continuous walking or jogging at an intensity of at least 70% VO_2 max) showed attenuation of HR, DBP and estimated myocardial oxygen consumption (MVO_2) during the task and lower HR, BP and MVO_2 during recovery. The SF group showed

only DBP reductions during the task which the authors contributed to habituation. In a very similar study by Sherwood, Light, & Blumenthal (1989) using AE vs. SF groups of Type A men, findings in AE was also seen. The most drastic results were exhibited in a general reduction in SBP, HR, and TPR (at rest, competition and recovery) in those hypertensive subjects. Thus, Sherwood et al. suggested that borderline hypertensives may benefit most to cardiovascular adjustments to aerobic conditioning.

These two studies showed significant results that were limited to special populations (Type A and hypertensives). Holmes & Roth (1985) studied 20 normal subjects; 10 high fit and 10 low fit who were drawn from a fitness distribution of 72 subjects to assure that extreme AF levels were represented. They responded to one mildly stressful psychological task and increases in HR in high fit subjects were smaller than low fit. Light, Obrist, James & Strogatz (1987) reported measures of HR, SBP and pre-ejection period as reflected by group differences. These measures revealed that low fit individuals responded more to exercise and reaction time task after covariate adjustments were made for baseline differences. Group differences resulted in response to the cold pressor test (CP), although they were smaller and generally nonsignificant. Light et al. then concluded that decreases in beta-adrenergic myocardial

responses are a result of aerobic training on physical and behavioral challenges. Overall, the general consensus of these training studies seem to indicate lessened CVR as a result of aerobic training.

There are also a number of studies finding no effect of exercise training on CVR (Hull, Young & Ziegler, 1984, Sinyor, Schwartz, Peronner, Brisson and Seraganian, 1983). One potential explanation may be methodological problems occurring in these studies. These could be eliminated in future work, thereby increasing the validity of the idea of a pro-active lifestyle. For example, Rejeski et al., (1990) cited low exercise levels (some studies employed 12 to 20 minutes at 55% VO₂ Max) as one reason. In addition, discrepancies may occur when HR was used as the primary stress response rather than BP. Steptoe, Moses, Mathews & Edwards (1990) proposed other methodological problems. They stated that uncertainties of response patterns (some high and some low) are due to variability to fitness levels among subjects. Secondly, they were concerned whether aerobic power or fitness level was the most influential parameter suggesting that aerobic power may have strong genetic determinants (ie., differences in aerobic capacity may exist regardless of fitness level).

Acute exercise studies. Most work has been done in training or chronic exercise protocols. In fact, few

research studies have to do with CVR and acute exercise. Ebbeson et al., (1989) addressed the issue of acute exercise and CVR. In order to confirm positive CVR adaptations, she adjusted for some methodological problems such as the exercise intensity. Twenty- four healthy males were used and random assignments were made to two different exercise durations of one- and two-hour durations at 55% VO_2 max. The SCW task, CP task and a public speaking test were used (each of which utilized primarily an alpha-adrenergic or beta-adrenergic receptors leading to the determinations of the increase of peripheral or central stimulation, respectively). Recovery at 1, 3, and 24 hour post-exercise were used in order to determine short- and long-term effects. The exercise bouts exhibited greatest effects in response to the CP task and DBP, thereby possibly indicating that acute exercise has a suppressive effect on alpha-adrenergic mediated stress reactions (Ebbeson, 1989; Williams, 1986). Reductions in the sympathetic alpha-adrenergic stimulation to behavioral stress may be a mechanism involved with reduced CVR in acute exercise. In addition, preliminary analysis from Hemesath (1990) shows a tendency towards similar results. In her study, college-aged, untrained males had a trend toward lower DBP CVR to the CP task after exercising at 55%-60% VO_2 max for 30

minutes than a comparison group undergoing five minutes of free-wheeling.

In contrast, several studies have not found acute exercise to be adequate in reducing CVR (Roth, 1989, Russell, Epstein & Erickson, 1983 and Duda, Sedlock, Melby & Theman, 1988). However, low exercise levels used in these studies are a common factor and, therefore, plausible explanation for the negative results.

Many of the above existing studies have examined the effects on untrained subjects. More recently, two studies done by Steptoe, Kearsly, & Roy, (1991) resulted in significant buffering effects of exercise on SBP, DBP and HR values on trained subjects. In support, Rejeski et al., (1990) also used a sample of 12 trained cyclists. HR and BP were the hemodynamic measurements taken. Baseline, task reactivity and recovery measurements were monitored in a heavy exercise group (80% VO_2 max for 60 minutes), and light exercise group (50% VO_2 max for 30 minutes) and a control (non-exercise) group in response to the SCW task. Significant conclusions were most prominent in BP. SBP during the task was significantly higher in the light and control group and DBP was significantly higher in the control group compared to either exercise group. HR showed no positive CVR effects across groups. Therefore, Rejeski and associates (1990) suggested that there may be a dose-

response relationship between acute exercise and the attenuation of psychophysiological reactivity to stressor trained subjects. These results also confirm studies involving trained subjects; possible mechanisms responsible for changes in total peripheral resistance are quite possibly alpha- and beta-adrenergic receptors.

Mechanisms of Influence. Psychological and physical exercise elicit similar physiological responses, (Blumental & McCubbin, 1987) ie., increases in SBP, HR, CO, and increases in epinephrine, norepinephrine and cortisol (Hartley, Mason, Hogan, Jones, Kotchen, & Ricketts, 1972; McCubbin, Richardson, Lauger, Kizer & Obrist, 1983). The major response difference between physiological and psychological stressors appears to be metabolic in nature, thereby resulting in a possible explanation of the mechanisms involved in the contribution of CHD. The elevations in cardiac output and HR associated with physical exercise result in increases in peripheral oxygen extraction. The same is not true for psychological stress, however. Increased cardiac output during psychological stress creates a relative state of circulatory overperfusion in oxygen (Langer et al., 1985) which may predispose vasoconstriction and ultimately hypertension. According to Blumental & McCubbin (1987) this adjustment to psychological stress may result in cardiopulmonary consequences when

maintained over long periods of time. In other words, prolonged behavioral stress could develop into a state of chronic hypertension.

With this debilitating adjustment, one can see the effects on the circulatory system. Thus, hypertension and CHD is a much greater possibility when exposed to prolonged behavioral stress, especially in highly reactive individuals.

Two differently mediated stressors were used in this study. The CP task, a physical stressor, requiring a passive coping response, primarily elicits dramatic increases in DBP. This is thought to reflect stimulation of alpha-adrenergic receptors leading to peripheral vasoconstriction. The SCW task, which requires a more active coping response, primarily elicits increases in HR and SBP which is primarily associated with increased stimulation of central beta-adrenergic receptors. Therefore, this study will utilize both of these stressors in order to better identify the mechanisms involved in variable responses to different mediated stressors.

Summary

The apparent relationship between cardiovascular reactivity and coronary artery disease has opened up a new field of interest within the past few years. Since cardiovascular reactivity in response to psychological

stressors may be attenuated as a result of aerobic exercise, identifying related non-pharmacological therapies to indirectly reduce CHD risk and/or mortality is a possibility. The link between CVR and physical exercise seems to be possibly the deactivation of the sympathetic nervous system. Numerous exercise training studies have shown positive results. To date, however, only a few studies on acute exercise and cardiovascular reactivity have been undertaken, and results have been reported that suggest a beneficial modulating effect of exercise. However, reports to the contrary may be found in the recent scientific literature. The reasons for this may be that BP seems to be the most important variable for positive results; many studies that failed to show CVR attenuation effects used HR as their primary variable. HR does not reflect peripheral effects of exercise on the circulation, as would measures related to total peripheral resistance (TPR). Additionally, HR may not be accurate because of the elevated post-exercise HR values that occur as a result of a prolonged aerobic exercise bout. Another reason is that low levels of exercise (50-55% VO_2) were often used as stated previously (Duda, 1988; Robertson & Epstein, 1985). Lastly, while several research studies suggest that acute exercise results in attenuated CVR following the exercise session (Ebbeson et al., 1989; Rejeski et al., 1990; Nixon,

Robertson, & Epstein, 1986), these are rare. Therefore, the current study will try to include procedures that reduce methodological problems apparent in the previous studies that have shown beneficial exercise effects relative to cardiovascular reactivity. Specifically, the purpose of this study is to show that acute aerobic exercise reduces one's CVR to psychological stress.

CHD is still very much a national health problem. Since evidence such as the incidence of CHD can be predicted as a result of DBP to CP task, for example (Keys, Taylor, Blackburn, Borsek, Anderson & Simonson, 1971), continued research is greatly needed in this area. Exercise, therefore, may well be worth the effort when it may not only improve one's quality of life, but reduce incidence and/or mortality of CHD.

CHAPTER III
JOURNAL MANUSCRIPT

**Effects of Acute Aerobic Exercise on Cardiovascular
Reactivity in response to
Psychological Stressors in Trained Cyclists**

by

Susi Palmer

Chairperson: Dr. Douglas R. Southard

Department of Health and Physical Education

(ABSTRACT)

The purpose of this study was to examine the effects of acute aerobic exercise on cardiovascular reactivity to psychological stress. Twenty-four trained male cyclists (aged 19-26) from local cycling teams and clubs in the Blacksburg, Virginia area participated in this exercise study. These subjects cycled a minimum of 4 times per week, 30 minutes per session and were randomly assigned to either the Cold Pressor (CP) or Stroop Color Word (SCW) tasks. All subjects participated in exercise and control sessions. The exercise session consisted of 30 minutes at 70% VO_2 max and the control session involved several health assessments with no exercise.

There was no significant difference in blood pressure (BP) or heart rate (HR) change scores (peak-baseline) for treatment or interaction effects. Baseline and recovery systolic blood pressure (SBP), however, showed significant differences for the main effect of treatment (exercise vs. control) ($p < .05$); interaction effects had a tendency

toward significance ($p=.09$) for baseline SBP. These differences in baseline prompted a re-analysis to statistically explore a possible reduction in anticipatory response to psychological stressors. No treatment or interaction effects were found in this secondary analysis.

The results of this study provide only limited support for the hypothesis that acute aerobic exercise reduces BP response to subsequent non-exercise stressors. The effects, if present at all, appear to be stronger in the baseline or anticipatory period. Future research designs should be capable of specifically examining effects in the anticipatory period. These results also provide little support for a selective effect of aerobic exercise on BP reactivity based upon the type of stressor presented.

INTRODUCTION

The inability to cope with the everyday stress associated with the workplace and the home may be a contributing factor in the development of coronary heart disease (CHD). Psychological stress may be defined as a process by which a variety of psychosocial stimuli disturb normal physiologic processes. The response of the cardiovascular system to psychological stress is termed cardiovascular reactivity (CVR). Excessive CVR may be deemed as a marker for a psychogenic risk factor in CHD (Keys et. al., 1971). Excessive CVR may also result in chronically elevated blood pressure measurements (especially diastolic blood pressure (DBP)) (Obrist, 1981; Menkes et. al., 1991). Diagnosed hypertension is a major risk factor for CHD. When the cardiovascular system is harmed in this manner, severe pathophysiologic consequences can result. One of the mechanisms for primary hypertension is abnormal CNS and sympathetic mediation of increased peripheral resistance. The biophysical effects of high blood pressure (BP), prolonged over long periods may disrupt the integrity of the vascular system. When this type of physiological stress occurs in response to injury, cell division and accumulation of the endothelium results, which in turn crystallizes and calcifies. This cell proliferation phase then hastens plaque formation. It is this link between CVR

pharmacological intervention for the reduction and/or prevention of CHD.

It is well documented that many people suffer from excessive levels of stress and that regular exercise has been suggested to reduce the harmful effects of CVR to repeated exposure to psychological daily stressors (Blumenthal & McCubbin, 1987; Surwit, 1986). Paffenbarger, Hyde, Wing & Hsieh, (1986) suggest that the protective results of aerobic exercise might reflect intermediate effects on the stress-related processes involved in CVR development and progression. In turn, this may help reduce CHD risk. Though the exact mechanism remains unclear, induction of accelerated atherosclerosis seems the most likely. The beneficial effects of exercise include increased likelihood of survival from a myocardial infarction and reduced morbidity and mortality among patients who participate in regular exercise training (Powell, 1987; Morris, 1973; May, 1982, Barnard, 1983, Kavanagh, 1979). Certainly, with all the documented physiological adaptations of exercise training, it is obvious that exercise should have an important role in the reduction of CHD.

Differences in fitness levels has been suggested to contribute to differences in CVR. Some research has shown that higher fit individuals showed faster autonomic recovery

to psychological stress, as well as lessened HR and DBP responses during the time that cognitive/mental stressors are administered (Light, Obrist, James & Strogatz, 1987; Lake, Suarez, Schneiderman & Tocci, 1985; and Ward, Carmelli, & McElroy, 1989). Other investigators reported lower HR and BP effects in recovery time only (Hollander & Seraganian, 1984, Keller & Seraganian, 1984, Sinyor et al., 1983, and deGeus, van Dornen, deVisser & Orlebkco, 1990). Numerous results have been found in support of the use of aerobic exercise training as method of reducing CVR (Blumental, Fredrikson, Kuhn, Ulmer, Walsh-Riddle & Applebaum, 1990; Blumenthal 1989). However, some discrepancies exist in regards to these results (Hull, Young & Ziegler, 1984, Sinyor, Schwartz, Peronner, Brisson and Seraganian, 1983) where an aerobic exercise group did not exhibit CVR reduction versus a strength-flexibility control group.

While most of these studies have focused on the effects of physical training, few investigators have examined the effects of acute bouts of aerobic exercise. Positive results were found by Ebbeson (1989) and Hemesath, (1990) in which exercise had a short-term suppressive effect on stress reactions. Whereas these results were found using untrained subjects, Rejeski, Gregg, Thompson, & Berry, (1990) and Steptoe, Kearsly, & Roy (1991) used acute exercise and

Steptoe, Kearsly, & Roy (1991) used acute exercise and trained cyclists and found significant reductions in SBP and DBP in response to exercise.

The tendency exists in the published literature for reduced CVR due to acute exercise. Though the exact mechanism(s) remain unclear, there appears to be a link between the type of CVR, type of stressor used and their respective neural receptor and autonomic pathway of mediation.

It was the purpose of this study to examine the possible attenuation of CVR resulting from psychological stress through acute aerobic exercise in trained subjects. A second the purpose was to examine differences in the moderation of physiological stress to different stressors, the Stroop Color Word (SCW) or Cold Pressor (CP) psychosocial tasks. These tasks were administered following an acute bout of aerobic exercise at 70% VO_2 max. A 40 minute health assessment condition was used for control purposes. The CVR responses following each session were observed to determine differences between the experimental sessions as well as differences between stressors. It was hypothesized that: 1) CVR (HR, SBP, and DBP) would be reduced following the 30 minute exercise session at 70% VO_2 max and; 2) the interacting effect of HR, SBP, and DBP with each stressor (CP and SCW tasks) would be different.

Use of the two differently mediated tasks helped to ensure the possibility of different physiological responses. The CP is known to elicit dramatic responses in DBP whereas the SCW task elicits SBP responses. The exercise intensity was chosen as 70% VO_2 max since this level elicits significant involvement of the sympathetic nervous system. Other researchers have found 70% VO_2 max optimal for observing physiological changes needed from resting and exercise for CVR differences. A duration of 30 minutes is easily generalizable as it is compatible with American College of Sports Medicine (ACSM) guidelines.

METHODS

Subject Selection

Twenty-four healthy college-aged males (19-26 years) were recruited from cycling clubs at Virginia Polytechnic Institute and State University for participation in the study. Specific criteria were required for participation as follows: 1) healthy males between 18-28 years; 2) free of diagnosed cardiovascular disease, diabetes, Raynaud's syndrome; 3) free of orthopedic and physical limitations that would contraindicate exercise or psychological stress testing, as stated in the ACSM guidelines; 4) trained cyclists as defined by exercising a minimum of four times per week, 30 minutes per session; 5) agreement to adhere to

study guidelines of abstinence of food, beverages, tobacco and analgesics prior to exercise sessions; and 6) non-smokers.

Instructional Procedures

At an organizational meeting subjects received verbal explanation of the study. A disclosure of potential risks and benefits were given to the subjects. Each gave written consent agreeing to the terms of the study and filled out a health history questionnaire form as well. Approval regarding this was sought and approved from the IRB.

Experimental Procedures

Each subject was instructed to abstain from ingesting food for 3 hours and alcohol and analgesics for 24 hours prior to each session. Session 1 was a screening session which consisted of baseline CVR to the CP or to the SCW psychological tasks to which the subjects were then randomly assigned to control for possible order effects of trials in the data. The session also consisted of a maximal effort exercise test on a stationary cycle ergometer to determine workloads imposed later in the experimental session.

Baseline CVR for the CP task was determined as follows: the subject sat quietly for 15 minutes in a chair with the cuff of an automated blood pressure system (Industrial and Biomedical Sensors Corporation Model S-D 700 A cuff) affixed to his non-dominant arm. The dominant hand

was placed in water (0-3 degrees Celsius) for one minute. Resting measures of HR and BP (systolic and diastolic) were determined by averaging the response to the last minutes prior to hand immersion. After the task, the parameters were recorded for 5 minutes and recovery measures were calculated by averaging the first and second minutes. A measured reaction of 5-30 mmHg DBP to the stressor was established a priori in order for a candidate to be eligible for the study.

Likewise, baseline measurements for the SCW were taken as follows: The SCW task consisted of a display on a computer screen in which the names of colors appeared in an ink that differed in color from the name. In addition, there was a cassette verbally announcing a different color not shown on the screen (written or displayed). For example, ("red" may have appeared in blue ink, while the color "yellow" may have been heard on the tape). After sitting quietly for 15 minutes, the subject's resting measures of HR and BP were determined by averaging the responses from the 5 minutes prior to each task. The subjects then responded to the stressor as fast as possible for a 3-minute session and the number of correct responses (to the color of the word) was recorded. HR, SBP and DBP were calculated by averaging the measures taken each minute. Each subject rested for 5 minutes in which recovery measures

were recorded as in the CP task (recovery scores were determined by averaging the responses of the first 2 minutes of recovery).

The maximal exercise test was performed on a cycle ergometer (Bodyguard Model 990) in which a protocol was used based on weight and exercise level. On-line gas analysis was used in conjunction with the Hans Rudolph low resistance low dead space valve. Inspired volumes were measured by the Parkinson-Cowan (P-C) dry gas meter. A visual display which presents minute ventilation and total ventilation to the nearest ± 0.1 liter was electronically integrated with the P-C meter. Inspired respiratory gases were passed through an Aquasorb absorbent analysis. Oxygen and carbon dioxide concentrations were determined by Ametac Analyzers. VO_2 max was computed on an IBM-PC.

Exercise Sessions

Subjects were randomly assigned to one of two different experimental conditions. Forty-eight hours after the initial screening (baseline) session, half of the subjects participated in a 30 minute exercise session at 70% VO_2 . HR and BP was monitored and recorded every 5 minutes. Maintenance of 70% VO_2 max was assured during these recordings by taking HR measurements throughout the exercise bout. Fluid replacement was encouraged throughout the exercise session.

Depending on the order of testing assigned, the subjects next participated in the control session. This was given 48 hours after the SCW or CP session and consisted of a 40-minute attention control procedure in which the subjects underwent several health assessments. These included estimations of percent body fat, skeletal frame size, height/weight recommendations, resting blood pressure and HR measurements.

One hour following each experimental and control session the subject underwent additional CVR testing to the CP or SCW tasks. These CVR procedures followed the protocol of those during the initial screening baseline session. The experimental sessions were counter-balanced across groups.

Statistical Procedures

In order to determine possible confounding differences in subject responses between the exercise and control trials, one-way repeated measures ANOVAs were conducted on age, weight, Max VO_2 , Max HR and Max workload. These one-way ANOVAs were run on three levels for acute exercise data: VO_2 , HR, and watts. Test-retest reliability was determined by comparing the baseline measurements from the control and screening sessions for SBP, DBP and HR. A split-plot specialized repeated measures ANOVA was conducted for the main analysis to determine and compare changes in CVR from

exercise and control sessions. Specifically, this was a two-sequence, two-period cross-over design. Significance for all analyses was set a priori at an alpha level of 0.05.

RESULTS

Subject Characteristics

Subjects did not differ significantly in weight, VO_2 max, or for VO_2 , Watts and HR during the exercise session. Means and SEM are presented in Table 1.

Insert Table 1. here

Reactivity qualification for each subject was set a priori at 5 mmHg for DBP measurement during the psychological stressor. Even though four subjects did not meet this reactivity qualification, analysis was performed using all the subjects (N=24). Analysis was then also performed using the qualified subjects only (N=20). Since results from both of these analyses were very similar and in order, the remainder of the discussion and the results refer to the larger sample size.

Figures 1-3 show experimental data. It appears from these figures that BP baseline measures were influenced by some mechanism related to the exercise effect. Anticipatory

physiologic adjustments are a natural phenomenon prior to any known stressor. According to Figures 1 and 2, it appears that exercise may have had a modulating effect on these anticipatory reactions (baseline values). This should be put forth in action with caution, however, as a washout effect of the residual effect of a bout of the previous aerobic exercise may override the sensitivity of HR for these baseline measures. To further examine this possibility, mean baseline measurements from the screening session were calculated and added to each figure to visually note differences. It should be noted that this screening session occurs on a *separate* day. These screening baselines were used to compare reactivity (peak-screening baseline). Additional analyses were performed on these "new" reactivity measures to statistically consider this theory of reduced anticipatory response. These new reactivity values (peak-screening) are designated as "change 2". Original reactivity values (peak-baseline) are designated as "change 1".

Therefore, mean changes, SEMs, F and P-Values are presented for each: baseline, peak, change 1, change 2 and recovery.

HYPOTHESIS ONE: Main effect of treatment (exercise vs. control) (Table 2).

SBP: Significant differences were found in hypothesis one for baseline, and recovery scores ($F_{1,22}=4.86$, $p=.03$) ($F_{1,22}=5.18$, $p=.03$), respectively. No differences for peak, change 1, or change 2 SBP scores were found.

Insert Table 2 Here

DBP: No significant differences were observed for DBP for baseline, peak, change 1, change 2 or recovery.

HR: Table 2 shows that HR values were significantly different for baseline, peak, change 1, change 2, and recovery scores ($F_{1,22}=20.78$, $p=.0002$); ($F_{1,22}=22.35$, $p=.0001$); ($F_{1,22}=16.30$, $p=.0006$); ($F_{1,22}=22.35$, $p=.0001$); ($F_{1,22}=5.23$, $p=.0322$), respectively.

HYPOTHESIS TWO: Interaction effect of treatment by stressor.

SBP: A trend was found for baseline reactivity measures only ($F_{1,2}=3.15$, $p=.09$). Peak, change 1, change 2 and recovery scores were not significant for SBP.

DBP: No significant findings were found for baseline, peak, change 1, change 2, or recovery scores.

HR: Change 1 measure was the only finding of significance for HR ($F_{1,22}=22.35$, $p<.05$). Baseline, peak, change 1 and recovery values were not significantly different.

Reliability estimates using test-retest for baseline reactivity between the screening and control sessions were $r=.67$, $r=.56$, and $r=.79$ for SBP, DBP and HR measurements, respectively.

DISCUSSION

Results from this study do not agree with others (Rejeski et al., 1990 and Steptoe et. al., 1991), in which dramatic CVR reduction to psychological stress were found in change scores for SBP and DBP following an exercise treatment. Baseline and recovery SBP measures, however, showed a significant difference in the current study between exercise and control (treatment effect) sessions ($p < .05$). A similar trend occurred for DBP values.

Several plausible explanations for inconsistent findings exist. The subjects may have been at different points in their training regimen. Data collection took place in late winter. Therefore, it is reasonable to assume that some cyclists may have been at the beginning of their training phase while others may have been tapering in preparation for a race.

The present study involved some subjects who were very competitive athletes. One may wonder if there is a difference between "trained" and "competitive" regarding the way one responds to stress. In other words, is the highly competitive athlete better able to cope? Has he "developed" the ability to handle and cope with stress (physiologically) well?

Considering previous findings involving reductions in SBP and DBP CVR, some researchers have failed to find

significant effects following exercise (Hull, Young, & Ziegler, 1984; Sinyor, Schwartz, Peronner, Brisson, & Seroginen, 1983). However, these were studies of the difference between high fit and low fit subjects and involved chronic training. The three previously mentioned studies involving acute aerobic exercise bouts (Ebbeson, 1989; Hemesath, 1990; Rejeski, 1990) support the theory of attenuated DBP (and/or MAP) due to exercise. A 1991 study by Steptoe, Kearsley, & Roy also found reduced CVR (SBP and DBP) in two studies involving acute aerobic exercise and mental stressors. Given these findings, CVR during mental stress is suggested to be reduced following exercise. Additionally, upon observation of actual SBP values, 10 out of the 13 subjects (SCW) and 8 out of 10 subjects (CP) SBP CVR values were lower following exercise as compared to the control session. Therefore, even though there is limited statistical significance, the overall trend for the group suggests that the actual SBP value was attenuated following exercise.

In support of these latter studies, observations of the current study reveal lower BP values following exercise. As mentioned earlier, it appears (from Figures 1 and 2) that baseline values were affected in some manner by exercise. These baselines were taken during a time in which the subject was possibly undergoing physiological adjustments

(ie., an anticipatory response) prior to the psychological stressor. This investigator theorizes that these reduced baseline values may have resulted from a modulated anticipatory CVR response to stress. Specific interaction patterns also evolved from the re-analysis (see Figures 1 and 2); anticipatory responses for SBP appeared to have greater reduction with the SCW, whereas the anticipatory effects involving DBP were effected to a greater extent in the CP task. However, it should be considered that the effects of the exercise session itself may explain the lowered BP values and/or lowered anticipatory responses. It is difficult to provide conclusive explanations without design changes; this deserves attention in future work.

As previously mentioned, a trend for attenuation of SBP and DBP after exercise occurred in the current study. Based on this, one might assume that this results in decreased TPR. Although the exact mechanism of this is undetermined, several theories exist. For example, Ebbeson (1989) suggests that exercise appears to have differential effects on stress. It is generally acknowledged that mechanisms involved in arousal to the CP task differ from those in the SCW task (Ebbeson, 1989). CP also is associated with stimulation of peripheral alpha-adrenergic receptors (Williams, 1986). With this in mind, the differences in hemodynamic responses, though modest, now carry more

relevance. According to these results, one task affects each receptor, but it is difficult to make the distinction. Based on our data, the interaction of exercise appeared to have a greater modulating effect with DBP and CP (the alpha-mediated task) for both analyses. In SCW (the beta-mediated task) the results suggested that exercise might have a greater attenuated affect on SBP CVR. This appears to be in agreement of Ebbeson, (1989) and may help further explain the mechanisms involved.

Another possibility might be suggested to have influence on these results. While BP measures were attenuated following the exercise trials, HR values were increased for the group. These elevated HR measurements may have been a compensatory mechanism to maintain cardiac output in the face of decreased TPR and venous return. In addition, decreased plasma volumes (fluid loss through sweating) may have also contributed to decreased TPR. A disturbing fact remains in that seemingly contradictory physiologic adjustments resulted during psychological stress following exercise. It appears that while TPR decreased, there is increased myocardial oxygen consumption as verified by determination of rate pressure product (RPP) following the exercise and control sessions. However, RPP measures were not computed statistically, thus must remain speculative. Therefore, exercise appears to have a

vasodilatory effect resulting in decreased TPR during psychological stress. However, due to limitations of this study one cannot interpret this as invasive measurement techniques such as blood volume testing, and strict observation of fluid loss during exercise are needed to make concrete conclusions.

This subject population, elite cyclists, may lead one to wonder about the implications and generalizations for other populations, ie., healthy middle-aged males who are a prime target for CVD. For example, CVD is a disease that progresses silently, usually unknown to the person. It often does not manifest itself until late in the diseased state, usually in middle-age, thereby increasing the danger. Additionally, stress (increased CVR) at the workplace may also be enhanced for this population. The applicability for decreased CVR to mental stress following an acute bout of exercise for other populations, middle-aged males for example, may provide great insight for a more healthy lifestyle and possibility the reduction for CVD risk through lessened CVR. There would also be a difference between those who are sedentary and those who exercise regularly, ideally those in the latter group would be at a lesser risk. However, it would be interesting to differentiate the difference in CVR between these two groups as well as other populations. However, separate study designs would be

needed before conclusive evidence could be determined. The results from this study would not be easily generalizable to other populations.

Results from this study provide limited support that acute aerobic exercise effectively reduces CVR to psychological stress; additionally, these results may also suggest that anticipatory response to non-exercise stressors may be reduced following exercise. The possibility that exercise may provide an invaluable intervention to CHD and/or it's progression is of utmost importance since CVD remains to be the number one killer in the United States today. Therefore, additional work in this area needs to be concluded.

Table 1. Table of Subject Characteristics

	<u>Cold Pressor</u>	<u>Stroop Color Word</u>
Weight (kg)	71.9 (6.0)	70.7 (5.0)
VO2 Max (ml/kg/min)	57.9 (8.8)	60.7 (6.9)
Max HR (bpm)	214.8 (15.0)	214.1 (15.6)
Max Workload (W)	260.0 (45.0)	235.0 (43.0)
<u>Acute Exercise Data:</u>		
VO2 (ml/kg/min)	41.0 (3.2)	43.4 (3.8)
HR (bpm)	150.3 (11.0)	153.3 (12.0)
Workload (W)	181.5 (27.0)	170.7 (28.2)
Values are M(SEM)	N=11	N=13

Table 2. Hypothesis One-Main Effect of Treatment on CVR
N=24

		<u>(exercise)</u>		<u>(control)</u>		<u>F</u> (1, 22)	<u>p</u>
		<u>x</u>	<u>SEM</u>	<u>x</u>	<u>SEM</u>		
SBP	Baseline	110.22	(1.27)	114.01	(1.01)	4.86	.0383
	Peak	113.97	(1.44)	118.61	(1.23)	.01	.9370
	Change 1	9.98	(1.37)	10.74	(1.74)	.15	.2052
	Change 2	2.10	(1.34)	6.10	(1.09)	.01	.9370
	Recovery	113.97	(1.44)	118.61	(1.35)	5.18	.0329
DBP	Baseline	66.62	(1.14)	68.63	(1.12)	1.65	.2121
	Peak	77.00	(2.25)	81.38	(2.05)	1.87	.2329
	Change 1	9.65	(2.16)	12.70	(1.92)	1.02	.3254
	Change 2	6.20	(2.40)	11.31	(2.10)	1.87	.1862
	Recovery	69.34	(2.04)	70.30	(2.01)	.11	.7420
HR	Baseline	65.94	(1.23)	57.95	(1.80)	20.78	.0002
	Peak	64.75	(2.18)	57.92	(2.45)	22.35	.0001
	Change 1	10.70	(0.93)	10.00	(0.98)	16.30	.0006
	Change 2	14.21	(1.98)	8.12	(1.99)	22.35	.0001
	Recovery	62.77	(2.18)	56.91	(2.08)	5.23	.0322

Values are: SBP and DBP=mm Hg (* p<.05)
HR=bpm

Baseline=avg of 5 minutes pre-task

Change 1=peak-baseline reactivity

Change 2=peak-screening reactivity

Recovery=avg of first 2 minutes post-task

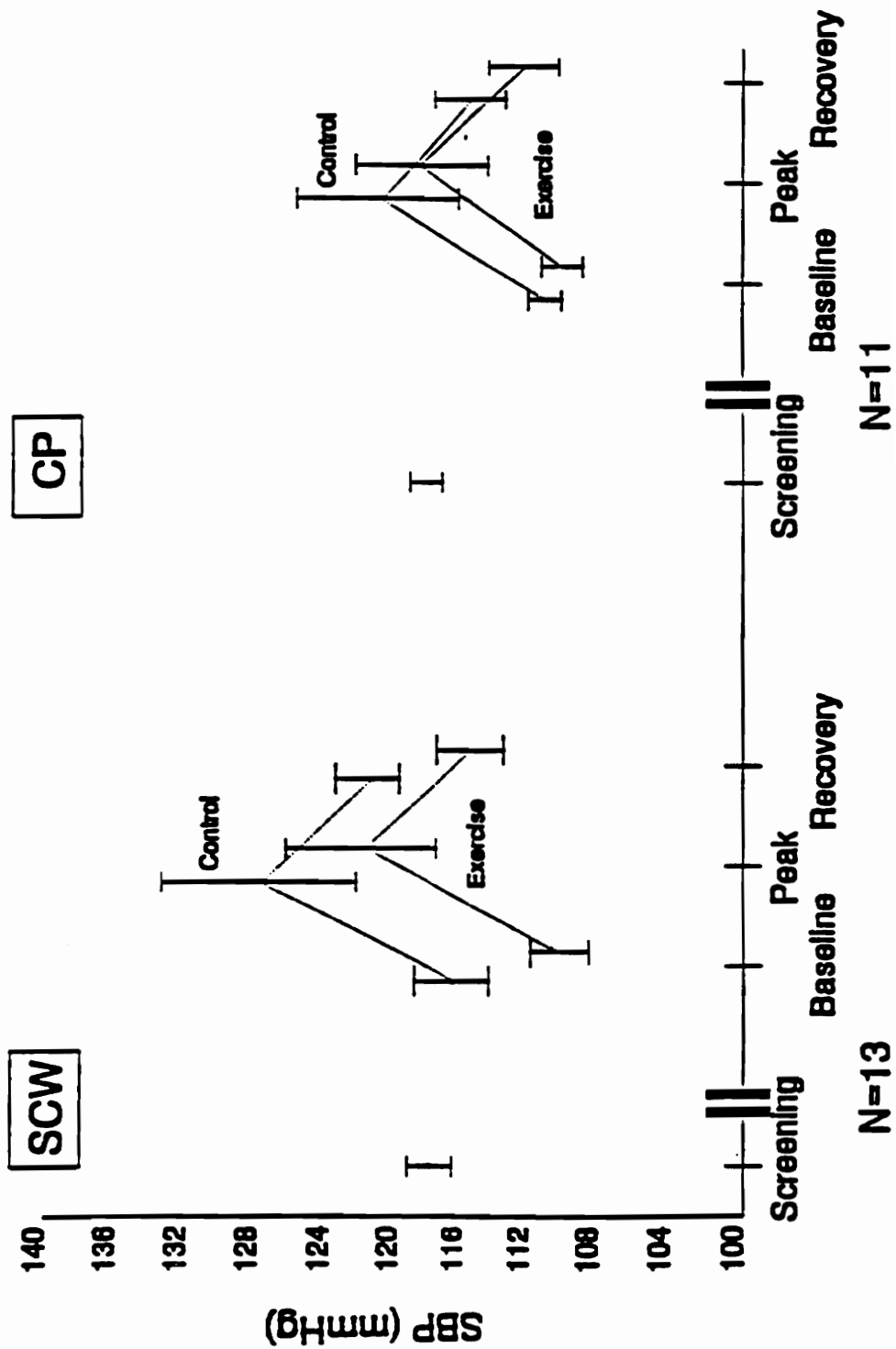


Figure 1. Systolic Blood Pressure Reactivity Measures

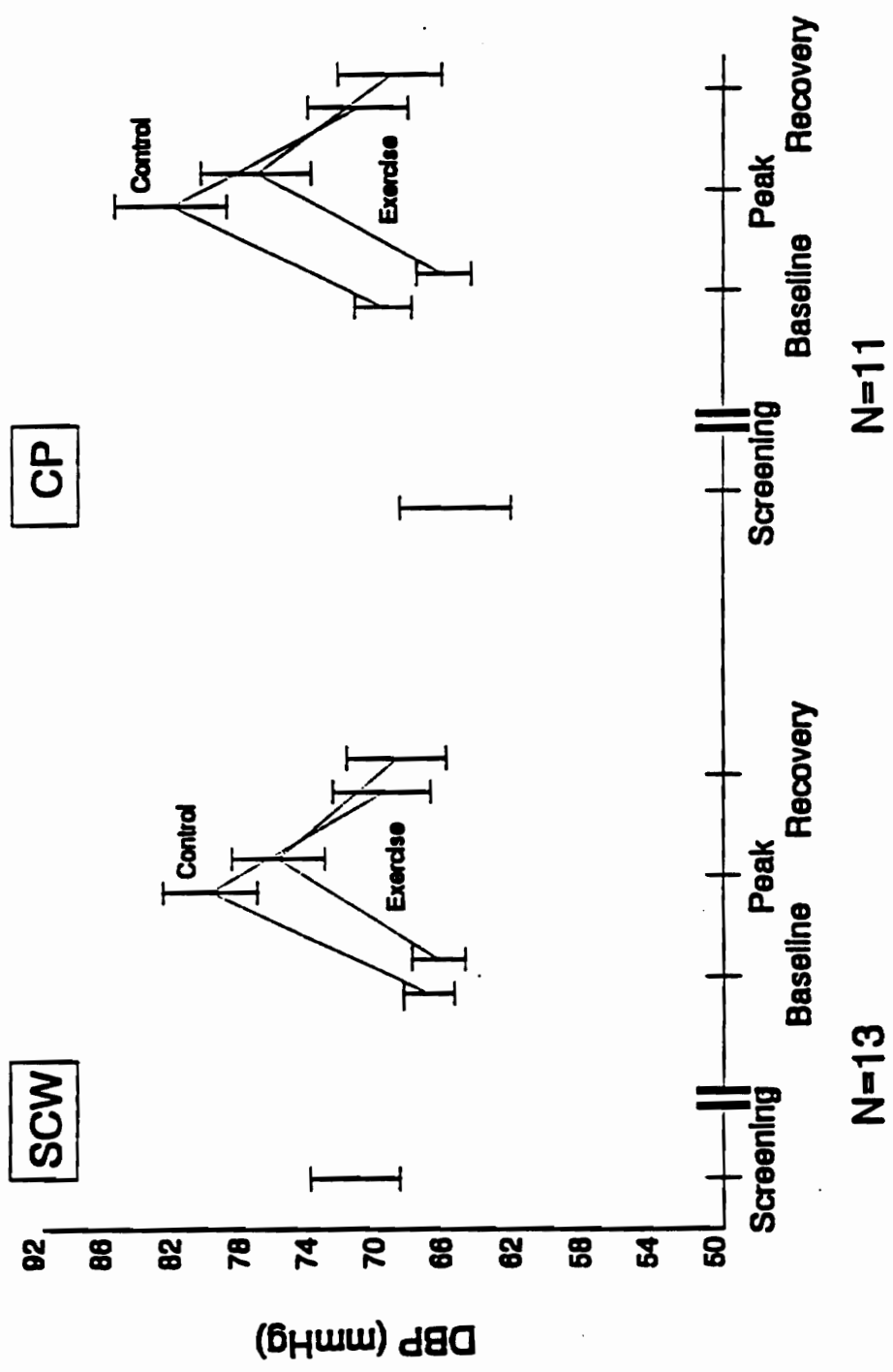


Figure 2. Diastolic Blood Pressure Reactivity Measures

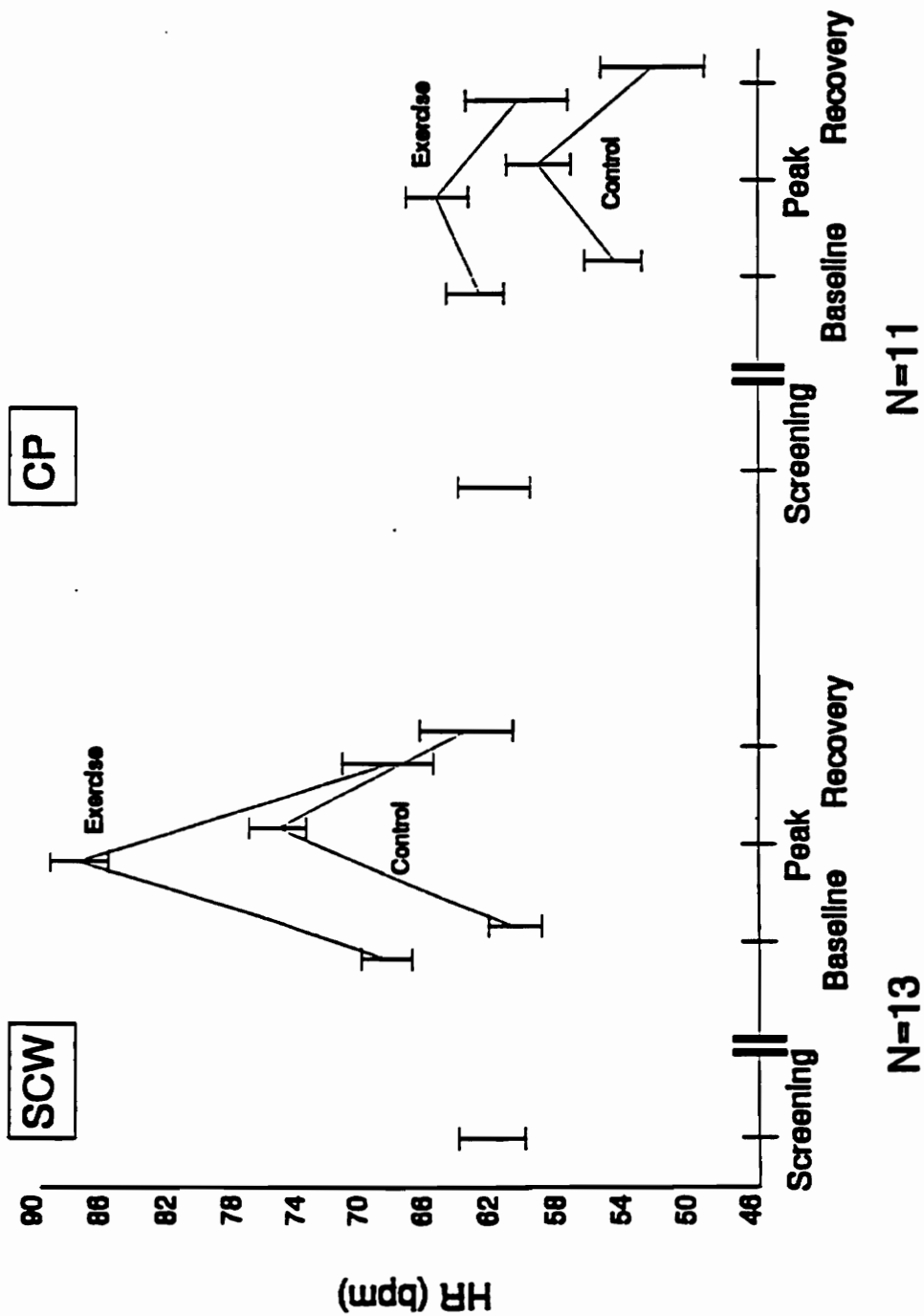


Figure 3. Heart Rate Reactivity Measures

REFERENCES

- Barnard, R. J. (1983). Effects of an intensive exercise and nutrition program in patients with coronary artery disease; Five year follow-up. Journal of Cardiac Rehabilitation, 3, 183.
- Bittker, Buchsbaum, Williams, & Wynne (1975). Cardiovascular and neurophysiologic correlates of sensory intake and rejection: II. Interview Behavior, Psychophysiology, 12, 434-438, 1975.
- Blumenthal, J.A., Emery, C.F., Walsh, M.A., Cox, D.R., Kuhn, C.M., Williams, R.B. & Williams, R.S. (1988). Exercise training in healthy, type A, middle-aged men: Effects on behavioral and cardiovascular responses. Psychosomatic Medicine, 50, 418-433.
- Blumenthal, J. A., McCubbin J. A. (1987). Physical exercise and stress management. In Handbook of Psychology and Health: Stress. Hillsdale, N. J. Lawrence Erlbaum Association, Hillsdale, N. J.
- Blumenthal, J. A., Fredrikson, M., Kuhn, C.M., Ulmer, R.L., Walsh-Riddle, M., & Applebaum, M. (1990). Aerobic exercise reduces levels of cardiovascular and sympathoadrenal responses to mental stress in subjects without prior evidence of myocardial infarction. American Journal of Cardiology, 65, 93-98.
- Bruce, R.A., Hossack, K.F., de Rouen, T.A., & Hofer, V. (1983). Enhanced risk assessment for primary coronary heart disease events by maximal exercise testing: 10 years' experience of Seattle heart watch. Journal of the American College of Cardiology, 2, 565-573.
- Crews, D.J. & D.M. Landers. (1987). A Meta-analytic review of aerobic fitness and reactivity to psychosocial stressors. Medicine and Science in Sports and Exercise, 19, (5), 114- 120.
- deGeus, E. J. C., vanDoornen, L. J. P., deVisser, D. C. & Orlebeke, J. F. (1990) Existing and training induced differences in aerobic fitness: Their relationship to physiological response patterns during different types of stress. Psychophysiology, 27, 457-478.

- Dembroski, T.M., & MacDougall, J.M. (1983). Behavioral and psychophysiological perspectives on coronary-prone behavior. In T.M. Dembroski, T.H. Schmidt, & G. Blumchen (Eds.), Biobehavioral Bases of Coronary Heart Disease: Karger Biobehavioral Medicine Series 2. New York: Karger.
- Dimsdale, J.E., Alpert, B.S., & Schneiderman. (1986). Exercise as a modulator of cardiovascular reactivity. In Handbook of Stress and Cardiovascular Reactivity New York: John-Wiley & Sons.
- Duda, J.L., Sedlock, D.A., Melby, C.L., & Thaman, C. (1988). The effects of physical activity level and acute exercise on heart rate and subjective response to a psychological stressor. International Journal of Sport Psychology, 19, 119-133.
- Ebbeson, B.L., Prkachin, K.M., & Mills, D.E. (1989). Effects of acute exercise in cardiovascular reactivity. Presentation at the Tenth Annual Meeting of the Society of Behavioral Medicine, San Francisco, CA, March-April, 1989.
- Hemesath, S. (1990). Effects of Acute Aerobic Exercise on Cardiovascular Reactivity to Stress in Healthy, Untrained Males. Thesis: Virginia Polytechnic Institute and State University.
- Hollander, B. J., & Seraganian, P. (1984). Aerobic fitness and psychophysiological reactivity. Canadian Journal of Applied Physiology, 3, 257-261.
- Holmes, D.S. & Roth, D. L. (1985). Association of aerobic fitness with pulse rate and subjective responses to psychological stress. Psychophysiology, 22, (5), 525-529.
- Hull, E. M. Young, S. H., & Ziegler M. G. (1984). Aerobic fitness affects cardiovascular and catecholamine responses to stressors. Psychophysiology, 21, 353-360.
- Kavanaugh, T. (1979). Prognostic indexes for patients with ischemic heart disease enrolled in an exercise-centered rehabilitation program. American Journal of Cardiology, 44, 1230-1238.

- Keller, S. & Seraganian, P. (1984). Physical fitness level and autonomic reactivity to psychosocial stress. Psychosomatic Research, 28, 279-287.
- Keys, A., Taylor H.L., Blackburn, H., Brozek, J., Anderson, J.T., & Simonson, E. (1971). Mortality and coronary heart disease among men studied for 23 years. Archives of Internal Medicine, 128, 201-214.
- Lake, B. W., Suarez, E. C., & Tocci, N. (1985). The type A behavior pattern, physical fitness, and psychophysiological reactivity, Health Psychology, 4, 169-187.
- Light, K.C., Obrist, P.A., Sherman, A.J., & Strogatz, D.S. (1987). Cardiovascular responses to stress: II. Relationships to Aerobic Exercise patterns. Psychophysiology, 24, 79-86.
- Martinez, L.R. (1990). Fitness level and stress response to a Cold Pressor test. Thesis: University of South Carolina.
- Maseri, A. (1978). Coronary vasospasm as a possible cause of myocardial infarction. A conclusion derived from the study of "preinfarction" angina. New England Journal of Medicine, 299, 1271-1282.
- May, G.S. (1982). Secondary prevention after myocardial infarction: A review of long term trials. Prognostic Cardiovascular Disease, 24, 331-335.
- Morpugo, E. (1968). Pharmacological modifications of sympathetic responses elicited by hypothalamic stimulation in the rat. British Journal of Pharmacology, 34, 532-542.
- Morris, J. N. (1953). Coronary heart disease and physical activity of work. Lancet, 2, 1053-1062.
- Mundel, R., Erikssen, J., & Rodahl, K. (1987). Assessment of physical activity by questionnaire and personal interview with particular reference to fitness and coronary mortality. European Journal of Applied Physiology, 56, 245-252.
- Obrist, P.A. (1981). Cardiovascular Psychophysiology: A perspective, New York: Plummer Press.

- Paffenbarger, Jr., R.S. Hyde R.T., Wing, A.L. & Hsieh, C. (1986). Physical activity, all-cause mortality, and longevity of college alumni. New England Journal of Medicine, 314, (10), 605-613.
- Powell, K. E. Physical activity and the incidence of coronary heart disease. Annual Review of Public Health, 8, 253.
- Raven, P.B., Niki, I., Dahms, T.E., & Horvath, S.M. (1970). Compensatory cardiovascular responses during an environmental cold stress, 5 degrees C. Journal of Applied Physiology, 29, (4), 417-421.
- Rejeski, W.J., Gregg, E., Thompson, A., & Berry, M. (1990). The effects of varying doses of acute exercise on psychophysiological stress responses in highly trained cyclists. Unpublished Manuscript.
- Sherwood, A., Light, K.C., & Blumenthal, J.A. (1989). Effects of aerobic exercise training on hemodynamic responses during psychosocial stress in normotensive and borderline hypertensive Type A men: A preliminary report. Psychosomatic Medicine, 51, 123-136.
- Sinyor, D., Schwartz, S.G., Peronnet, F., Brisson G., & Seraganian, P. (1983). Aerobic fitness level and reactivity to psychosocial stress: Physiological, biochemical, and subjective measures. Psychosomatic Medicine, 45, 205-216.
- Sobolski, J.C., Kolesar, J.J., Kornitzer, M.D., de Backer, G.G., Mikes, Z., Dramaix, M.M., Degre, S.G., & Denolin, H.F. (1988). Physical fitness does not reflect physical activity patterns in middle-aged workers (part b). Medicine Science Sports Exercise, 20, 6-13.
- Steptoe, A., and Roy, M. (1991). Cardiovascular responses to mental stress are suppressed following an episode of physical exercise (abstract). 12th annual meeting of Society of Behavioral Medicine, Washington, D.C., March 1991.
- Surwitt, R. S. (1986). Pharmacologic and behavioral modulators of cardiovascular reactivity: an overview. In K.A. Matthews, S. M. Weiss, T. Detre, T.M.

- Surwitt, R. S. (1986). Pharmacologic and behavioral modulators of cardiovascular reactivity: an overview. In K.A. Matthews, S. M. Weiss, T. Detre, T.M.
- Ward, M. M., Carmelli, D., & McElroy, M.R. (1984). Exercise effects on cardiovascular responses and reaction time performance (abstract). Vol. 21 (5), 603.
- Williams, Jr., R.B. (1986). Patterns of reactivity and stress. In Matthews, K.A., Weiss, S.M., Detre, T., Dembroski, T.M., Falkner, B., Manuck, S.B., & Williams, R.B. (Eds.), Handbook of Stress Reactivity and Cardiovascular Disease. New York: John Wiley and Sons, Inc.

CHAPTER IV

Summary and Research Recommendations

Summary

This study was conducted to determine the effects of acute aerobic exercise on reduced cardiovascular reactivity to psychological stress in trained males as well as determining possible mechanisms for such effects. Specifically, this study evaluated the effects of a 30-minute aerobic exercise session at 70% VO_2 max on SBP, DBP and HR responses to the Cold Pressor or Stroop Color Word psychological tasks in trained cyclists.

Twenty-four subjects were recruited from the Virginia Tech cycling club and various city wide cycling clubs. All subjects were screened according to training frequency and qualified subjects participated in three experimental sessions. Each subject was requested to follow to study adherences. These subjects were to abstain from exercise, alcohol or ingest analgesics 24 hours prior to each session. Additionally, they were not to eat or drink (except for water) three hours prior to each exercise session.

Each subject was randomly assigned to his experimental session and then participated in session I. This consisted of completing informed consent forms, health history questionnaires and preliminary instructions as to the

experimental session. Screening values for the assigned stressor were administered which was then followed by the maximal aerobic capacity test on the cycle ergometer. Screening reactivity testing consisted of the collection of baseline, peak and recovery values. Baseline values were determined after the subject sat resting quietly in the chair for 15 minutes. The last five minutes were averaged to determine baseline values. The assigned stressor was then administered for each subject. For the CP task, the subject immersed his dominant arm into cold water (1-3 degrees Celsius) for one minute, while the SCW participants performed a color word identification game on a computer screen for three minutes. Peak measures for the CP task were simply the recorded value taken for the minute. Peak measures for the SCW were determined by averaging the three values recorded for each minute. Change reactivity scores were calculated by subtracting the baseline from the peak reactivity values. Resting measures for each stressor were concluded by averaging the first two minutes of a five minute resting recovery period following by each stressor. At the conclusion of each stressor session, each subject completed a post-session questionnaire which inquired about the subjects' perceived difficulty, actual difficulty and subjects' fear of computers or cold. A priori for subject

Next, the subjects participated in a graded maximal aerobic capacity test on a bicycle ergometer. An ACSM protocol was used for appropriate exercise intensities and gradations.

Each subjects then participated in sessions II and III which consisted of either the exercise or control session, depending on how each subject was randomly assigned. The exercise session consisted of 30 minutes aerobic exercise at 70% VO_2 max (determined from the result of the maximal aerobic capacity test). One hour following each experimental session, the subject reported to behavioral physiology laboratory and underwent his assigned stressor again. CVR baseline, peak, change and recovery scores were determined following the same protocol as that for the screening session.

The Statistical Analysis System (SAS) was used to perform all statistical analyses involved in this study. Significance for all analysis was set a priori at an alpha level of 0.05. One-way ANOVAs were run on each of the following: weight, VO_2 max, max HR and max workload. One-way ANOVA analyses were also run on the acute aerobic exercise data which included exercise VO_2 , HR and watts. Results showed that subjects did not differ significantly in weight, or max VO_2 . Likewise, subjects did not differ in exercise data as insignificant results were yielded for

these measures. Test-rest reliability estimates were determined by comparing screening baseline and control session baseline measurements for SBP, DBP and HR. In addition, peak CVR was also compared using test-retest reliability estimates for control and screening sessions. These values ranged from .60-.70 for SBP, DBP and HR values which is consistent with past research. A split-plot specialized repeated measures ANOVA was conducted for the main analysis to determine and compare changes in CVR from exercise and control sessions. Specifically, this was a two-sequence, two-period cross-over design. For the main effects of treatment (the differences in exercise vs. control sessions), exercise showed reduced CVR for baseline and recovery SBP values. No significant reductions were seen for DBP or HR measures as originally hypothesized, though trends for attenuated DBP following exercise did exist. Interaction effects revealed significance in SBP baseline and scores and change 2 scores for HR measurements. Other SBP and HR, as well as all DBP values, were insignificant.

Research Implications

Results of this study had limited significance that exercise had a modulating effect on SBP, DBP or HR as Rejeski et al., 1990 or Steptoe. et. al., 1991, found. However, baseline and recovery SBP were significantly

different ($p < .05$) between the two treatments. DBP baselines had similar tendencies, though not to a significant level. This point prompted a re-analysis using change scores (peak-screening baselines) to statistically determine a possible reduced *anticipatory* response following exercise and found SBP to be significantly different. This may have relevant implications regarding the benefits of exercise on excessive CVR as well as further determinations of the mechanisms involved.

Previously mentioned studies finding significant results were a result of addressing most of former methodological problems. These included inappropriate intensity and/or durations for the exercise session, HR being used as the primary index of CVR and variability in perceived level of fitness. Using heterogeneous subject groups also may be a weakness since low fit individuals react with greater CVR to psychological stress than high fit. The three recent acute studies finding positive results utilized homogeneous groups, either untrained or trained.

In hopes of improving upon the results of past research, this study addressed and adjusted for these factors by: 1) utilizing the proper exercise intensity to elicit appropriate hemodynamic changes, as well as a duration (70% VO_2 max) that may be easily generalizable to a

sedentary or untrained population (30 minutes). 2) two differently mediated non-exercise stressors were used. 3) a homogeneous subject group was used in order to ensure similar CVR responses to psychological stress. 4) The use of SBP, DBP and HR to measure CVR responses. Even though the aforementioned trends resulted from this study, methodological changes made in this study failed to result in statistical significance pertaining to the main hypotheses.

Several considerations may be plausible explanations for this. Values of true screening baseline hemodynamics measurements may have been too variable. Each subject reported for screening measurements for one session only which was at high risk for biases.

Additional variability may have resulted in technician differences and possible equipment limitation, specifically the automated BP cuff in which several readings were questionable.

Subjects in this study were taken from a homogeneous subject pool. However, several factors lead this investigator to believe that perhaps some heterogeneity may have been evident. For example, differences in training phases were possible. Considering the nature of the study, extraneous mental pressures on the cyclists may have affected CVR responses. The time of school year (finals

week for some), may have resulted in additional worries about academics (ie., exams, papers). Other personality considerations such as purpose/motivation of training, their history of performance under pressure and level of competition may have all affected CVR and their ability to cope to such stress. Past experience may have also influenced results. Further gleaning into the competitive personality may be the question as to how one responds to stress.

Speculating upon these psychological and physiological relationships may provide insight into explanation for variable CVR responses. However, Crews (1987) points out, that time might be better spent examining underlying mechanisms for aerobic fitness effects. Thus, it may be possible to determine how differences in fitness actually reduce stress-response or time in stress. Aerobic exercise may cause a decrease in sympathetically mediated CV responses to psychological stress. The mechanism for this hypothesis remains unclear, however. For example, one author speculated that there may be an decreased sensitivity to beta-adrenergic stimulation (Light, 1982) while others suggest that hemodynamic training adaptations, cerebral heating and increases in core temperature which stimulate brain metabolism or increases in serotonin levels may also

be possible mechanisms for decreased CVR to non-exercise stress (Schulhan, Scher, & Furedy, 1986).

deGeus, vanDoornen, deVisser, & Orlebeke, (1990) suggest that an underlying genetic component may exist which in turn may alter aerobic power capacities. If this is true, perhaps VO_2 max values which were used to determine exercise session intensities were confounded.

Finally, reductions in anticipatory response also carry some considerations. While it may be appeared from this study that modulations in anticipatory response occurred, one must examine the time period in which the excessive CVR transpired. For example, some might react to the *thought* (ie., anticipatory response) of stress and respond minimally to the actual task. In contrast, others may not have an anticipatory response to the stress, but the major hemodynamic changes may take place during the actual task. The design of this study was not set up in order to make this distinction.

Finally, HR values were surprisingly highly variable. The idea that increased may have resulted in order to balance out decreased BP values in order to maintain cardiac output may be one explanation. However, other researchers in which positive findings and trends were reported found HR to be an invalid index for CVR (Hemesath, 1990; Rejeski et al., 1990) mostly because of a lack of time to establish

stable baselines previous to the psychological stressor. Even though one hour was established between exercise and behavioral session, it probably was not enough to establish a stable HR baseline.

Recommendations for Future Research

The relationship between CVR and exercise is a fairly new area of interest. However, the results from this study point to the need for further research for which the following recommendations and changes in design are advised.

Reliable screening measures are necessary. If research circumstances permit, perhaps this could be achieved if each subject were to report for resting values for three or more (preferable five) consecutive days at the same time each day. These values could be averaged to determine screening values. Additionally, actual testing values should be administered at the same time as well. To further ensure reliable measures, a fourth post-exercise session with no stressor following may provide a more reliable measure as there would be no stress expectations.

Homogeneous subject groups, as seen by differences in stress response with varying fitness levels, are of utmost importance. It appears that the subjects used here were homogeneous. Specific factors however, such as past experience, competitive experience and competitive personality characteristics may affect CVR responses within

the same subject pool and thus alter results. A preliminary questionnaire inquiring of about such considerations may be helpful to differentiate differences between individuals. In addition, stressors which simulate the manner in which people handle everyday stress are critical when trying to understand the relationship between CVR, exercise and CHD.

Even though SBP, DBP, in addition to HR were used in this study to determine CVR changes in psychological stress due to exercise, it has been noted that other measures may be necessary to glean deeper understanding into the mechanisms involved (Crews, 1987). Other measures such as core temperature, catecholamine responses (ie., epinephrine, norepinephrine and cortisol), stroke volume and cardiac output which more directly tie the connection should be examined.

The issue of a possible underlying genetic component (deGeus et al., 1990) may be addressed by the estimation of anaerobic threshold (ie., via ventilatory equivalent). Given such information, it would be possible to differentiate between VO_2 and anaerobic threshold, thereby overriding the confusion regarding the genetic component issue.

The idea of modulated anticipatory response is fairly new and warrants more research. However, it may be advisable for future designs to examine when excessive CVR

occurs among individuals. In other words, steps should be taken to observe changes in CVR prior to the task, during the task as well as time spent elevated. This would be important for more appropriately applying exercise prescription for reducing the effects of chronically elevated CVR.

Finally, future research should have methods for controlling for extraneous variables which may adversely affect BP and HR measures prior to testing situations.

Results from his study indicate the need for further research this area. Further probing into the notion of a reduced anticipatory response to a non-exercise stressor is especially needed as findings in this study pointed toward this notion. The implications that would result could be valuable. Possible extraneous hemodynamic variability, individual response differences, and the lack of other measures may help explain some of insignificance in this study. The possibility that exercise does not in fact reduce CVR responses in trained cyclists also exists. Therefore, the recommendations mentioned above should be employed to further explore the beneficial effects of acute aerobic exercise on CVR to psychological stress.

BIBLIOGRAPHY

- Barnard, R. J. (1983). Effects of an intensive exercise and nutrition program in patients with coronary artery disease; Five year follow-up. Journal of Cardiac Rehabilitation, 3, 183.
- Billman, G. E., Schwartz P.J., Stone H. L. (1984). The effects of daily exercise on susceptibility to sudden cardiac death. Circulation, 69, 118-28.
- Bittker, Buchsbaum, Williams, & Wynne (1975). Cardiovascular and neurophysiologic correlates of sensory intake and rejection: II. Interview Behavior, Psychophysiology, 12, 434-438, 1975.
- Blumenthal, J.A., Emery, C.F., Walsh, M.A., Cox, D.R., Kuhn, C.M., Williams, R.B. & Williams, R.S. (1988). Exercise training in healthy, type A, middle-aged men: Effects on behavioral and cardiovascular responses. Psychosomatic Medicine, 50, 418-433.
- Blumenthal, J. A., McCubbin J. A. (1987). Physical exercise and stress management. In Handbook of Psychology and Health: Stress. Hillsdale, N. J. Lawrence Erlbaum Association, Hillsdale, N. J.
- Blumenthal, J. A., Fredrikson, M., Kuhn, C.M., Ulmer, R.L., Walsh-Riddle, M., & Applebaum, M. (1990). Aerobic exercise reduces levels of cardiovascular and sympathoadrenal responses to mental stress in subjects without prior evidence of myocardial infarction. American Journal of Cardiology, 65, 93-98.
- Bruce, R.A., Hossack, K.F., de Rouen, T.A., & Hofer, V. (1983). Enhanced risk assessment for primary coronary heart disease events by maximal exercise testing: 10 years' experience of Seattle heart watch. Journal of the American College of Cardiology, 2, 565-573.
- Crews, D.J. & D.M. Landers. (1987). A Meta-analytic review of aerobic fitness and reactivity to psychosocial stressors. Medicine and Science in Sports and Exercise, 19, (5), 114-120.

- deGeus, E. J. C., vanDoornen, L. J. P., deVisser, D. C. & Orlebeke, J. F. (1990) Existing and training induced differences in aerobic fitness: Their relationship to physiological response patterns during different types of stress. Psychophysiology, 27, 457-478.
- Dembroski, T.M., & MacDougall, J.M. (1983). Behavioral and psychophysiological perspectives on coronary-prone behavior. In T.M. Dembroski, T.H. Schmidt, & G. Blumchen (Eds.), Biobehavioral Bases of Coronary Heart Disease: Karger Biobehavioral Medicine Series 2. New York: Karger.
- Dimsdale, J.E., Alpert, B.S., & Schneiderman. (1986). Exercise as a modulator of cardiovascular reactivity. In Handbook of Stress and Cardiovascular Reactivity New York: John-Wiley & Sons.
- Duda, J.L., Sedlock, D.A., Melby, C.L., & Thaman, C. (1988). The effects of physical activity level and acute exercise on heart rate and subjective response to a psychological stressor. International Journal of Sport Psychology, 19, 119-133.
- Ebbeson, B.L., Prkachin, K.M., & Mills, D.E. (1989). Effects of acute exercise in cardiovascular reactivity. Presentation at the Tenth Annual Meeting of the Society of Behavioral Medicine, San Francisco, CA, March-April, 1989.
- Freed, C.D., Thomas, S.A., Lynch, J.J., & Friedman, S.R. (1989). Blood pressure, heart rate and heart rhythm changes in patients with heart disease while talking. Heart Lung, 18, 17-22.
- Goldman, L. & Cook, E. F. (1984). The decline in ischemic heart disease mortality rates. Annals of Internal Medicine, 101, 825.
- Hartley, L.H., Mason, J.W., Hogan, R.P., Jones, L.G., Kotchen, T.A., Mougey, E.H., Wherry, L.H., Pennington, L.L., & Ricketts, P.T. (1972). Multiple hormonal responses to prolonged exercise in relation to physical training. Journal of Applied Physiology, 33, 607-610.
- Hemesath, S. (1990). Effects of Acute Aerobic Exercise on Cardiovascular Reactivity to Stress in Healthy, Untrained Males. Thesis: Virginia Polytechnic Institute and State University.

- Hollander, B. J., & Seraganian, P. (1984). Aerobic fitness and psychophysiological reactivity. Canadian Journal of Applied Physiology, 3, 257-261.
- Holmes, D.S. & Roth, D. L. (1985). Association of aerobic fitness with pulse rate and subjective responses to psychological stress. Psychophysiology, 22, (5), 525-529.
- Hull, E. M. Young, S. H., & Ziegler M. G. (1984). Aerobic fitness affects cardiovascular and catecholamine responses to stressors. Psychophysiology, 21, 353-360.
- Karasek, R., Baker, D., Marxer, F., Ahlbom, A., & Theorell, T. (1981). Job decision latitude, job demands and cardiovascular disease: A prospective study of Swedish men. American Journal of Public Health, 71, 694-705.
- Kavanaugh, T. (1979). Prognostic indexes for patients with ischemic heart disease enrolled in an exercise-centered rehabilitation program. American Journal of Cardiology, 44, 1230-1238.
- Keller, S. & Seraganian, P. (1984). Physical fitness level and autonomic reactivity to psychosocial stress. Psychosomatic Research, 28, 279-287.
- Kenney, W. L. (1985). Parasympathetic control of resting heart rate: Relationship to aerobic power. Medicine and Science in Sports and Exercise, 17, 451-460.
- Keys, A., Taylor H.L., Blackburn, H., Brozek, J., Anderson, J.T., & Simonson, E. (1971). Mortality and coronary heart disease among men studied for 23 years. Archives of Internal Medicine, 128, 201-214.
- Kramsch, W. L. (1981). Reduction of coronary atherosclerosis by moderate conditioning exercise in monkeys on an atherogenic diet. New England Journal of Medicine, 305, 1483-1495.
- Lake, B. W., Suarez, E. C., & Tocci, N. (1985). The type A behavior pattern, physical fitness, and psychophysiological reactivity, Health Psychology, 4, 169-187.

- Langer, A.W., McCubbin, J.A., Stoney C.A., Hutchison, J.S., Charlton, J.D., & Obrist, P.A. (1985). Cardiac adjustments during exercise and an aversive reaction time task: Effects of beta-adrenergic blockade. Psychophysiology, 22, 59-67.
- Light, K.C., Obrist, P.A., Sherman, A.J., & Strogatz, D.S. (1987). Cardiovascular responses to stress: II. Relationships to Aerobic Exercise patterns. Psychophysiology, 24, 79-86.
- Martinez, L.R. (1990). Fitness level and stress response to a Cold Pressor test. Thesis: University of South Carolina.
- Maseri, A. (1978). Coronary vasospasm as a possible cause of myocardial infarction. A conclusion derived from the study of "preinfarction" angina. New England Journal of Medicine, 299, 1271-1282.
- May, G.S. (1982). Secondary prevention after myocardial infarction: A review of long term trials. Prognostic Cardiovascular Disease, 24, 331-335.
- McCubbin, J.A., Richardson, J.E., Lauger, A.W., Kizer, J.S., & Obrist, P.A. (1983). Sympathetic neuronal function and left ventricular performance during behavioral stress in humans: The relationship between plasma catecholamine and systolic time intervals. Psychophysiology, 20, 102-110.
- Morpugo, E. (1968). Pharmacological modifications of sympathetic responses elicited by hypothalamic stimulation in the rat. British Journal of Pharmacology, 34, 532-542.
- Morris, J. N. (1953). Coronary heart disease and physical activity of work. Lancet, 2, 1053-1062.
- Mundel, R., Erikssen, J., & Rodahl, K. (1987). Assessment of physical activity by questionnaire and personal interview with particular reference to fitness and coronary mortality. European Journal of Applied Physiology, 56, 245-252.
- Neill, W. A., & Oxendine, J. M. (1979). Exercise can promote coronary collateral development without improving perfusion of ischemic myocardium. Circulation, 60, 1513-1520.

- Nixon, P.A., Robertson, L.H., & Epstein, L.H. (1986). Effects of aerobic fitness level and parental history of hypertension on cardiovascular reactivity to psychological stress. Medicine and Science in Sports & Exercise, 19, 456-463.
- Obrist, P.A. (1981). Cardiovascular Psychophysiology: A perspective, New York: Plummer Press.
- Paffenbarger, Jr., R.S. Hyde R.T., Wing, A.L. & Hsieh, C. (1986). Physical activity, all-cause mortality, and longevity of college alumni. New England Journal of Medicine, 314, (10), 605-613.
- Powell, K. E. Physical activity and the incidence of coronary heart disease. Annual Review of Public Health, 8, 253.
- Raven, P.B., Niki, I., Dahms, T.E., & Horvath, S.M. (1970). Compensatory cardiovascular responses during an environmental cold stress, 5 degrees C. Journal of Applied Physiology, 29, (4), 417-421.
- Rejeski, W.J., Gregg, E., Thompson, A., & Berry, M. (1990). The effects of varying doses of acute exercise on psychophysiological stress responses in highly trained cyclists. Unpublished Manuscript.
- Roth, D.L. (1989). Acute Emotional and Psychophysiological effects of aerobic exercise. Psychophysiology, 26, 593-602.
- Russell, P.O., Epstein, L.H., & Erikson, K.T. (1983). Effects of acute aerobic exercise and cigarette smoking on autonomic and neuromuscular responses to a cognitive stressor. Psychological Reports, 53, 199-206.
- Sherwood, A., Light, K.C., & Blumenthal, J.A. (1989). Effects of aerobic exercise training on hemodynamic responses during psychosocial stress in normotensive and borderline hypertensive Type A men: A preliminary report. Psychosomatic Medicine, 51, 123-136.
- Sinyor, D., Schwartz, S.G., Peronnet, F., Brisson G., & Seraganian, P. (1983). Aerobic fitness level and reactivity to psychosocial stress: Physiological, biochemical, and subjective measures. Psychosomatic Medicine, 45, 205-216.

- Sobolski, J.C., Kolesar, J.J., Kornitzer, M.D., de Backer, G.G., Mikes, Z., Dramaix, M.M., Degre, S.G., & Denolin, H.F. (1988). Physical fitness does not reflect physical activity patterns in middle-aged workers (part b). Medicine Science Sports Exercise, 20, 6-13.
- Steptoe, A., and Roy, M. (1991). Cardiovascular responses to mental stress are suppressed following an episode of physical exercise (abstract). 12th annual meeting of Society of Behavioral Medicine, Washington, D.C., March 1991.
- Surwitt, R. S. (1986). Pharmacologic and behavioral modulators of cardiovascular reactivity: an overview. In K.A. Matthews, S. M. Weiss, T. Detre, T.M. Dembroski, B. Falkner, S.B. Manuck, & R. B. Williams (Eds.), Handbook of Stress, Reactivity, and Cardiovascular Disease. New York: John Wiley and Sons, Inc.
- Ward, M. M., Carmelli, D., & McElroy, M.R. (1984). Exercise effects on cardiovascular responses and reaction time performance (abstract). Vol. 21 (5), 603.
- Williams, Jr., R.B. (1986). Patterns of reactivity and stress. In Matthews, K.A., Weiss, S.M., Detre, T., Dembroski, T.M., Falkner, B., Manuck, S.B., & Williams, R.B. (Eds.), Handbook of Stress Reactivity and Cardiovascular Disease. New York: John Wiley and Sons, Inc.
- Williams, R. S. (1980). Physical conditioning augments the fibrinolytic response to venous occlusion in healthy adults. New England Journal of Medicine, 302, 987.
- Wyatt, H. L., & Mitchell, J. (1978). Influences of physical conditioning and deconditioning on coronary vasculature of dogs. Journal of Applied Physiology, 45, 619-625.

APPENDIX A
METHODOLOGY

METHODOLOGY

Subject Selection

Twenty-four healthy college-aged males (19-26 years) were recruited from cycling clubs at Virginia Polytechnic Institute and State University for participation in a study which consisted of three exercise sessions taking place over one week. The sample size was determined from previous research (Ebbeson, 1989, Hemesath, 1990 and Rejeski, 1990). A power analysis was done in order to confirm this sample size. Specific criteria was required for participation:

- 1). healthy males between 18-28 years
- 2). free of cardiovascular disease, diabetes, Raynaud's syndrome, orthopedic and physical relative or absolute contraindications to exercise or psychological stress testing as stated in the ACSM guidelines
- 3). trained cyclists as defined by exercising more than four times per week, 30 minutes per session
- 4). willingness to adhere to study guidelines of abstinence of food, beverages, tobacco and analgesics prior to exercise sessions
- 5). non-smokers.

Instructional Procedures

At an organizational meeting subjects received oral and verbal explanation of the study. Risks and benefits were included. All subjects signed a written consent form (Appendix B) agreeing to the terms of the study and filled out a health history questionnaire form (Appendix C) as well to ensure that minimal risk was involved for each subject.

Experimental Procedures

Each subject was instructed to abstain from ingesting food 3 hours and alcohol and analgesics 24 hours prior to each session. Session 1 was a screening session which consisted of baseline CVR to the CP or SCW psychological tasks to which the subjects were randomly assigned. This session also consisted of a maximal exercise test on a bicycle ergometer to determine workloads imposed later in the experimental session. Upon arrival to the Behavioral Physiology Laboratory, each subject was questioned to ensure that he had conformed to pre-session adherences. He was then seated properly in the chair with the BP cuff placed just superior to the antecubital fossa of his non-dominant arm. Instructions were then given to each subject concerning either the CP task or SCW task (Appendix D). The subject then began his screening baseline measurements. Following each psychological stressor session, each subject filled out a post-session questionnaire inquiring about

perceived mental difficulty by the subject (see Appendix C). The maximal exercise test in the Human Physiology Laboratory then followed the stressor task in the first experimental session.

Baseline CVR

Baseline CVR for each subject to the psychological stressors were as follows:

Cold Pressor (CP): The subject sat quietly for 15 minutes in a chair with an automated blood pressure system (Industrial and Biomedical Sensors Corporation Model S-D 700 A cuff) on his non-dominant arm while his dominant hand was placed in water (0-3 degrees Celsius) for one minute. Resting measures of heart rate and blood pressure (systolic and diastolic) were determined by averaging the last five minutes prior to the task. After the task, the parameters were recorded for five minutes in which recovery measures were calculated by averaging the first and second minutes. A measured reaction of 5-30 mmHg DBP to the stressor was established a priori for eligibility for the study.

Stroop Color Word (SCW): The Stroop Color Word task consisted of a display on a computer screen in which the names of colors appeared in an ink that differed in color from the name. In addition, there was a cassette verbally announcing a different color not shown on the screen (written or displayed). For example, ("red" may have

appeared in blue ink, while the color "yellow" may have been heard on the tape). After sitting quietly for 15 minutes, resting measures of HR and BP were determined by averaging the five minutes prior to each task. The subjects then responded to the stressor as fast as possible for a three-minute session and the number of correct responses (to the color of the word) were recorded. HR, SBP and DBP were calculated by averaging the measure taken each minute. Each subject rested for five minutes in which recovery measures were recorded as in the CP task.

Maximal Exercise Test: A maximal exercise test was performed on a calibrated cycle ergometer (Bodyguard Model 990) in which an ACSM protocol was used based on weight and exercise level (see Appendix D). An on-line gas analysis was used in conjunction with the Hans Rudolph low resistance low dead space valve. Inspired volumes were measured by the Parkinson-Cowan dry gas meter. A visual display which reports minute ventilation and total ventilation to the nearest .1 liter was electronically integrated to the P-C meter. Inspired gasses passed through an Aquasorb absorbent. Oxygen and carbon dioxide were determined by Ametac Analyzers which were calibrated prior to each exercise session and HR was monitored each minute via the Lifepack 6 EKG system. Ratings of perceived exertion (RPE) were determined the last 15 seconds of each stage. Maximal

exercise capacity was attained when one or more of the following was achieved: with an increase in workload, oxygen consumption did not change or decreased, RPE reached 19 or 20, age-predicted maximal heart rate was attained (Karavonen method), and/or physical limitations by the subject made it impossible to continue at the required cadence (rpm=50). VO_2 max was computed on an IBM-PC. Subjects then went through a proper cool down and arrangements were made for the next session. 70% of the subject's maximal aerobic capacity was determined as well as estimated HR and workload for that level. This was calculated in order to determine the workload for the exercise session.

Exercise Sessions

Subjects were then randomly assigned to one of two exercise sessions. Forty-eight hours later, half of the subjects participated in a thirty minute exercise session at 70% VO_2 on a calibrated cycle ergometer. HR and BP was monitored and recorded every 5 minutes. Maintenance of 70% VO_2 max was assured during these recordings by taking measurements throughout the exercise bout. Fluid replacement was encouraged throughout the exercise session.

An additional 48 hours later, the subjects participated in the remaining session in which he was not initially assigned to. This consisted of a forty minute

attention control session in which the subjects underwent several health assessments. These included estimations of percent body fat, frame size, height/weight recommendations, and resting blood pressure and HR measurements.

Following each exercise session, each subject then underwent additional CVR testing to his randomly assigned stressor. These were counter-balanced across groups.

Research design

A randomized split-plot repeated measures design was used in this study. The subjects were randomly assigned to either the SCW or CP task as well as ordering for their experimental sessions. During their exercise session, they cycled at 70% VO_2 max for thirty minutes. The control session consisted of several health assessment measurements which included resting HR and BP, estimation of percent body fat, frame size estimation, height/weight recommendation, and a flexibility assessment.

Statistical Analyses

Reliability Estimates In order to estimate the consistency of measurements taken to assess true hemodynamic values, reliability estimates were calculated by comparing SBP, DBP and HR baseline measures for the control session with baseline values from the screening session.

External validity: The findings from this experimental study may be generalized to those individuals having similar

characteristics as that of the subject group; that is, males, 19-26 years old who cycle more than 4 times per week for at least 30 minutes per session.

Internal validity: Variance was controlled in several ways. First, subjects were oriented to the experimental trials. The equipment was maintained in proper running order and was calibrated before each subject was tested. Random selection was administered for subjects, exercise trials and psychological stressors. Additionally, the subjects were non-smokers and were instructed not to eat, drink, or exercise prior to each exercise trial. The subjects were also approximately the same age. These considerations helped eliminate variance among subjects.

Data Analyses

Significance for all analyses was set a priori at an alpha of 0.05. In order to determine group differences between exercise and control, one-way ANOVAs were conducted on age, weight, Max VO_2 , Max HR and Max workload. One-ways were also run on acute exercise data which included exercise VO_2 , HR, and Watts. Test- retest reliability estimates for SBP, DBP and HR for the baseline control session were calculated and compared with the same measures for the screening baseline session.

A split-plot specialized repeated measures ANOVA was conducted for the main analysis of treatment and stressor

code (SCW or CP) to determine and compare changes in CVR from exercise and control sessions. Specifically, this was a two-sequence, two-period cross-over design. (See Appendix E). This analysis was performed using both N=24 and N=20 sample sizes (the latter omitting those who were "non-reactors"). Results simulated each other, thus to increase power, results and discussion involved the larger sample size. An additional analysis using the same design was executed for the statistical determination of the alternate hypothesis where change scores involved peak and screening baseline measures. All data was analyzed using the Statistical Analysis System (SAS).

Conclusion

The results of this study indicate that thirty minutes of acute aerobic exercise at 70% VO_2 max does not effectively reduce CVR responses to psychological stress. However, based on these results, exercise may modulate excessive CVR resulting from one's anticipatory response of psychological stress.

APPENDIX B
INFORMED CONSENT

Division of Health and Physical Education
Virginia Polytechnic Institute and State University

INFORMED CONSENT

I, _____, do hereby voluntarily agree and consent to participate in a research project conducted by _____ of the Division of Health and Physical Education of Virginia Polytechnic Institute and State University.

Title of Study:

The effects of acute aerobic exercise on cardiovascular reactivity in response to psychological stressors on trained cyclists.

The purposes of this study include:

Aerobic exercise has been suggested to reduce cardiovascular reactivity and thereby provide a possible non-pharmacological intervention to reduced cardiovascular risk and disease. The purposes of this study will be to examine the effects of acute aerobic exercise on diastolic blood pressure (DBP), systolic blood pressure (SBP) and heart rate (HR) following the cold pressor (CP) and Stroop Color Word (SCW) psychological stressors. Previous methodological problems will be addressed and adjusted for in order to help determine possible mechanisms involved.

I voluntarily agree to participate in this research study.
It is my understanding that my participation will include:

1. Participation in an organizational meeting. The purpose of this meeting will be to explain the procedures of the study, sign an informed consent form, fill out a health history questionnaire.

2. Participation of the Cold Pressor (CP) or Stroop Color Word (SCW) psychological stressors on three separate occasions in which heart rate and blood pressure will be continually monitored. The CP test will involve placement of the dominant hand up to the wrist in cold water (0-3 degrees Celsius) for one minute. Blood pressure and HR will be recorded 50 seconds into the minute via the Industrial and Biomedical Sensors Corporation blood pressure system, Model SD-700 A. Readings will be displayed on a monitor with the blood pressure cuff is on the non-dominant arm. The SCW test will be a 3-minute stressor in which words of colors will flash on a computer screen at 1.5 second intervals. The colored word will appear on the screen in a color different color than the actual word. In addition, a cassette tape playing in the background will verbally be announcing a different color than the other two. This is to cause confusion (and thus changed CVR) as correct responses of the color of the word will be recorded. Appropriate responses

will be recorded by pressing the correct computer button. Resulting CVR (HR and BP) will be measured via the Industrial and Biomedical Sensors Corporation blood pressure system, Model SD-700 A at one minute intervals.

3. Participation in a maximal exercise test on a bicycle ergometer in which heart rate and blood pressure will be continually monitored and in which expired gases will be taken to determine VO₂ Max.

4. Participation in an exercise session on a bicycle ergometer for 30 minutes at 70% VO₂ Max. Heart rate and blood pressure will be continually monitored.

5. Participation in an attention control session in which several health assessments will be taken.

6. Adherence to guidelines such as abstinence from alcohol, food, beverages, and tobacco for pre-designated periods prior to the study.

I understand that participation in this experiment may produce certain discomforts and risks. These discomforts and risks include:

Possible discomforts during the CP task may occur as the subject places his hand in cold water up to the wrist (0-3 degrees Celsius) for one minute. This may involve some discomfort or pain. The maximal exercise test may produce some discomfort as the subject cycles until volitional fatigue. Possible risks may include, but are not limited to strains, fractures, immediate and/or delayed muscle soreness and a small possibility of death.

I understand that certain personal benefits may also be expected from participation in this experiment. These include:

I will gain knowledge related to my personal health via several health assessments such as blood pressure measurements, percent body fat, body girth measurements, height, and weight. In addition, aerobic capacity will be determined via a VQ₂ Max cycle ergometer test. This is especially beneficial as these tests are expensive in hospitals or other settings. Also, information regarding ventilatory equivalent. This is relevant as this has been suggested to be possible indicator of anaerobic threshold, thus providing information pertaining to one's training level.

I will gain insight into my cardiovascular and hypertension risk factors.

I may also request results of the study.

I understand that there may be one or more appropriate alternative procedures that might be advantageous to me. These include:

I may participate in stress tests at other facilities (ie., hospitals, fitness centers) on a cycle ergometer or treadmill. In addition, health history information via questionnaires as well as other health information is possible, however a fee is probable. Psychological stress testing is unique to our study and I am unaware of other places where this procedure is available.

I understand that any data of a personal nature will be held confidential and will be used for research purposes only. I also understand that these data may only be used when not identifiable with me.

I understand that I may abstain from participation in any part of the experiment or withdraw from the experiment should I feel the activities might be injurious to my health. The experimenter may also terminate my participation should he feel that the activities might be injurious to my health.

I understand that it is my personal responsibility to advise the researchers of any preexisting medical problem that may affect my participation or of any medical problems that might arise in the course of this experiment and that no medical treatment or compensation is available if injury is suffered as a result of this research. A telephone is available which would be used to call the local hospital for emergency service.

I have read the above statements and have had the opportunity to ask questions. I understand that the researchers will, at any time, answer my inquiries concerning the procedures used in this experiment.

Date _____ Time _____ a.m./p.m.

Participant signature _____

Witness _____

Project Director Susi Palmatier Telephone 231-4900

HPE Human Subjects Chairman Dr. Charles Baffi Telephone 231-8284

Dr. Ernie Stout, 301 Burruss Hall, 231-5281.

To receive the results of this investigation, please indicate this choice by marking in the appropriate space provided below. A copy will then be distributed to you as soon as the results are made available by the investigator. Thank you for making this important contribution.

_____ I request a copy of the results of this study.

APPENDIX C
SUBJECT DATA FORMS

HEALTH STYLE QUESTIONNAIRE

Name: _____ ID#: _____

Sex: _____ Age: _____ Phone #: _____

Please circle year in school: 1 = Freshman 2 = Sophomore
3 = Junior 4 = Senior

Medical History

Have you ever had:	<u>YES</u>	<u>NO</u>
Heart disease or heart problems	_____	_____
Lung disease or difficulty breathing	_____	_____
Difficulty with cold hands or feet	_____	_____
Stroke	_____	_____
Kidney disease	_____	_____
High cholesterol	_____	_____
High triglycerides	_____	_____
Diabetes	_____	_____
Raynaud's syndrome	_____	_____
Any operations (Type/Date: _____)	_____	_____
_____	_____	_____
_____	_____	_____
Have you ever had a blood pressure reading above normal (140/90)?	_____	_____
Have you ever been diagnosed as having hypertension?	_____	_____

Please list any medications you are currently taking: _____

Are you allergic to any medications, drugs or foods? Please list: _____

Has anyone in your family been diagnosed as having:	<u>YES</u>	<u>NO</u>	<u>RELATIONSHIP</u>	<u>AGE AT ONSET</u>
High blood pressure/hypertension	_____	_____	_____	_____
Heart attack or heart disease	_____	_____	_____	_____
Stroke	_____	_____	_____	_____
Diabetes	_____	_____	_____	_____
Kidney disease	_____	_____	_____	_____

Health Habits

	<u>NO</u>	<u>YES</u>
Drink caffeinated tea, coffee or soda	() _____	cups/day
Drink alcohol	() _____	drinks/day
Add salt to meal before tasting	() _____	
Smoke cigarettes	() _____	cigarettes/d
Sleep	() _____	hours/night

Exercise Habits

During the past 3 months have you engaged in physical exercise?

YES _____ NO _____

If yes, please list below any activities in which you participated during an average week in the past month (please include competitive, recreational or leisure time activities). Also include frequency and duration (or number of laps, miles, sets, games, etc.) of the activity. BE AS SPECIFIC AS POSSIBLE!!!

<u>Activity</u>	<u>Frequency</u> (times/week)	<u>Duration</u>
1. _____		
2. _____		
3. _____		
4. _____		
5. _____		

(If you need more space, continue on the back)

Do you have any orthopedic problems which may restrict your ability to participate in exercise consisting of stationary cycling?

YES _____ NO _____

If so, please explain: _____

The majority of the testing for this study will be taking place Monday through Saturdays, mostly during the days. Do you foresee a problem with being able to participate on these days?

YES _____ NO _____

If so, please indicate any specific days and/or times during which you are not available _____

This study requires the ability to abstain from food, beverages (except water), tobacco products and analgesics (aspirin, etc.) for up to three hours prior to all experimental sessions. Do you feel capable and willing to abide by these requests?

YES _____ NO _____

Name: _____

SSN: _____

Date: _____

Pre-Exercise Measures

PBar _____ mmHg

Weight _____ kg

Rest HR _____

Temp _____ C

BP _____

Exercise Session

MIN	WKLD	HR	BP	VIATPS	FeO2	FeCO2	RPE	VO2
1	_____	_____	_____	_____	_____	_____	_____	_____
2	_____	_____	_____	_____	_____	_____	_____	_____
3	_____	_____	_____	_____	_____	_____	_____	_____
4	_____	_____	_____	_____	_____	_____	_____	_____
5	_____	_____	_____	_____	_____	_____	_____	_____
9	_____	_____	_____	_____	_____	_____	_____	_____
10	_____	_____	_____	_____	_____	_____	_____	_____
15	_____	_____	_____	_____	_____	_____	_____	_____
19	_____	_____	_____	_____	_____	_____	_____	_____
20	_____	_____	_____	_____	_____	_____	_____	_____
29	_____	_____	_____	_____	_____	_____	_____	_____
30	_____	_____	_____	_____	_____	_____	_____	_____

BICYCLE EXERCISE TEST

Name: _____

SSN : _____

Date: _____

PRE-TEST MEASURES:

PBar _____ mmHg Wt _____ kg Rest HR _____ BPM Baseline $\dot{V}O_2$ _____ % $\dot{V}O_2$
Room Temp _____ C Rest BP _____ mmHg _____ % $\dot{V}CO_2$

EXERCISE MEASURES

MIN	WKLOAD	HR	BP	VIATPS(L)	FeO ₂	FeCO ₂	RPE
1	_____	_____	_____	_____	_____	_____	_____
2	_____	_____	_____	_____	_____	_____	_____
3	_____	_____	_____	_____	_____	_____	_____
4	_____	_____	_____	_____	_____	_____	_____
5	_____	_____	_____	_____	_____	_____	_____
6	_____	_____	_____	_____	_____	_____	_____
7	_____	_____	_____	_____	_____	_____	_____
8	_____	_____	_____	_____	_____	_____	_____
9	_____	_____	_____	_____	_____	_____	_____
10	_____	_____	_____	_____	_____	_____	_____
11	_____	_____	_____	_____	_____	_____	_____
12	_____	_____	_____	_____	_____	_____	_____
13	_____	_____	_____	_____	_____	_____	_____
14	_____	_____	_____	_____	_____	_____	_____
15	_____	_____	_____	_____	_____	_____	_____
16	_____	_____	_____	_____	_____	_____	_____

POST EXERCISE MEASURES

MIN	WKLOAD	HR	BP	VIATPS(L)	FeO ₂	FeCO ₂	RPE
1	_____	_____	_____	_____	_____	_____	_____
2	_____	_____	_____	_____	_____	_____	_____
3	_____	_____	_____	_____	_____	_____	_____
4	_____	_____	_____	_____	_____	_____	_____
5	_____	_____	_____	_____	_____	_____	_____
6	_____	_____	_____	_____	_____	_____	_____

SUBJECT HEALTH ASSESSMENT

NAME _____

ID _____

HEIGHT (CM/IN) _____

WEIGHT (KG/LB) _____

HEART RATE _____

BLOOD PRESSURE _____

FLEXIBILITY (CM) _____

FRAME SIZE _____

HT/WT REC _____

% BODY FAT: TRICEP _____

 BICEP _____

 CHEST _____

 THIGH _____

 ABDOMEN _____

 SUPRAIL. _____

 SUBSCAP. _____

% BODY FAT _____

#1 pretesting baseline
 #2 exercise session or
 #3 control session

EXPERIMENTAL SESSION # _____

NAME/ID _____
 DATE/TIME _____

1. Exercise today? Y N How much and when? _____
2. Alcohol today? Y N How much and when? _____
3. Caffeine today? Y N How much and when? _____
4. Tobacco today? Y N How much and when? _____
5. Cold medication? Y N How much and when? _____
 (and/or analgesics)
6. Any food w/in the past 3 hours? Y N How much and when? _____

BASELINE 1 (Inflate to 150)

ime	SBP	DBP	HR	Time	SBP	DBP	HR

TRESSOR 1 (Inflate to 180)

ime	SBP	DBP	HR

POST SESSION QUESTIONNAIRE

NAME/ID _____

DATE _____

SESSION # _____

1. How stressful did you perceive this task?

0-----1-----2-----3-----4-----5-----6

2. How well did you cope during this task?

0-----1-----2-----3-----4-----5-----6

3. Do you have a fear of computers/cold?

yes no

APPENDIX D
RESEARCH PROTOCOLS

Maximal Aerobic Capacity Test Protocol

<u>Stage</u>	<u>Workload</u>	<u>Minutes</u>
I	25 W	2
II	50 W	2
III	100 W	2
IV	150 W	2
V	200 W	2
VI	250 W	2
VII	300 W	2
VIII	350 W	2

General Instructions

1. Obtain subject's folder.
2. Use new stressor data sheet for each session and clearly identify as baseline, exercise, or control session.
3. Turn on the two main power switches to the left of the computer.
4. Turn on the computer following the posted instructions above the monitor, but stop at the BP-HOOK-UP prompt!!!DO NOT PRESS ENTER!!!!
5. Select correct stressor tape and place in the stereo, and set the counter to zero.
6. Remember to have the subject place both feet flat on the floor with his arms on the arm rests and emphasize minimal movement. Record any significant movement which is inappropriate during data collection on the data sheet.
7. After baseline sessions (Monday and Tuesday), escort the subject with his file to the exercise physiology lab.
8. Make sure that towels are present for the CP task.

Stroop Color Word Task

1. Touch the rim of the computer monitor to ground from static electricity.
2. Disconnect the black monitor cable and connect it into the computer, using the far right slot.
3. Turn on main power switch (on black power strip) and make sure both computer switches are on.
4. MAKE SURE CAPS LOCK IS ON !!!!!!!!!!!!!!!
5. Adjust contrast button all the way to the right to maximum brightness.
6. When the first screen appears press the ENTER key.
At the prompt, type "cd\stroop" and ENTER.
STROOP will appear, retype "stroop" AND DO NOT PRESS ENTER!
7. Turn the computer table at a diagonal angle.
8. Turn chair to face the computer.
9. Turn on main power switch for the camera and set to VCR.
Turn on the power switch on top of the camera. Put in the JY and SP videotape and make sure that it is rewound. Adjust the position of the camera to include the easy chair and computer screen. Slide open the door which covers the control buttons on top of the camera. Press play and record simultaneously to begin taping. Leave the door open.
10. After the subject arrives have him sit in the easy chair and ask him the preliminary test questions at the top of the data sheet.
11. Inquire about the subject's dominant arm, then hook up the blood pressure cuff to the nondominant arm. Set the blood pressure system for 1 minute intervals, at 150 mm Hg pump up pressure. Flip switch to HOLD position.
12. Tell the subject that:
 - a. They will be performing the task for 3 minutes.
 - b. They will need to identify the color of the words that flash up on the screen.
 - c. Further instructions will be given later in the tape.

- d. We will be video taping him and keeping his scores.
The best score will win \$50!!!!!!!!!!!!
13. Explain to the subject that you will be showing him samples of the five colors that are in the task. Make sure that the subject pays attention to the shades. Press ENTER and review each color with the subject.
 14. Set up for the task by:
 - a. Pressing #2 for the timed stimuli when the next screen appears.
 - b. Set the task for 1 second intervals and press ENTER.
 - c. Set the task for 3 minutes and DO NOT press ENTER.
 15. Cover the key board and place it in the subject's lap and tell him to remain as still as possible throughout the task.
 16. Leave the room and immediately start timing. Begin the tape. Stop the tape when you hear click on the tape (the click designating the start of recording). This is at approximately 43 on the tape counter.
 17. At minute 4, go back to the subject and start the BP system at minute 5 by turning the volume knob on and flip switch to test and then cycle. If the cuff does not inflate immediately, flip to test and then cycle again.
 18. Dash down to the observation room and press the ENTER key immediately.
 19. Begin collecting data at the 6th BP reading. Collect the next 5 readings for baseline measurement.
 20. As the 5th reading comes up on the screen restart the tape.
 21. Take the three blood pressure readings during the task.
 22. Turn the tape off at the conclusion of the task and take the next 5 recovery BP readings.
 23. Unhook the subject and give the post-session questionnaire.
 24. Obtain the subject's score by:
 - a. pressing any key to continue.
 - b. press "Y" to write to disk.
 - c. press "0" for ID number.
 - d. select "3" for subject's score and write down the subject's score (number correct) on the data sheet in the upper right hand corner.
 - e. press any key to continue.
 - f. press "4" to exit.

Cold Pressor Task

1. To conserve time later, turn the chair to face the Stroop computer because a Stroop test will probably follow at some point.
2. Prepare the ice bath in the small cooler at 0-3 C. Use the long thermometer next to the BP system.
3. Turn on the main power switch to the camera and also set to VCR. Turn on the power switch on top of the camera. Put in JY and SP videotape and make sure that it is rewound. Slide open the door covering the control buttons on top of the camera and press record and play simultaneously (leave the little door open). Adjust the camera so that the easy chair

- and the ice bath are seen clearly.
4. When the subject arrives determine which is his dominant hand. Place the ice bath on the extra chair in the room and move them to the side of the chair which is his dominant side. Place a towel on the arm rest of this side also.
 5. Have the subject sit in the easy chair. Ask him the questions at the top of the data sheet.
 6. Put the BP cuff on his nondominant arm. Set BP system for 1 minute intervals and 150 mm Hg pump up pressure. Flip switch to HOLD.
 7. Tell him, "you will be hearing a tape recorded message with instructions you are to follow."
 8. Leave the room and immediately start timing.
 9. Return to the observation room. Begin the CP 1 tape. Stop the tape when you hear the click which designates the next recording (at approximately 34.5 on the counter).
 10. After 4 minutes return to the subject and start the BP system at minute 5 by turning the volume knob on and flipping the switch to test and then cycle. If the cuff does not start right away flip the switch again to test and then cycle.
 11. Return to the observation room and push ENTER on the computer keyboard.
 12. Record the 11th through the 15th baseline measurements (5 recordings). After the 15th measurement has come up on the screen, restart the tape.
 13. Record the one measurement during the 1 minute task.
 14. After the task is concluded, stop the tape and record 5 minutes of recovery data.
 15. Make print out of data.
 16. Unhook the subject and give him the post-session questionnaire.

APPENDIX E
STATISTICAL DESIGN

Statistical Design

Subject Pool (trained cyclists)	CP	Sequence 1	C-E
		Sequence 2	E-C
	SCW	Sequence 1	C-E
		Sequence 2	E-C
	Whole-plot		Sub-plots

DIAGRAM DESIGN

	Baseline	Peak	Recovery	
Ex				SCW
Ctr				
Ex				CP
Ctr				

APPENDIX F
STATISTICAL ANALYSIS

Table 1. Table of Subject Characteristics

	<u>Cold Pressor</u>	<u>Stroop Color Word</u>
Weight (kg)	71.9 (6.0)	70.7 (5.0)
VO2 Max (ml/kg/min)	57.9 (8.8)	60.7 (6.9)
Max HR (bpm)	214.8 (15.0)	214.1 (15.6)
Max Workload (W)	260.0 (45.0)	235.0 (43.0)
<u>Acute Exercise Data:</u>		
VO2 (ml/kg/min)	41.0 (3.2)	43.4 (3.8)
HR (bpm)	150.3 (11.0)	153.3 (12.0)
Workload (W)	181.5 (27.0)	170.7 (28.2)
Values are M(SEM)	N=11	N=13

Table 2. Hypothesis One-Main Effect of Treatment on CVR
N=24

		<u>(exercise)</u>		<u>(control)</u>		<u>F</u> (1, 22)	<u>p</u>
		<u>x</u>	<u>SEM</u>	<u>x</u>	<u>SEM</u>		
SBP	Baseline	110.22	(1.27)	114.01	(1.01)	4.86	.0383
	Peak	113.97	(1.44)	118.61	(1.23)	.01	.9370
	Change 1	9.98	(1.37)	10.74	(1.74)	.15	.2052
	Change 2	2.10	(1.34)	6.10	(1.09)	.01	.9370
	Recovery	113.97	(1.44)	118.61	(1.35)	5.18	.0329
DBP	Baseline	66.62	(1.14)	68.63	(1.12)	1.65	.2121
	Peak	77.00	(2.25)	81.38	(2.05)	1.87	.2329
	Change 1	9.65	(2.16)	12.70	(1.92)	1.02	.3254
	Change 2	6.20	(2.40)	11.31	(2.10)	1.87	.1862
	Recovery	69.34	(2.04)	70.30	(2.01)	.11	.7420
HR	Baseline	65.94	(1.23)	57.95	(1.80)	20.78	.0002
	Peak	64.75	(2.18)	57.92	(2.45)	22.35	.0001
	Change 1	10.70	(0.93)	10.00	(0.98)	16.30	.0006
	Change 2	14.21	(1.98)	8.12	(1.99)	22.35	.0001
	Recovery	62.77	(2.18)	56.91	(2.08)	5.23	.0322

Values are: SBP and DBP=mm Hg (* p<.05)
 HR=bpm

Baseline=avg of 5 minutes pre-task

Change 1=peak-baseline reactivity

Change 2=peak-screening reactivity

Recovery=avg of first 2 minutes post-task

Summary Results for CVR

SBP

Hypothesis One--Main Effect of Treatment

Baseline	(p<.05)	F(1,22)=4.86
Peak		NS
Change 1		NS
Change 2		NS
Recovery	(p<.05)	F(1,22)=5.18

Hypothesis Two--Interaction of Treatment by Stressor

Baseline	(trend p=.09)	
Peak		NS
Change 1		NS
Change 2		NS
Recovery		NS

DBP

Hypothesis One--Main Effect of Treatment

Baseline		NS
Peak		NS
Change 1		NS
Change 2		NS
Recovery		NS

Hypothesis Two--Interaction of Treatment by Stressor

Baseline		NS
Peak		NS
Change 1		NS
Change 2		NS
Recovery		NS

HR

Hypothesis One--Main Effect of Treatment

Baseline	(p<.05)	F(1,22)=20.78
Peak	(p<.05)	F(1,22)=22.35
Change 1	(p<.05)	F(1,22)=16.30
Change 2	(p<.05)	F(1,22)=22.35
Recovery	(p<.05)	F(1,22)= 5.23

Hypothesis Two--Interaction of Treatment by Stressor

Baseline		NS
Peak		NS
Change 1	(p<.05)	F(1,22)=22.35
Change 2		NS
Recovery		NS

**Change 1=peak-baseline reactivity
Change 2=peak-screening reactivity

Table 3. Hypothesis Two: Main Effect of Treatment by Stressor
N=24

		<u>F</u> (1,22)	<u>p</u>
SBP	Baseline	3.15	**
	Peak	1.32	
	Change 1	.99	
	Change 2	1.30	
	Recovery	.45	
DBP	Baseline	.69	
	Peak	.01	
	Change 1	.34	
	Change 2	.01	
	Recovery	0.00	
HR	Baseline	0.00	
	Peak	2.61	
	Change 1	7.87*	
	Change 2	2.58	
	Recovery	.09	

(p* <.05)

(** p<.05)

Table of Rate Pressure Product (RPP) Values during task
for baseline, control and exercise sessions (N=24)

<u>Subject</u>	<u>Baseline</u>	<u>Control</u>	<u>Exercise</u>
1	10810	80203	10244
2	8710	7911	11221
3	5368	5060	5029
4	8432	7015	8307
5	9720	8378	9810
6	9472	8190	7605
7	12093	14400	14754
8	8932	8085	6649
9	11928	8742	11398
10	8946	8060	8352
11	6438	5610	7068
12	14523	12150	11375
13	9594	10336	9744
14	11739	9600	11484
15	16000	10880	12814
16	10586	9017	10160
17	6039	7080	6820
18	10208	10648	9936
19	11250	10877	10790
20	5207	6012	6048
21	6372	6273	5664
22	9525	7598	7638
23	10564	9676	7412
24	<u>8846</u>	<u>7744</u>	<u>7672</u>
	=231,302	=279,708	=217,994
	x=9,637.58	x=11,647.71	x=9,083.08

Summary ANOVA Table for Treatment Effect across Stressors (N=20)

		<u>F</u> (1, 18)	<u>p</u>
SBP	Baseline	2.43	.1384
	Peak	.02	.8964
	Change	1.28	.2749
	Recovery	3.64	.0746
DBP	Baseline	.55	.4964
	Peak	1.41	.2517
	Change	.62	.4410
	Recovery	.65	.0433
HR	Baseline	12.51	.0027
	Peak	14.77	.0014
	Change	.15	.7509
	Recovery	10.41	.0053

Summary ANOVA Table for Interaction effect across groups (N=20)

		<u>F</u> (1, 18)	<u>p</u>
SBP	Baseline	.56	.4640
	Peak	1.92	.1854
	Change	.09	.7658
	Recovery	.88	.3608
DBP	Baseline	.04	.8526
	Peak	1.67	.2141
	Change	1.22	.2558
	Recovery	.19	.6686
HR	Baseline	2.31	.1478
	Peak	11.68	.0035
	Change	17.48	.0007
	Recovery	4.82	.0433

TABLE OF RELIABILITY ESTIMATES ON
CVR MEASURES
(Pearson Product Moment Correlation)

(Screening-control session):

	BASELINE	PEAK
	<u>r</u>	<u>r</u>
SBP	.67	.68
DBP	.56	-.15
HR	.79	.50

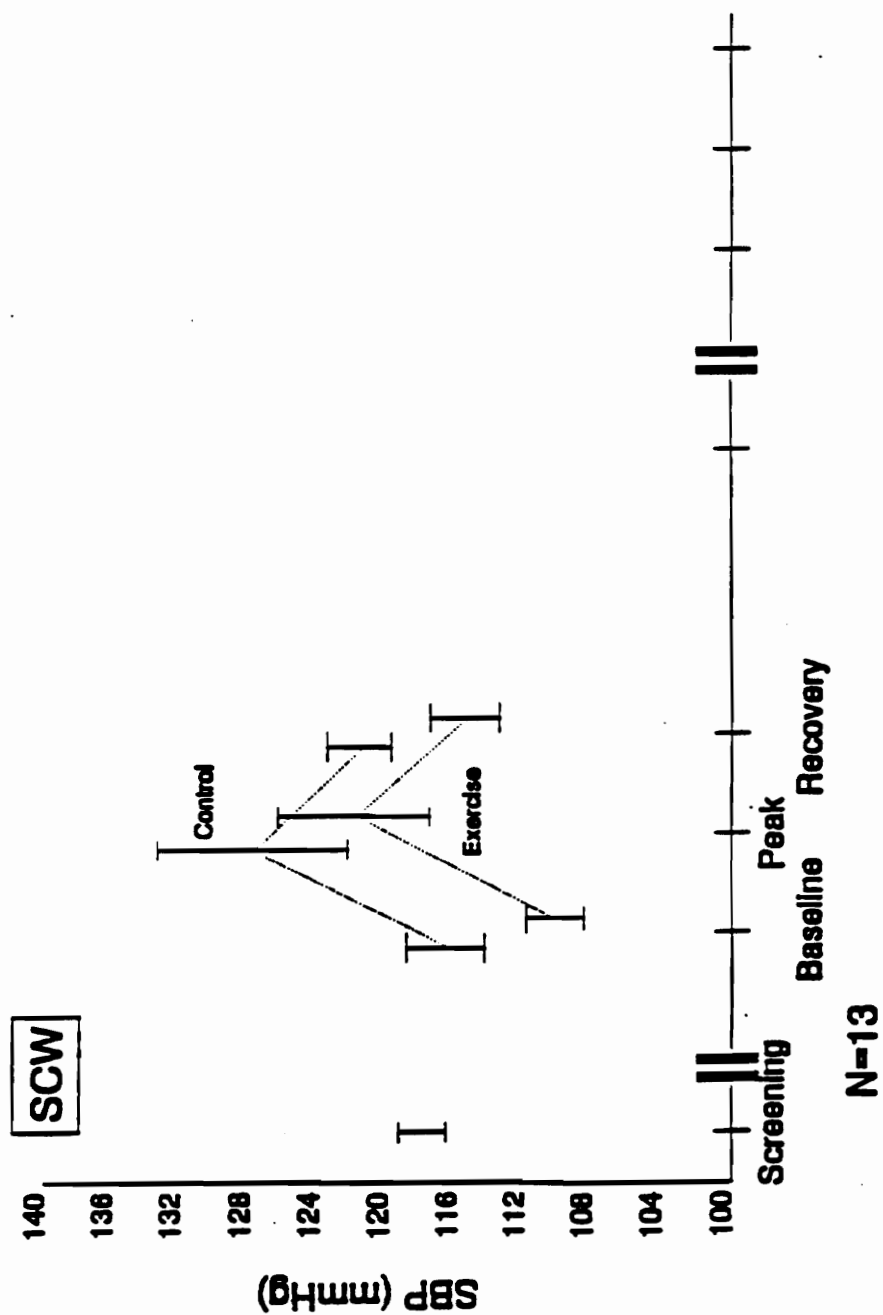


Figure 1. Systolic Blood Pressure Reactivity Measures

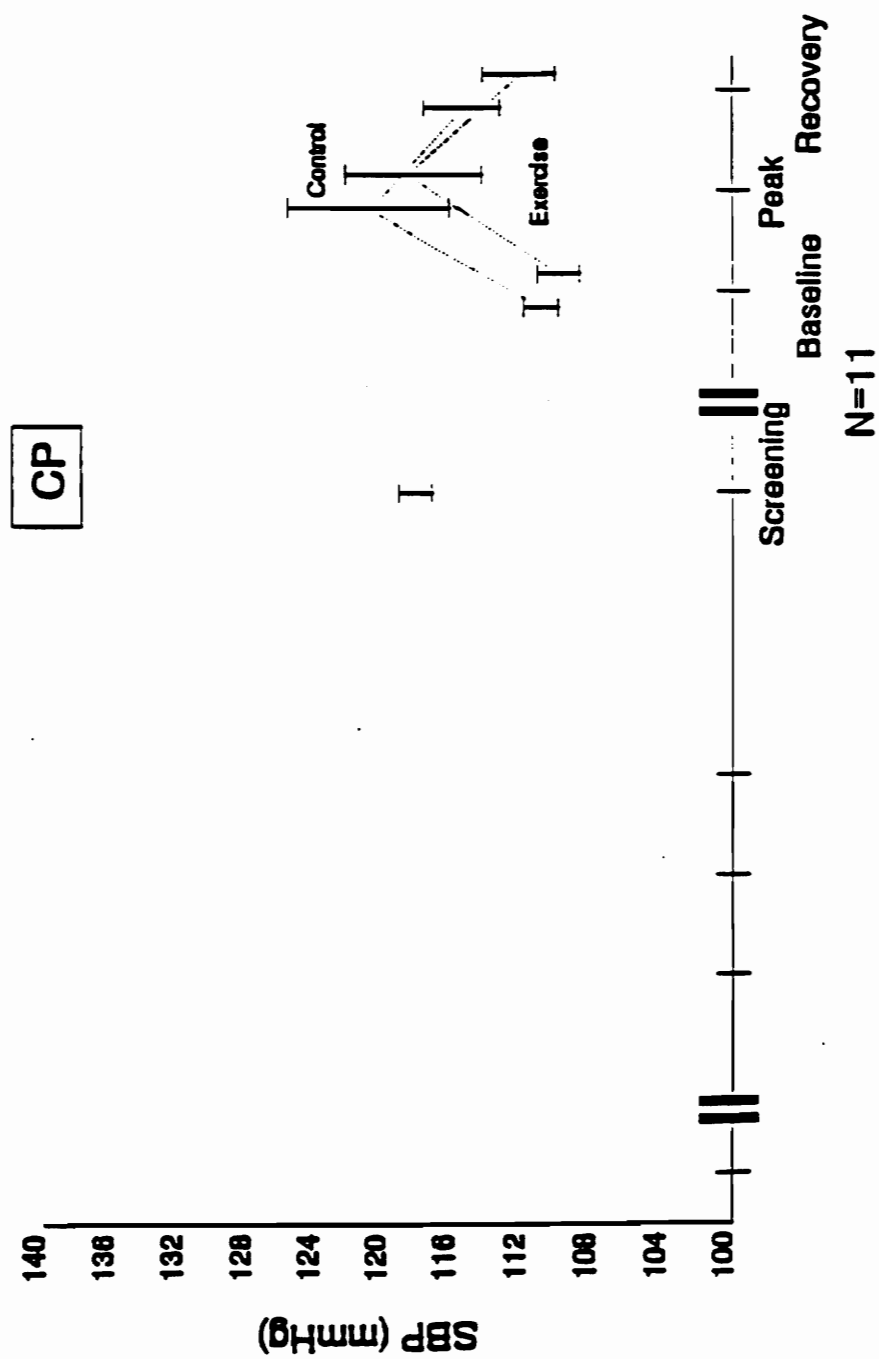


Figure 1. Systolic Blood Pressure Reactivity Measures

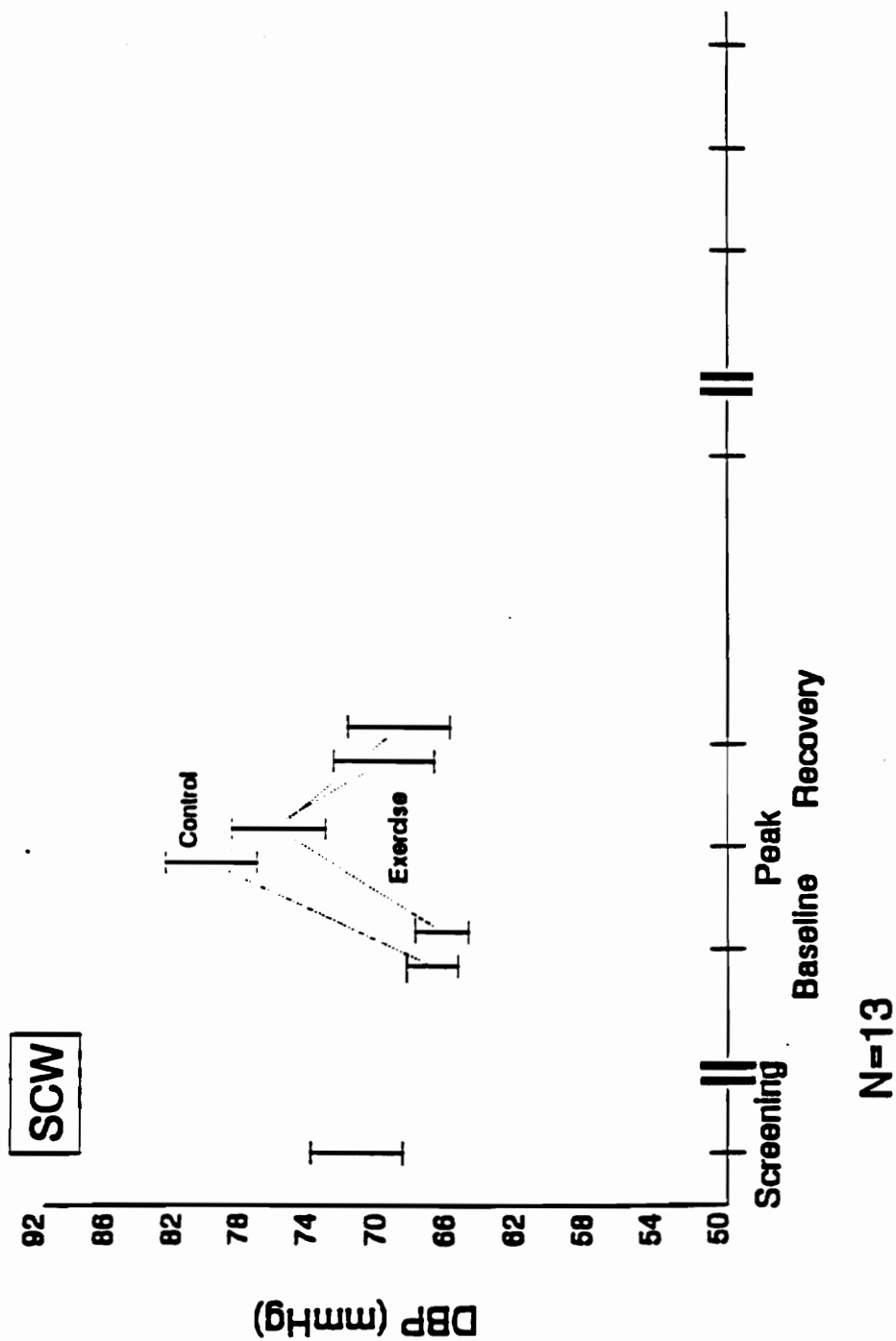


Figure 2. Diastolic Blood Pressure Reactivity Measures

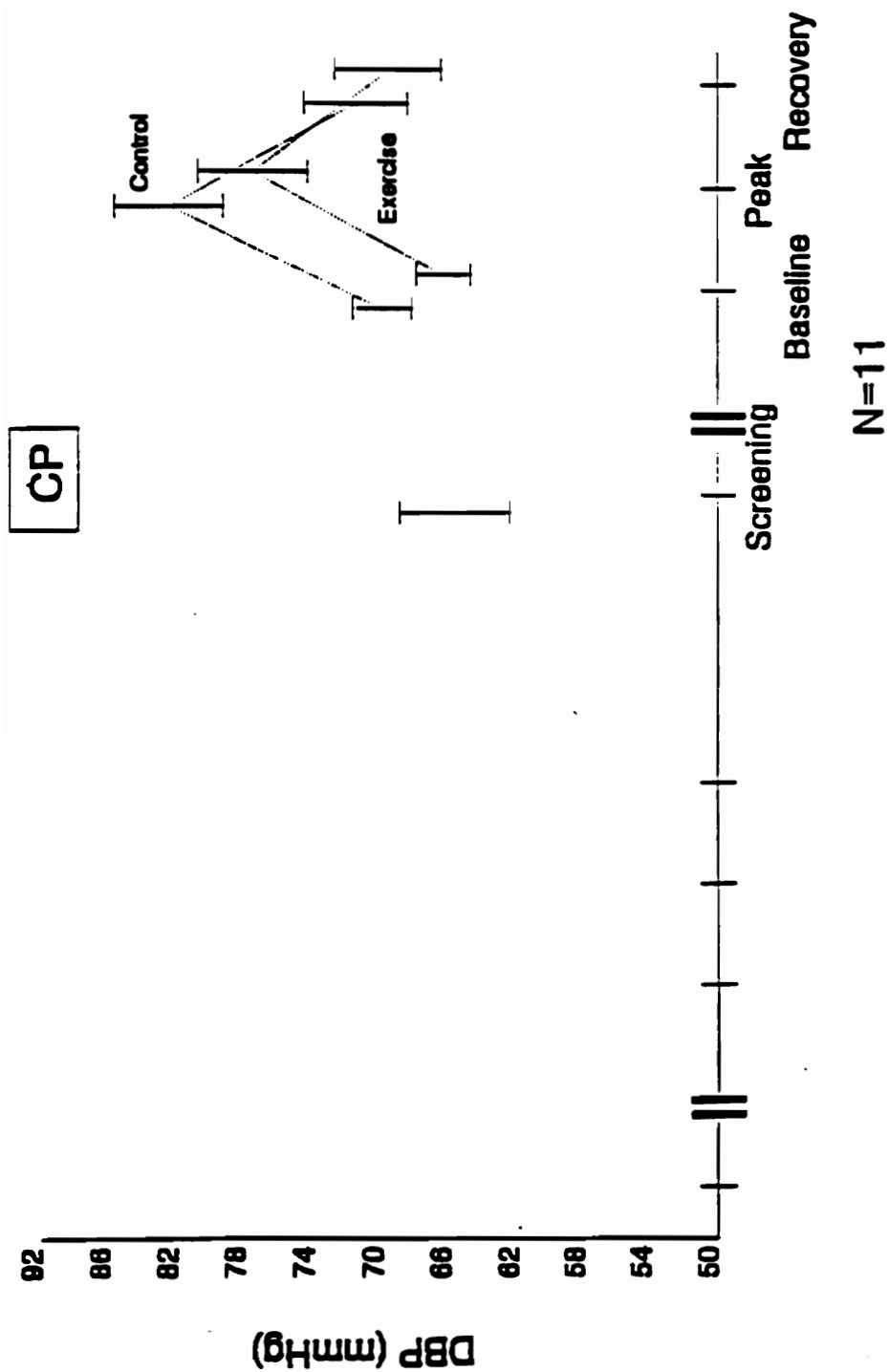


Figure 2. Diastolic Blood Pressure Reactivity Measures

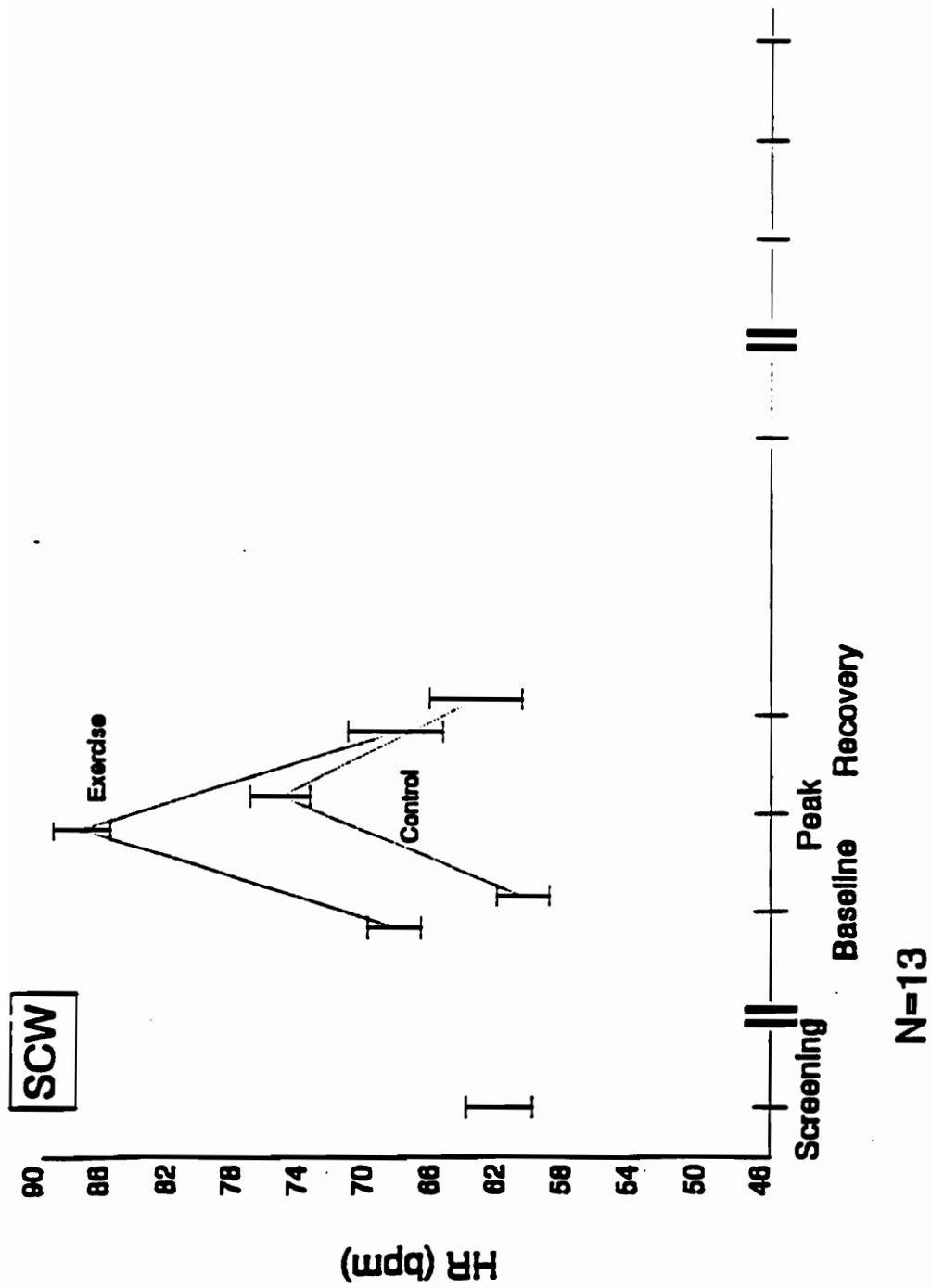


Figure 3. Heart Rate Reactivity Measures

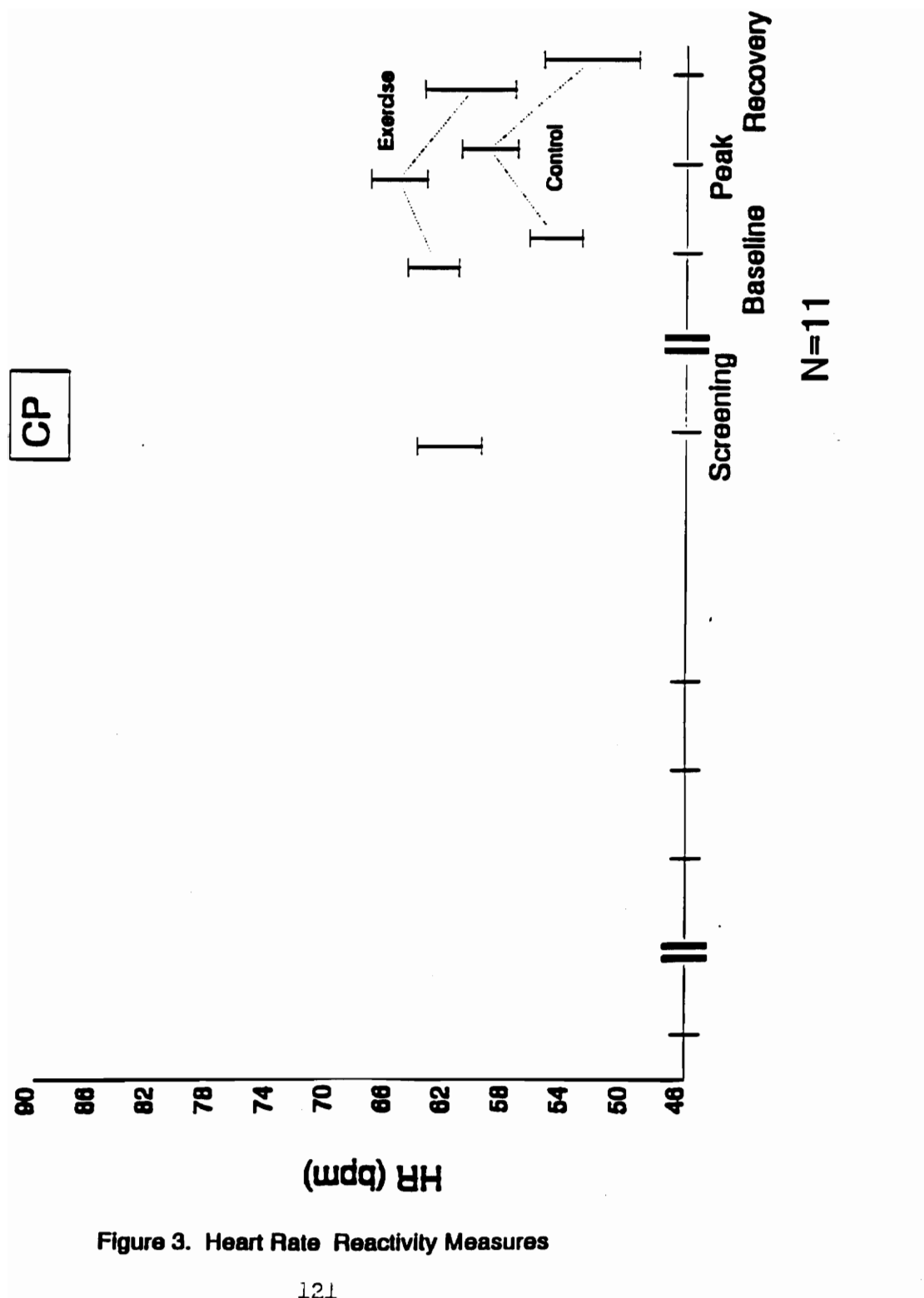
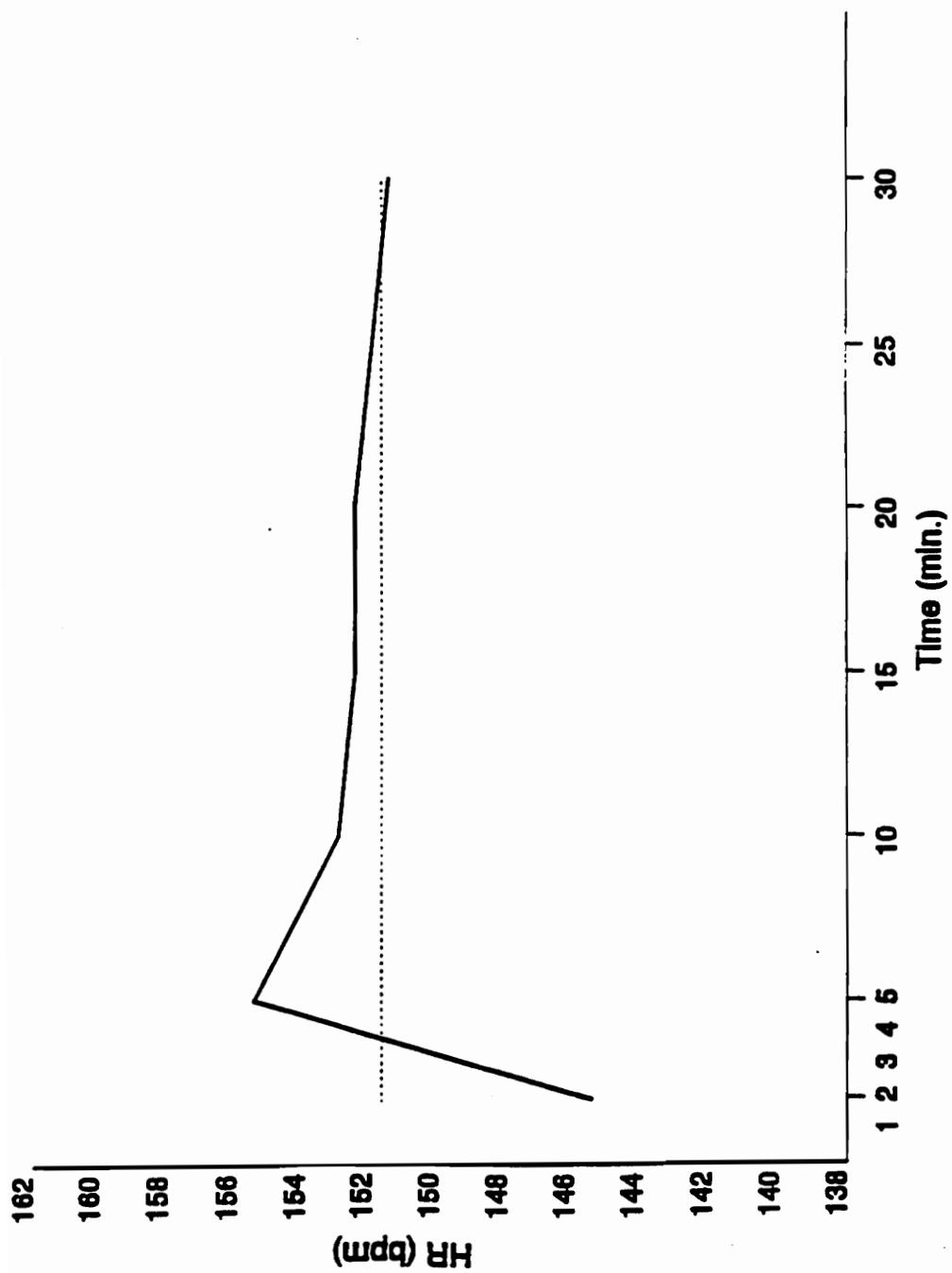


Figure 3. Heart Rate Reactivity Measures



Note: Average THR for 70% VO_2 max = 151.7

APPENDIX G

RAW DATA

Acute Exercise
Data

Table of Subject Characteristic and Raw Data

Subject	Age	VO ₂ (ml/Kg/min)	f BF	BHR	RRP	Watts	HR
1	22	52.01	7.0	54	112/60	125	145
2	22	64.44	10.7	46	116/48	175	140
3	22	42.09	11.6	78	102/66	175	153
4	22	70.69	8.0	60	112/78	175	145
5	25	70.09	7.6	66	92/60	200	146
6	22	48.84	12.5	72	120/74	200	162
7	25	55.9	11.5	72	110/84	200	174
8	22	58.02	8.9	76	128/78	175	155
9	22	61.87	7.0	52	114/72	225	136
10	21	52.35	7.0	54	108/62	165	148
11	21	60.6	6.1	44	104/80	200	150
12	22	72.22	12.5	72	140/64	200	158
13	21	58.20	10.7	84	108/68	135	150
14	22	64.43	4.2	72	118/76	200	150
15	19	63.36	12.3	76	118/84	125	168
16	21	60.18	6.1	72	160/80	175	160
17	23	62.22	9.4	72	102/68	180	166
18	20	55.18	7.0	66	114/72	150	140
19	26	46.23	10.4	88	120/72	140	150
20	21	59.49	7.0	72	102/58	150	150
21	20	71.90	11	74	120/78	150	130
22	21	61.92	7.0	56	114/82	125	156
23	23	55.32	10.4	66	116/61	180	143
24	19	57.67	6.1	60	126/70	225	170

* Note: Subjects 1-11 = CP Task
Subjects 12-24 = SCW Task

Table of Peak CVR

Subject	I			II			III		
	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR
1	135	72	80	126	80	81	134	74	60
2	122	91	44	107	91	47	115	98	44
3	117	83	75	124	72	80	120	76	66
4	142	88	62	127	79	66	130	81	62
5	111	78	58	114	81	62	102	69	55
6	134	80	78	115	69	77	123	74	65
7	125	79	80	109	82	68	118	88	82
8	127	80	75	114	71	67	131	92	58
9	118	82	54	118	92	48	123	91	51
10	127	68	41	126	79	48	126	74	49
11	120	93	59	110	60	62	104	76	37
12	141	86	80	130	72	83	147	66	73
13	116	80	88	108	72	92	121	78	88
14	160	79	100	149	80	86	128	85	85
15	129	72	91	116	67	99	128	59	75
16	123	73	78	112	74	87	136	72	76
17	125	78	68	117	80	71	115	79	61
18	131	78	108	122	87	111	117	85	92
19	120	78	81	109	82	90	118	73	71
20	128	71	74	117	68	65	130	70	63
21	140	61	87	133	61	112	144	120	100
22	116	70	77	105	78	77	109	81	61
23	142	58	84	139	71	82	141	80	62
24	134	65	79	126	73	80	127	87	71

Values are SBP & DBP = mm Hg; HR = bpm;
 I = Screening Session; II = Exercise Session; III = Control Session.

Table of Baseline CVR

Subject	I			II			III		
	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR
1	128	58	64	119	56	73	132	67	55
2	113	76	47	103	71	52	93	67	42
3	121	67	73	120	69	82	110	62	62
4	123	67	63	113	66	61	99	61	55
5	108	69	58	108	74	63	104	69	51
6	134	80	78	115	69	77	123	74	65
7	108	79	75	105	76	70	111	83	79
8	125	77	68	112	67	62	120	79	60
9	117	75	60	104	63	51	118	68	52
10	118	63	45	112	61	46	126	59	43
11	116	95	54	101	60	58	104	75	40
12	132	62	66	114	60	63	130	62	57
13	132	72	71	101	65	77	106	70	75
14	140	70	61	127	82	57	134	71	61
15	124	67	84	107	68	75	117	60	63
16	123	69	69	115	66	62	128	69	64
17	110	71	60	111	76	66	113	73	57
18	110	64	79	106	73	92	105	77	74
19	113	72	65	110	82	78	115	63	61
20	111	63	56	98	54	58	102	64	59
21	107	69	70	93	67	72	107	74	58
22	107	69	70	93	67	72	107	74	58
23	121	57	59	123	60	63	127	63	44
24	120	63	68	111	76	66	113	73	57

Values are SBP & DBP = mm Hg; HR = bpm;
 I = Screening Session; II = Exercise Session; III = Control Session.

VITA

Virginia Sue Palmer ("Susi") was born in 1964 in Berea, Ohio, where she grew up in the Cleveland/Akron area. She graduated from Stow High School in Stow, Ohio and received her Bachelor of Science degree from Bowling Green State University in Ohio. Susi majored in Sports Management (Fitness Specialist). Her graduate work was done at Virginia Tech in 1989 where she received her Master of Science degree in Exercise Physiology in September, 1991.

Susi participates in running, swimming and most recreational activities. She also enjoys time with her family, her two cats, and Collie, Prince. She is looking forward to beginning her life with Andrew as well as a career in cardiac rehabilitation.