1991

A Retrospective study on the effects of a 9-month exercise program on selected physiological indices of hypertensive and non-hypertensive coronary artery disease patients

Mahmound R. Sadri
San Jose State University

Order Number 1347179

A retrospective study on the effects of a 9-month exercise program on selected physiological indices of hypertensive and non-hypertensive coronary artery disease patients

Sadri, Mahmoud Reza, M.A.

San Jose State University, 1991

U·M·I
300 N. Zeeb Rd.
Ann Arbor, MI 48106

A RETROSPECTIVE STUDY ON THE EFFECTS OF A 9-MONTH EXERCISE PROGRAM ON SELECTED PHYSIOLOGICAL INDICES OF HYPERTENSIVE AND NON-HYPERTENSIVE CORONARY ARTERY DISEASE PATIENTS

A Thesis Project

Presented to

The Faculty of the Department of Human Performance

San Jose State University

in Partial Fulfillment

of the Requirements for the Degree

Master of Arts

By

Mahmoud R. Sadri

August, 1991

APPROVED FOR THE DEPARTMENT OF HUMAN PERFORMANCE

Carol L. Christensen

Dr. Carol L. Christensen

Greg Payne

Dr. Greg Payne

Edith Lindquist

Dr. Edith Lindquist

APPROVED FOR THE UNIVERSITY

Serena H. Stanford

ABSTRACT

THE EFFECTS OF A 9-MONTH EXERCISE PROGRAM
ON CORONARY ARTERY DISEASE PATIENTS

by M. R. Sadri

The purpose of this retrospective study was to determine if nine months of aerobic exercise performed three days a week for 35 minutes per session, at intensities of 60-85% of symptom-limited maximum heart rate, could significantly reduce the weight, resting and submaximum exercise heart rates, resting and submaximum exercise mean blood pressures, and submaximum exercise rate-pressure product. The subjects were 100 (50 experimentals, 50 controls) adult white males with coronary artery disease (CAD), with and without hypertension.

Results of t-tests for independent groups revealed that participation of CAD subjects within a structured exercise training program brought about statistically significant reductions (improvements) on all the aforementioned dependent variables. The results of two two-way ANOVA indicated that being hypertensive or not had no influence on the results obtained. The most noteworthy result was the improvement in the work tolerance index (reduction of submaximum exercise rate-pressure product).

ACKNOWLEDGEMENTS

The ability to complete an acceptable Master's thesis is not only a result of years of university studies, but the result of the culmination of a lifetime of experiences, academic and otherwise. Space does not allow acknowledgements of all the fine individual teachers with whom I studied. Suffice it to say, the education system has been good to me. However, it is my honor and obligation to name a few good people without whom this thesis would never have been written.

Dr. Carol L. Christensen is truly a graduate student's best ally. While she insists on high standards of scholarship, she is always kind and encouraging. She is the best teacher I've ever had.

Dr. Gregory Payne, who helped me with all the statistical work, allowed me a great deal of freedom to find my own level of competence without letting me become overwhelmed or self-doubting. His constructive criticism of my initial statistical approach made me become more analytical and certainly a stronger student.

Dr. Edith Lindquist's probing questions and insightful critiques helped keep this thesis well defined and on course. I was fortunate to have her on my committee.

My heart is filled with gratitude towards my entire family, who never gave up on me. My father and my mother, who worked hard for many years to give their children an education, continue to set a perfect example of constant pursuit of knowledge and self-improvement.

It is impossible to measure the emotional and financial contributions of my older brother, Ahmad, to this effort and to my education as a whole. He is the greatest man I have ever personally known. I wish to thank my two sisters, Minoo and Nikoo, and my younger brother, Hossein. Their all-around support and love kept me going throughout the years. I wish to acknowledge the five apples of my eyes: my daughter, Niloufar, and my

brother's children, Amir, Kristina, Ashly and Alexi. The love I have for them motivated me to complete this project.

Special thanks to my sister-in-law, Laurie, for introducing me to the wonderful world of word processors, and to Debbie's Secretarial Services for her advice and help, enabling me to produce a professional product.

Special warm thanks to my ex-wife, Clorissa, whose support and encouragement through the better part of my college education, I will never forget.

I especially want to dedicate this thesis to my four grandparents, Mr. and Mrs. Sadri and Mr. and Mrs. Mirfakhrai.

Again, without the love, help and support of all my teachers, friends and relatives, this project would not have been possible and I am indebted to you all.

TABLE OF CONTENTS

TABLE OF CONTENTS

Chapter III (continued)

Chapter IV

Chapter V

Appendices

LIST OF TABLES

LIST OF TABLES

LIST OF TABLES

Chapter I

Introduction

More people in the United States suffer from cardiovascular diseases than any other chronic ailment (Tortora & Anagnostakos, 1981). Two forms of cardiovascular disease, coronary artery disease (CAD) and hypertension, are the most significant contributors to human heart attack (Tortora & Anagnostakos).

During the past 20 years, exercise training programs have been used in the management and treatment of cardiovascular diseases. Several studies have demonstrated that properly executed training programs, used in conjunction with proper medication, improve the exercise tolerance in the majority of CAD patients, allowing them to do more physical work with less discomfort and pain, and thus enhancing their quality of life (Clausen & Trap-Jensen, 1970; Stuart, Sanker & Lundstorn, 1985; Vanhees, Fagard, & Amery, 1982).

To understand why and how exercise tolerance or work capacity can be altered in patients with cardiovascular disease, a measure of the understanding of the nature of the disease is needed. The physiological components that enable the organism to exercise more, the system of exercise, and the variables which influence the physiological components to adjust to the stress of physical work, need to be understood.

The physiological components that are most clearly involved are the central circulatory components, mainly the heart, and the

peripheral circulatory components, mainly the skeletal muscle tissue (Clausen, 1976). It is through the collaboration between these two separate, but integrated components, that the organs receive and utilize adequate oxygenated blood to fuel muscular activities for physical movements of all kinds (Tortora & Anagnostakos, 1981). In fact, the equation below demonstrates how these two components exert their influences in the basic fundamental equation of human exercise physiology (Froelicher, 1983).

Endurance Fitness = heart's pumping action x muscle tissue's ability to extract oxygen from the circulating blood

Improvements or changes in exercise capacity are possible by changes in the heart, the skeletal muscle tissues, or both of these (Froelicher, 1983). These changes can be effected by a program of regular exercise.

Any system of exercise has four basic components: intensity, frequency, duration, and the mode of exercise or activity. By direct manipulation of one or more of these components a specific training effect can be achieved (Davis & Convertino, 1975; Fox, et al., 1975; Pollock, Dimmick, Miller, Kendrick & Linnerud, 1975).

Although athletes may regularly perform exercises of an intensity and duration requiring near maximum oxygen uptake, the beneficial effects non-athletes and cardiac patients obtain from physical training are obtained at a lower circulatory intensity during submaximum exercise. Hence, all the discussions in this study are based on results

that have been obtained from CAD subjects during submaximum exercise performance.

Statement of the Problem

Exercise is used widely in the rehabilitation of patients with CAD, and often results in major improvements in work capacity (Clausen, 1976). The purpose of this study was to determine the effects of a 9-month walk-jog exercise program performed three days a week for 35 minutes per day on changes in submaximum physical working capacity in adult male cardiac patients between the ages of 45 and 60 years. The training program was designed to have an intensity corresponding to 60-75% of each subject's symptom-limited maximum heart rate during the first six weeks, and 85% of their symptom-limited maximum heart rate for the remainder of the 9-month exercise program.

Research Hypothesis

A walk-jog exercise program performed three days a week for nine months with progressive increases in intensity (60-75% to 85% of symptom-limited maximum heart rate) and duration of 35 minutes will produce significant physiological improvements in the following indices:

1. Resting heart rate
2. Resting blood pressure
3. Submaximum heart rate
4. Submaximum blood pressure
5. Submaximum rate-pressure product

6. Body weight

A second research hypothesis is that there will be no differences between hypertensives and non-hypertensives when pre-test and post-test scores are compared on all six dependent variables.

Limitations

The factors in this study which could not be controlled included:

1. The lack of strict adherence to the prescribed training intensities.

2. The pharmaceutical drugs taken by the patients on a daily basis.

3. Patient diet.

4. The retrospectiveness of the study – all tests were performed by other people and were beyond the control of this author.

Delimitations

This study was limited to 100 adult white male CAD patients, half of whom were also hypertensives, between the ages of 45 and 60 years. All subjects were three to six months post-MI. Fifty of these subjects belonged to the experimental group (25 hypertensives and 25 normotensives) and 50 belonged to the control group (25 hypertensives and 25 normotensives). The experimental subgroups included only members of the San Jose Central YMCA Cardiac Therapy Rehabilitation Program between the years 1978 and 1985. The control subgroups were patients in a private medical firm in San Jose. Their medical files pertaining to pre-test and post-test data and diagnosis of

their diseases (CAD and hypertension) came from the years between 1978 and 1985.

Definition of Terms

For this study, the following terms are defined.

Arteriosclerosis is a group of diseases characterized by thickening and loss of elasticity of artery walls. This may be due to an accumulation of fibrous tissue, fatty substances, and/or minerals (Wittie, 1980).

Angina Pectoris is chest pain caused by the ischemia of the myocardium (Tortora & Anagnostakos, 1981).

Arterio-venous oxygen difference is the difference between the oxygen content of arterial and mixed venous blood. It represents the amount of oxygen that is extracted or consumed by the tissues from each milliliter of blood perfused (Froelicher, 1983).

Coronary Artery Disease (CAD) is a disease of the blood vessels feeding the heart muscle. It is synonymous with the terms coronary heart disease and ischemic heart disease (Froelicher, 1983).

Endothelial (endothelium) is the thin layer of cells lining the heart, the blood vessels, lymph vessels, and forms the walls of microscopic blood vessels called the capillaries (Tortora & Anagnostakos, 1981).

Hypertension is a persistent elevation of blood pressure above the normal range (Tortora & Anagnostakos, 1981). Diastolic pressures of greater than 90 mmHg, and systolic pressures greater than 160 mmHg constitute a hypertensive state (report of the Third Joint National Committee, 1984).

Ischemia (Ischemic) is a local, usually temporary, deficiency of oxygen in some part of the body, often caused by a constriction in the blood vessels supplying that part (Wittie, 1980).

Myocardium is the muscular wall of the heart. The thickest of the three layers of the heart wall, it lies between the inner layer (endocardium) and the outer layer (epicardium) of cells (Wittie, 1980).

Myocardial Infarction is the death of an area of heart muscle tissue because of an interrupted blood supply (Tortora & Anagnostakos, 1981).

Rate-Pressure Product is systolic blood pressure reading times heart rate reading (Tortora & Anagnostakos, 1981).

Stroke Volume is the amount of blood pumped by the left ventricle per contraction or beat (McArdale, 1986).

Submaximum Exercise is an exercise that can be performed in an aerobic steady state, where physiological functions such as oxygen consumption or heart rate remain at constant values (Fox & Mathews, 1981).

Chapter II

Review of Literature

This chapter contains reviews of four separate but interrelated topics:

1. The nature and relationship between coronary artery disease and hypertension.

2. The physiological parameters responsible for adaptive changes.

3. The exercise systems needed to develop changes.

4. Review of literature pertaining to training programs similar to that used in this study.

Nature of Coronary Artery Disease and Hypertension

Coronary artery disease involves degenerative changes in the intima (inner lining) of the larger arteries that supply the heart muscle (McArdale, 1986). These vessels become congested with either lipid-filled plaques or fibrous scar tissue or both. This change progressively reduces the capacity for blood flow and causes the myocardium to become ischemic. The progressive occlusion of an artery with a buildup of calcified fatty substances is the process of atherosclerosis. Different categories of atherosclerosis are determined by the number of the occlusions (e.g., one, two, or three vessel blockage), and the site of its manifestation within the coronary network feeding the heart muscle (e.g., transmural, subendocardial) (McArdale, 1986).

In this degenerative process, the roughened, hardened lining of the coronary artery causes the slowly flowing blood to clot. This blood clot (thrombus) may plug one of the smaller coronary vessels. In such cases, a portion of the heart muscle dies and the person is said to have suffered a heart attack or myocardial infarction (MI). If the blockage is not too severe, but blood flow is reduced below the heart's requirement, the person may experience temporary chest pains termed angina pectoris. These pains are felt usually during exertion, since this causes the greatest demand for myocardial blood flow. Such anginal attacks provide painful evidence of the importance of adequate oxygen supply to this vital organ (McArdale, 1986).

Almost all people show evidence of CAD, and it may be severe in seemingly healthy young adults. Actually, the disease probably starts early in life, since fatty streaks are common in the coronary artery of children by the age of five (McArdale, 1986). There seems to be little harm however, unless a marked narrowing of the arteries is present (McArdale).

The mechanism by which fatty-type deposits or plaques develop is not entirely clear (McArdale, 1986). One thing is clearly demonstrated in epidemiological studies; environmental factors such as diet, cigarette smoking, and hypertension are significant contributors to the development of CAD (Froelicher, 1983).

Hypertension

Hypertension is the medical term for blood pressure chronically elevated above normal healthy values. The 1984 report of the Third Joint National Committee on The Detection, Evaluation, and Treatment of Hypertension (Report of the Third Joint National Committee, 1984) provides the following definitions for diastolic and systolic blood pressures (DBP and SBP, respectively):

DBP:

less than 85 mmHg = normal

85-89 mmHg = high normal

90-104 mmHg = mild hypertension

105-114 mmHg= moderate hypertension

115 mmHg or greater = severe hypertension

SBP:

less than 140 mmHg = normal

140-159 mmHg = borderline hypertension

160 mmHg or greater = severe hypertension

It is uncommon for the diastolic pressure to be elevated as an isolated abnormality unaccompanied by systolic hypertension. Isolated systolic hypertension, however, may occur with normal diastolic blood pressure (report of the Third Joint National Committee, 1984).

There are two kinds of hypertension. Primary, or essential, hypertension is a persistent elevated blood pressure that can not be attributed to any particular organic cause (Kannel, 1990). Approximately 85% of all hypertension cases fit this definition. The

other 15% of cases are secondary hypertension which are caused by medical problems such as arteriosclerosis, kidney abnormalities, and hormonal disturbances (Kannel, 1990).

Relationship between CAD and Hypertension

There is ample evidence that hypertension accelerates the development of atherosclerosis (Kannel, 1990). Atherosclerotic cardiovascular consequences including stroke, coronary artery disease, and peripheral arterial disease; all occur with a two to three fold increased frequency in hypertensives as compared to normotensives of the same age (Kannel). There is evidence that blood pressure is critical to the atherosclerotic process, because lesions seldom occur in low-pressure parts of the circulation such as the pulmonary arteries or veins, despite their exposure to atherogenic blood lipids (Kannel). Animal studies demonstrate that lipid-induced atherogenesis can be accelerated or retarded by manipulating the blood pressure (Kannel).

Although hypertension may accelerate the development of atherosclerosis, it is not expected to be a strong initiating factor. Both epidemiological (Jablon, 1966) and experimental studies (Pick, Johnson, & Glick, 1974) suggest that hypertension accelerates atherogenesis only if hyperlipidemia is present. Hypertension promotes the process by means of increased endothelial permeability and accelerated low density lipoprotein (C-LDL) entry into the artery wall, both by chronic and repeated stretch (tension) on the artery wall (Kannel).

Physiological Parameters Responsible for Adaptive Changes

The primary cause of symptoms and pathophysiological changes in CAD is related to the impaired coronary circulation (Sonnenblick, Ross, & Braunwald, 1968). Therefore, the main focus of this section will be on factors and parameters that determine the myocardial oxygen consumption. Research has shown that heart rate, intra-myocardial tension (i.e., cardiac wall tension), and the contractile state of the heart are major factors determining myocardial oxygen consumption (Sonnenblick, et al.). In healthy hearts, oxygen supply, as determined by the amount and distribution of coronary blood flow and by the arterial-venous oxygen difference across the coronary bed, is greater than the oxygen demand of the heart. In ischemic hearts, this relationship is reversed; for a certain workload, the oxygen supply cannot keep up with the oxygen demand, and pain (the main factor for the cessation of exercise performance), electrocardiographic, metabolic abnormalities, and ventricular dysfunction occur. Remediation is based on restoration of a favorable balance between myocardial oxygen demand and supply, a balance that can be achieved by increasing supply or decreasing demand, or both (Patterson, Shephard, & Cunningham, 1979).

Because of the essentially aerobic metabolism of the myocardium, changes in myocardial oxygen consumption (MVO2) correlate highly with work of the heart. Measuring changes in MVO2 allows the study of various medical, surgical, and physical (rehabilitation training program) interventions on the ability of the heart to perform work (May & Naple, 1984). Direct measurement of MVO2 involves invasive

technique and is not feasible for routine clinical evaluation. Indirect techniques that are non-invasive have been used as early as 1912 (May & Naple).

A close correlation between the product of systolic pressure and heart rate, using isolated cat and rabbit heart muscle, has been established (May & Naple, 1984). In one study, using healthy human subjects, Sarnoff (1958) examined the relationship between several readily measured hemodynamic variables and MV02. He found a correlation of .88 between heart rate and MV02. A correlation of .90 between the product of heart rate and arterial blood pressure measurement, or rate-pressure product (RPP), and MV02 was also established. In another study, aortic blood pressure was found to exert the dominant influence in myocardial oxygen use, using an isolated, supported dog-heart preparation (Sarnoff). Many investigators have studied the correlation of myocardial oxygen consumption with various cardiac indices in subjects with CAD (Kitamura, Jorgensen, & Gobel, 1972; May & Naple, 1984; Sarnoff, 1958; Sonnenblick et al., 1968). These cite RPP as having the highest correlation (.90, .92, .96) to myocardial oxygen consumption (Kitamura, et al., 1972; May & Naple, 1984; Sarnoff, 1958; Sonnenblick, et al., 1968).

Investigators have used RPP and other indices to estimate changes in myocardial oxygen consumption in physical training of individuals with CAD (Frick & Katila, 1968; Gobel, 1978; May & Naple, 1984). In a study involving 55 post MI subjects (29 experimental, 26 control), researchers found a significant decrease in RPP at each of

three submaximum work loads in the experimental group (Debacker, 1974). In a study of 11 subjects with angina, researchers found a decrease in time-tension index (ejection period) at submaximum work loads, mainly because of a fall in heart rate (Clausen & Trap-Jensen, 1970). Effort angina pectoris (anginal threshold) was reproducible at fixed levels of the RPP in this study, elucidating the validity and reliability of this index in quantitatively determining the extent of adaptive changes in cardiac exercise physiology. In another study, RPP was found to significantly decrease in 25 cardiac patients after training at submaximum levels (Clausen & Trap-Jensen, 1976).

A consistent and significant decrease in RPP after training at submaximum work loads constitutes the physiological basis for the symptomatic improvement seen in cardiovascular diseased patients at daily-living activity levels (Froelicher, 1984). The imbalance between oxygen demand and blood supply to the heart is lessened by lowering the demand of the same absolute work load after training compared with before training (Froelicher). Physical training can alter the cardiovascular response to exercise so that a decrease in heart rate and pressure load is imposed on the myocardium when performing the same work load as before training. The physiological mechanism that causes the training bradycardia and decreased blood pressure has been associated with a general reduction in sympathetic drive at a given submaximum work load because of increased oxygen (O2) extraction capacity in the trained skeletal muscle (Clausen & Trap-Jensen, 1970). The main reason trained muscles extract more O2 from blood is

mitochondrial and enzymatic adaptations (Hollowzy, 1976). Additional factors that may be changed include myoglobin content (Pattengale & Hollowzy, 1967) and the displacement of the oxygen-hemoglobin dissociation curve to the right (Rassmusen, 1975). The exact mechanism relating local adaptation in skeletal muscle to the decreased heart rate is unclear, but the decreased blood pressure that accompanies the bradycardia points to a muscle afferent feedback hypothesis (Coote & Hilton, 1971). When muscle contraction occurs, afferent signals are sent by reflex action from receptors in the exercising muscles to the sympathetic centers in the brain (Coote & Hilton). Training is believed to somehow alter the metabolic processes and needs of the exercised muscle so that the afferent signals from muscles reaching the sympathetic centers in the brain are similar to resting conditions (Coote & Hilton). In a study of enzymatic physiology in man, endurance training caused a marked augmentation in the number and size of muscle cell mitochondria with a concomitant increase in the concentration and the activity of the enzymes involved in aerobic metabolism (Hollowzy, 1975). It follows that, after training, because of the increased capacity for oxidative phosphorylation, adenosine tri-phosphate and creatine phosphate in exercising muscles stabilize at a higher steady state level. At the same time glycolysis occurs at a slower rate, blood acidity is relatively decreased, and the concentration of multiple intermediate metabolic products may be lower (Hollowzy, 1971). In other words, the metabolic state of the exercising muscle, when

compared with the conditions at rest, is less disturbed in the trained exercising muscle than in the untrained exercising muscle.

Thus, a decrease in RPP at a given submaximum work load after training suggests that the imbalance between oxygen demand and oxygen supply in ischemic hearts is lessened by decreasing the myocardial oxygen demand of a given work load (Gobel, 1978). An increase in maximal RPP, however, suggests that not only does training diminish the imbalance between oxygen demand and supply of the heart by decreasing demand but also it has a direct effect on the myocardium and increases oxygen supply to the heart (Gobel). If maximum heart rate and blood pressure are increased, the heart is capable of developing a greater intraventricular pressure and performing at a greater rate; both these factors determine MVO2. The increase in oxygen supply to the myocardium after training could be due to either an increase in blood supply through collaterals or to an increased myocardial enzymatic capacity so that oxygen uptake is greater, or both (Lassen & Larsen, 1970). One study has shown that blood flow is redistributed within the myocardium after training so that the ischemic tissue receives a greater supply of blood (Lassen & Larsen). The healthy tissue requires less perfusion because of its increased ability to extract oxygen from blood. In an animal study using rats, the heart hypertrophied after training (Lassen & Larsen). Muscle tissue and total enzymatic activity also increased (Lassen & Larsen). In a study of young athletes, the above findings were supported (Ehsani, 1978). Using echocardiographic estimates of left ventricular indices, Ehsani found increased left

ventricular end-diastolic dimensions, and increased left ventricular posterior wall thickness after training. These findings suggest an increase in total myocardial enzymatic activity (Ehsani).

In light of the above discussions, conclusions can be made that, after training, the imbalance between oxygen demand and oxygen supply in the ischemic hearts is lessened by both a decrease in oxygen demand, suggested by the decrease in submaximum RPP, and an increase in oxygen supply, suggested by the increase in maximal RPP.

The Exercise Systems Needed to Develop Adaptive Changes

For adaptive changes to occur within the physiological system of individuals going through an exercise training program, the various components of exercise need to be facilitating. Research has shown that the intensity of exercise needs to be of sufficient magnitude to adequately overload the circulatory system (Froelicher, 1983). Intensities requiring anywhere from 60 to 80 percent of the maximum heart rate (MHR) are needed to elicit training responses in both healthy and cardiac patients (Patterson, et al., 1979; Stuart, et al., 1985; Vanhees, et al., 1982). Frequency and duration of the exercise program are crucial to the development of any adaptive changes. A frequency of three to five days per week, along with a duration of at least three months, are needed to produce adaptive changes in all population of exercisers (Fox, et al., 1975). The mode, or type, of exercise is also critical (Pollock, et al., 1975) especially to CAD and CAD-hypertensive subjects (Froelicher). Dynamic work, like bicycling, running, and jogging, requires the movement of

large muscle masses and requires a high blood flow and increased cardiac output. Since this movement is rhythmic there is little resistance to flow and, in fact, there is the "milking" action by the lower-extremity muscles that returns blood to the heart. Dynamic work resulting in limb movement can be accurately calibrated and the physiological response easily measured. Therefore, it is preferred for clinical training and testing. Using progressive work loads of dynamic exercise, patients with CAD and/or hypertension can be protected from rapidly increasing myocardial oxygen demand (Froelicher, 1983).

Another type of muscular movement is static (isometric) muscular work. Isometric exercise, defined as constant muscular contraction without movement (i.e., hand grip, or pushing against an immovable object), imposes a disproportionate pressure load on the left ventricle relative to the body's ability to supply oxygen (Froelicher, 1983). In isometric exercise, the sudden and disproportionate increase in blood pressure imposes a great demand on heart's muscle tissues for oxygen. In ischemic hearts, where oxygen supply is of paramount importance, this extra load is not needed. Unlike dynamic work, isometric work can not be calibrated. Although this kind of exercise is good for peripheral muscle tone and function, resulting in strength, it does not result in the beneficial cardiac and hemodynamic effects of dynamic exercise training (Froelicher).

Review of Literature Pertaining to Post MI Training Studies

Vanhees and colleagues (1982) compared two groups of post-MI patients who exercised at an intensity that was between 60% and 80% of their maximum capacity. The program required both groups to jog three days a week and for a total of three months. One group (15 subjects) received constant beta-blocking medication, and the other (15 subjects) did not. Both groups showed lower heart rates, systolic blood pressure, and rate pressure product, both at rest and at submaximum exercise testing (Vanhees, et al.).

May and Nagel (1984) showed in a study of 121 CAD subjects that after 10 to 12 months of aerobic exercise training, significant reductions occurred in resting and in submaximum exercise heart rates, mean blood pressure, and RPP. The intensity and frequency of the program was similar to that used in the Vanhees, et al. study (1982).

In a study of 10 men, aged 44 to 62 years, an aerobic exercise program of jogging performed three days a week for a total of 12 weeks produced increases in exercise duration time and estimated maximum oxygen consumption, and decreases in RPP at a given absolute submaximum work load. Since subjects were under beta-blockage medicine therapy, the researchers concluded that such medicines do not prevent the normal physiological adaptation to exercise (McGhee, Siconolfi, & Bouchard, 1984). The intensity of this program required heart rates of 65 to 80 percent of each individual's maximum exercise heart rates, as determined during pre-training graded exercise stress tests.

Stuart and Associates (1985) studied CAD patients who went through a bicycle-exercise training program of 12 to 16 weeks. Training sessions lasted 30-40 minutes, three times a week, with training heart rates of 75 to 85 percent of pre-training peak heart rates. The mean values for maximum oxygen consumption and total exercise time increased significantly after the training program. Researchers reported that the increase in maximum oxygen consumption was due to increases in stroke volume and peripheral oxygen extraction.

Froelicher (1984) reported beneficial hemodynamic responses to a running exercise training program lasting one year. The exercise sessions lasted forty-five minutes and were performed three times a week. The initial training intensity was set at a minimum of 60 percent of the estimated maximum oxygen uptake determined with an initial treadmill test. The intensity was increased to 85 percent by the eighth month of training. The 72 middle-aged male CAD patients showed significant differences in hemodynamic measurements when compared to the 74 male counterparts forming the non-exercising control group. Parameters under investigation were aerobic capacity, left ventricular function, and thallium-ischemic scores.

Summary

Dynamic exercise training programs utilizing large muscle groups as in running and bicycling have been shown to benefit CAD and hypertensive subjects by increasing their physical working capacity (May & Nagle, 1984). Programs lasting for three months or more, at

intensities between 60 to 85% of maximum heart rates, performed three days or more per week, have been utilized by researchers (McGhee, Siconolfi, & Bouchard, 1984).

Peripheral adaptive responses bringing about physiological and hemodynamic changes have been elucidated. Decreases in resting and submaximum exercise heart rate and blood pressure have been shown to result from participation in training programs (Debacker, 1974). These changes bring about a state of efficiency within the cardiovascular system by which myocardial oxygen consumption is reduced and thus reduces the demand on the heart muscle during work. This reduction in demand is especially appreciated within the CAD population whose ischemic condition has reduced the heart's ability for doing work. The reduced myocardial oxygen consumption secondary to reduced demand on the heart's muscle tissues delays the onset of angina pains and allows subjects to exercise for longer periods of time (May & Nagle, 1984).

Neurological changes, as evidenced by lower sympathetic nervous system participation and increased parasympathetic nervous system involvement, and biochemical adaptations, as evidenced by increased enzymatic activity within the muscle cells, are cited as the main reason for cardiovascular training effects (Froelicher, 1983).

CHAPTER III

Methodology

This chapter contains information about the subjects, the YMCA exercise protocol, measurement of variables, research design, and statistical procedures.

Subjects

The medical records (pre-test and post-test scores on all six dependent variables) of a total of 100 male patients between the ages of 45 and 60 years of age were analyzed. A letter of permission to use medical files was obtained from the Medical Director of YMCA Cardiac Therapy Program and was filed with San Jose State University's Institutional Review Board. A copy has been provided in Appendix A. Fifty were in the trained (experimental) group and fifty in the control group. The experimental group included only members of the Central YMCA Cardiac Therapy Program in San Jose, California, between the years 1978 and 1985. They were approved for participation into the YMCA program by their personal physicians and by the physician directing the YMCA program. The control group consisted of patients in a private medical practice and did not participate in any structured exercise programs between the years 1978 and 1985. The control group medical files came from between the years 1978 and 1985. The following criteria were used to select subjects for this study:

1. All subjects were adult white males between the ages of 45 and 60.
2. All subjects were diagnosed as CAD patients.

3. All subjects had experienced heart attacks (MI) and were at least three month post-MI before their initial stress test.

4. All experimental group subjects entered the YMCA exercise program within a month of the initial stress test.

5. All subjects had coronary bypass surgery performed on them within a month from the time of their MI.

6. To belong to a hypertensive subgroup, patients had to be clearly labeled as such in their respective files, and have blood pressure recordings of greater than 160 mmHg and 90 mmHg for systolic and diastolic readings, respectively.

7. Records contained all information collected for this study.

8. To belong to the experimental group, patients had to have attended 75% or more of the exercise sessions lasting nine months.

The YMCA Exercise Protocol

The participants reported to the gymnasium of the YMCA three days a week for a total of 60 minutes per day for nine consecutive months. The program consisted of three separate but interrelated segments. Warm-ups, consisting of calisthenics and stretching, comprised the initial ten minutes of each session. The conditioning phase, consisting of jogging, lasted for 35 minutes. During this phase, individually prescribed intensities were monitored by each subject, who had been taught to monitor their heart rates by palpating the carotid artery. This intensity corresponded to 60 to 75 percent of each subject's symptom-limited maximum heart rate (MHR) during the first six weeks of the training program and to 85 percent of their

symptom-limited MHR for the remainder of the nine months. The cool-down period was the last segment and included stretching routines lasting 15 minutes. A detailed description of the entire YMCA protocol, which is a standard protocol at all the YMCA Cardiac Rehabilitation Programs, is available in Gary and Berra (1981). A copy of the YMCA data form is provided in Appendix B.

Dependent Variables

Medical files belonging to both experimental (YMCA Cardiac Therapy Program), and control (private medical practice) groups were examined in this study. Raw data pertaining to subjects' body weights, resting and submaximum exercise heart rates, resting and submaximum exercise mean blood pressure, and submaximum exercise rate pressure product were extracted from the files. The pre and post-testing submaximum figures for the above variables were collected at the end of the third stage of a modified Bruce exercise testing protocol (Bruce, 1971). Both groups were tested using the same YMCA testing protocol. All subjects were tested twice, with a nine month separation time between tests.

Research Design

This study was a pre-test, post-test control group design. The 50 CAD subjects comprising each group were divided equally into two subgroups: hypertensives and non-hypertensives. The following subgroups were delineated for study:

EXHT	Exercise group with hypertension
EXNT	Exercise group who are normotensive

CHT	Control group with hypertension
CNT	Control group who are normotensive

Each group contained 25 subjects.

Data Analysis

Changes in weight, resting and submaximum heart rates, resting and submaximum mean blood pressures, and submaximum rate-pressure product from initial tests to final tests for all subjects were calculated. Descriptive statistics (mean, standard deviation, and range) for all variables belonging to each subgroup's pre-testing and post-testing scores were determined. Two different approaches to inferential statistics were utilized. First, t-tests for independent groups were applied to compare the 50 subjects of the experimental group (25 hypertensives and 25 non-hypertensives) with the 50 subjects of the control group (25 hypertensives and 25 non-hypertensives) on all six dependent variables. Through this method, the differences between pre-test means and post-test means were obtained and then compared. This approach enabled the author to test the first hypothesis. Second, to account for possible contrast between hypertensive and non-hypertensive subgroups within each main group, two 2-way ANOVAS were calculated. This approach enabled the author to test the second hypothesis.

The alpha level for the determination of statistical significance was set at .05 at the outset. However, to account for the multiple cross-group (t-tests) and cross-subgroup comparisons (two 2-way ANOVAS), a new alpha level was obtained by dividing .05 by the number 18 (six 2-way ANOVAS for

the experimental group, six 2-way ANOVAS for the control, and six t-tests for the comparisons between experimental and control groups. Thus .003 became the adjusted new alpha level. The book titled Applied Statistics for Social Sciences (Glass & Stanley, 1970) was consulted for statistical purposes pertaining to this study.

CHAPTER IV

Results & Discussion

Introduction

The following chapter includes descriptive statistics pertaining to all four subgroups and t-tests for independent groups pertaining to the comparison of the experimental and control groups (irrespective of the hypertensive/non-hypertensive subdivisions). It also contains inferential statistics utilizing two 2-way ANOVAS to determine if interaction exists between the main effects of hypertension and exercise within the experimental and control groups and a discussion of the results.

Descriptive Data

Experimental Hypertensive Subgroup (EXHT)

Age and Weight

The personal data for EXHT is listed in Table 1. Raw scores, means, standard deviations, and ranges for age and weight are listed. This subgroup ranged in age from 45 to 60 years of age, with a mean of 54.6. The weight had group means of 73 kg. for pre-testing and 71.2 kg. for post-testing. A change of body weight of -2.5% was calculated.

Resting Heart Rate (RHR)

Raw scores and descriptive statistics for RHR are listed in Table 2. This subgroup had means of 70.4 bpm for pre-testing and 67.2 bpm for post-testing. A change of -4.8% was calculated.

TABLE 1

Means, Standard Deviations, and Range

for Age and Weight for EXHT, Pre and Post-Testing

Subject	Age (years)	Weight (kg.)	
		Pre-testing	Post-testing
1	60	72	88
2	50	82	80
3	55	65	64
4	59	65	64
5	60	77	72
6	49	69	71
7	53	64	63
8	53	72	76
9	60	70	69
10	53	72	68
11	52	70	70
12	58	60	62
13	60	71	70
14	45	60	60
15	57	74	77
16	51	69	59
17	51	81	84
18	50	79	76
19	60	73	70
20	59	72	70
21	47	75	72
22	49	81	75
23	58	74	70
24	57	79	76
25	60	80	74
Mean	54.6	73.0	71.2
SD	4.8	7.4	7.1
Range	60-45	92-60	88-59

TABLE 2

Means, Standard Deviations, and Range for Resting Heart Rate (RHR), Submaximum Heart Rate (SHR), Resting Mean Blood Pressure (RMBP), Submaximum Mean Blood Pressure (SMBP), and Submaximum Rate Pressure Product (SRPP) for EXHT, Pre & Post-Tests

	RHR		SHR		RMBP		SMBP		SRPP	
Subject	pre	post	pre	post	pre	post	pre	post	pre	post
1	62	55	146	136	103	101	114	112	236	212
2	66	62	140	132	103	102	108	106	207	192
3	70	72	160	162	101	97	130	114	272	268
4	84	80	180	175	103	101	113	110	324	315
5	84	84	128	132	103	106	122	122	240	240
6	60	65	130	122	107	104	120	117	234	213
7	56	54	150	150	89	88	103	106	195	198
8	62	55	146	136	103	101	126	118	292	242
9	75	72	170	160	109	103	112	106	289	256
10	50	42	110	110	99	99	107	107	165	165
11	84	80	120	110	115	112	143	140	252	224
12	70	64	130	135	107	120	125	101	252	221
13	54	58	130	110	96	79	133	111	234	170
14	63	60	140	132	97	97	120	114	254	227
15	80	72	170	161	97	93	120	112	272	260
16	72	73	112	104	89	87	106	103	188	176
17	68	70	130	120	87	72	95	93	188	150
18	84	82	180	185	89	87	133	130	360	239
19	80	76	180	178	103	98	133	129	360	334
20	64	58	148	140	109	140	140	140	310	288
21	75	72	160	164	101	102	118	114	252	249
22	70	65	160	162	103	95	114	109	268	259
23	72	68	172	164	107	105	126	120	323	295
24	75	70	185	183	105	104	119	120	336	334
25	81	72	179	170	104	101	115	109	332	306
Mean	70.4	67.2	150.2	145.1	101.2	98.6	119.8	114.6	265.4	245.3
SD	7.9	10.1	22.9	23.8	7.0	10.2	11.7	11.1	54.8	55.2
Range	84-50	84-42	185-110	183-109	109-87	140-72	143-95	140-95	360-165	334-150

Submaximum Heart Rate (SHR)

Raw scores and descriptive statistics for SHR are listed in Table 2. This subgroup had means of 150.2 bpm for pre-testing and 145.1 bpm for post-testing. A change of -3.6% was calculated.

Resting Mean Blood Pressure (RMBP)

Raw scores and descriptive statistics for RMBP are listed in Table 2. This subgroup had means of 101.2 mmHg for pre-testing and 98.3 mmHg for post-testing. A change of -2.6% was calculated.

Submaximum Mean Blood Pressure (SMBP)

Raw scores and descriptive statistics for SMBP are listed in Table 2. This subgroup had means of 119.8 mmHg for pre-testing and 114.6 mmHg for post-testing. A change of -4.6% was calculated.

Submaximum RPP (SRPP)

Raw scores and descriptive statistics for SRPP are listed in Table 2. This subgroup had means of 265.4 for pre-testing and 245.3 for post-testing. A change of -8.2% was calculated.

Experimental Non-hypertensive Subgroup (EXNT)

Age and weight

The personal data for EXNT is listed in Table 3. Raw scores, means, standard deviations, and range for age and weight are listed. This subgroup ranged in age from 48 to 60 years, with a mean of 53.5. The group mean for body weight was 70.7 kg for pre-testing and 68.4 kg for post-testing. A change of -3.3% was calculated.

TABLE 3

Means, Standard Deviations, and Range

for Age and Weight for EXNT, Pre and Post-Test Scores

Subject	Age (in years)	Weight (kg.) Pre-testing	Weight (kg.) Post-testing
1	59	69	68
2	58	74	78
3	59	80	80
4	60	63	59
5	51	61	64
6	48	72	74
7	60	70	63
8	55	63	63
9	55	72	64
10	52	80	74
11	59	77	72
12	60	80	76
13	60	82	74
14	60	69	62
15	60	72	68
16	54	66	62
17	55	72	69
18	56	60	62
19	49	59	63
20	56	69	68
21	57	71	74
22	52	70	65
23	53	59	58
24	52	79	77
25	60	80	74
Mean	56.7	70.7 kg.	68.4 kg.
SD	3.9	7.2	6.4
Range	60-48	82-59	80-58

Resting Heart Rate (RHR)

Raw scores and descriptive statistics for RHR are listed in Table 4. This subgroup had means of 69.3 bpm for pre-testing and 64.7 bpm for post-testing. A change of -7.2% was calculated.

Submaximum Heart Rate (SHR)

Raw scores and descriptive statistics for RHR are listed in Table 4. This subgroup had means of 159.2 bpm for pre-testing and 152.7 bpm for post-testing. A change of -4.3% was calculated.

Resting Mean Blood Pressure (RMBP)

Raw scores and descriptive statistics for RMBP are listed in Table 4. This subgroup had means of 96.7 mmHg for pre-testing and 93.7 mmHg for post-testing. A change of -3.1% was calculated.

Submaximum Mean Blood Pressure (SMBP)

Raw scores and descriptive statistics for SMBP are listed in Table 4. This subgroup had means of 114.5 mmHg for pre-training and 111.0 mmHg for post-training. A change of -3.1% was calculated.

Submaximum RPP (SRPP)

Raw scores and descriptive statistics for SRPP are listed in Table 4. This subgroup had means of 276.6 for pre-testing and 258.6 for post-testing. A change of -7.0% was calculated.

TABLE 4

Means, Standard Deviations, and Range for Resting Heart Rate (RHR), Submaximum Heart Rate (SHR), Resting Mean Blood Pressure (RMBP), Submaximum Mean Blood Pressure (SMBP), and Submaximum Rate Pressure Product (SRPP) for EXNT, Pre & Post-Tests

	RHR		SHR		RMBP		SMBP		SRPP	
Subject	pre	post	pre	post	pre	post	pre	post	pre	post
1	69	64	149	141	101	100	121	112	262	242
2	71	70	159	160	100	96	118	113	289	281
3	79	75	179	175	106	97	116	114	318	304
4	70	68	152	144	102	99	116	113	261	241
4	60	57	161	148	96	91	102	100	260	236
5	61	55	148	140	94	88	118	111	236	238
7	62	59	140	132	92	89	110	110	226	211
8	64	63	170	168	87	93	111	111	302	305
9	70	70	170	162	103	103	120	111	302	272
10	69	63	150	146	90	88	106	112	240	245
11	76	70	149	142	98	92	113	110	268	244
11	67	65	149	141	92	93	114	112	268	250
13	72	66	170	162	106	102	124	120	316	291
14	71	66	161	154	99	93	98	91	228	206
15	70	67	181	172	89	83	112	107	325	292
16	69	63	150	145	95	95	115	112	255	237
17	71	60	177	167	92	87	114	108	304	277
18	66	61	143	140	100	89	113	110	252	238
19	78	72	173	165	86	85	107	103	294	273
20	69	64	169	163	104	105	123	119	294	277
21	75	70	175	170	103	101	123	122	325	312
22	80	69	166	100	97	90	115	106	282	256
23	60	59	130	129	96	97	115	115	221	219
24	73	66	170	161	92	91	116	110	294	264
25	61	55	170	161	103	99	122	124	264	254
Mean	69.3	64.7	159.2	152.7	96.7	93.7	114.5	111.0	276.6	258.6
SD	5.8	5.3	14.1	13.8	5.9	6.0	6.4	6.8	30.7	29.1
Range	80-60	72-55	181-130	175-129	106-87	103-83	124-98	124-91	325-221	312-206

Control Hypertensive Subgroup (CHT)

Age and Weight

The personal data for CHT is listed in Table 5. Raw scores, means, standard deviation, and range for age and weight are listed. This subgroup ranged in age from 49 to 60 years, with a mean of 58.7. The weight had group means of 63.6 kg for pre-testing and 64.1 kg for post-testing. A change of +.7% was calculated.

Resting Heart Rate (RHR)

Raw scores and descriptive statistics for RHR are listed in Table 6. This subgroup had means of 64.1 bpm for pre-testing and 64.8 bpm for post-testing. A change of +1.1% was calculated.

Submaximum Heart Rate (SHR)

Raw scores and descriptive statistics for SHR are listed in Table 6. This subgroup had means of 140.7 bpm for pre-testing and 141.2 bpm for post-testing. A change of +.3% was calculated.

Resting Mean Blood Pressure (RMBP)

Raw scores and descriptive statistics for RMBP are listed in Table 6. This subgroup had means of 102.6 mmHg for pre-testing and 102.4 mmHg for post-testing. A change of -.2% was calculated.

Submaximum Mean Blood Pressure (SMBP)

Raw scores and descriptive statistics for SMBP are listed in Table 6. This subgroup had means of 113.9 mmHg for pre-testing and 115.1 mmHg for post-testing. A change of +1.0% was calculated.

TABLE 5

Means, Standard Deviations, and Range

for Age and Weight for CHT, Pre and Post-Test Scores

Subject	Age (in years)	Weight (kg.)	
		Pre-testing	Post-testing
1	60	55	55
2	60	62	63
3	60	70	70
4	55	65	64
5	60	60	62
6	53	59	56
7	49	64	66
8	55	70	71
9	50	59	56
10	49	65	67
11	59	70	69
12	60	64	60
13	60	69	69
14	53	62	70
15	60	71	72
16	51	60	62
17	54	54	53
18	55	61	70
19	57	59	66
20	60	70	69
21	49	60	63
22	60	60	62
23	60	63	58
24	59	72	68
25	60	66	61
Mean	56.3	63.6	64.1
SD	5.6	5.1	5.5
Range	60-49	72-54	72-53

TABLE 6

Means, Standard Deviations, and Range for Resting Heart Rate (RHR), Submaximum Heart Rate (SHR), Resting Mean Blood Pressure (RMBP), Submaximum Mean Blood Pressure (SMBP), and Submaximum Rate Pressure Product (SRPP) for CHT, Pre & Post-Test Scores

	RHR		SHR		RMBP		SMBP		SRPP	
Subject	pre	post	pre	post	pre	post	pre	post	pre	post
1	55	55	118	117	105	101	120	126	214	222
2	70	74	129	134	102	105	118	118	229	241
3	70	69	140	147	109	113	126	124	266	285
4	71	74	160	158	108	109	121	122	275	274
5	75	74	150	152	97	95	108	108	252	249
6	79	78	149	146	107	107	118	121	268	268
7	62	68	132	135	102	102	118	120	227	234
8	70	68	148	148	107	108	122	125	278	278
9	67	64	140	136	93	89	102	100	240	225
10	66	70	122	120	96	93	109	113	195	196
11	70	72	150	154	105	106	112	115	249	255
12	60	57	130	131	100	100	111	114	200	206
13	71	74	148	146	103	103	107	107	239	230
14	54	64	135	150	107	113	119	128	245	294
15	71	71	150	144	99	97	111	110	255	244
16	64	60	149	142	99	97	109	108	250	232
17	54	53	130	132	101	107	108	109	205	205
18	57	60	149	152	98	101	111	110	253	261
19	60	62	160	160	105	106	116	118	275	272
20	60	60	155	152	101	103	107	112	232	237
21	55	60	140	144	95	99	110	115	238	250
22	50	54	120	124	107	109	116	117	204	213
23	60	64	132	140	99	95	107	100	213	221
24	73	66	150	140	110	102	126	122	282	260
25	59	50	132	126	109	101	116	116	234	214
Mean	64.1	64.8	140.7	141.2	102.6	102.4	113.9	115.1	240.7	242.0
SD	7.7	7.9	12.1	11.6	4.7	6.0	6.3	7.5	25.6	26.0
Range	79-50	78-50	160-118	160-117	110-93	113-89	126-102	128-100	282-195	294-205

Submaximum RPP (SRPP)

Raw scores and descriptive statistics for SRPP are listed in Table 6. This subgroup had means of 240.7 for pre-testing and 242.0 for post-testing. A change of +.8% was calculated.

Control Non-hypertensive Subgroup (CNT)

Age and Weight

The personal data for CNT is listed in Table 7. Raw scores, means, standard deviation, and range for age and weight are listed. This subgroup ranged in age from 45 to 60 years, with a mean of 55.3. The weight had group means of 67.0 kg for pre-testing and 66.6 kg for post-testing. A change of –.7% was calculated.

Resting Heart Rate (RHR)

Raw scores and descriptive statistics for RHR are listed in Table 8. This subgroup had means of 70.7 bpm for pre-testing and 69.5 bpm for post-testing. A change of –1.4% was calculated.

Submaximum Heart Rate (SHR)

Raw scores and descriptive statistics for SMBP are listed in Table 8. This subgroup had means of 139.0 bpm for pre-testing and 139.2 bpm for post-testing. A change of –.6% was calculated.

Resting Mean Blood Pressure (RMBP)

Raw scores and descriptive statistics for RMBP are listed in Table 8. This subgroup had means of 98.0 mmHg for pre-training and 99.2 mmHg for post-training. A change of +.9% was calculated.

TABLE 7

Means, Standard Deviations, and Range

for Age and Weight for CNT, Pre and Post-Test Scores

Subject	Age (in years)	Weight (kg.)	
		Pre-testing	Post-testing
1	60	55	55
2	60	62	63
3	60	70	70
4	55	65	64
5	60	60	62
6	53	59	56
7	49	64	66
8	55	70	71
9	50	59	56
10	49	65	67
11	59	70	69
12	60	64	60
13	60	69	69
14	53	62	70
15	60	71	72
16	51	60	62
17	54	54	53
18	55	61	70
19	57	59	66
20	60	70	69
21	49	60	63
22	60	60	62
23	60	63	58
24	59	72	68
24	60	66	61
Mean	56.4	67.0	66.6
SD	4.5	8.0	9.3
Range	60-45	83.5	89.5

TABLE 8

Means, Standard Deviations, and Range for Resting Heart Rate (RHR), Submaximum Heart Rate (SHR), Resting Mean Blood Pressure (RMBP), Submaximum Mean Blood Pressure (SMBP), and Submaximum Rate Pressure Product (SRPP) for CNT, Pre & Post-Test Scores

	RHR		SHR		RMBP		SMBP		SRPP	
Subject	pre	post	pre	post	pre	post	pre	post	pre	post
1	55	55	118	117	105	101	120	126	214	222
2	70	74	129	134	102	105	118	118	229	241
3	70	69	140	147	109	113	126	124	266	285
4	71	74	160	158	108	109	121	122	275	274
5	75	74	150	152	97	95	108	108	252	249
6	79	78	149	146	107	107	118	121	268	268
7	62	68	132	135	102	102	118	120	227	234
8	70	68	148	148	107	108	122	125	278	278
9	67	64	140	136	93	89	102	100	240	225
10	66	70	122	120	96	93	109	113	195	196
11	70	72	150	154	105	106	112	115	249	255
12	60	57	130	131	100	100	111	114	200	206
13	71	74	148	146	103	103	107	107	239	230
14	54	64	135	150	107	113	119	128	245	294
14	71	71	150	144	99	97	111	110	255	244
16	64	60	149	142	99	97	109	108	250	232
16	54	53	130	132	101	107	108	109	205	205
18	57	60	149	152	98	101	111	110	253	261
19	60	62	160	160	105	106	116	118	275	272
20	60	60	155	152	101	103	107	112	232	237
21	55	60	140	144	95	99	110	115	238	250
22	50	54	120	124	107	109	116	117	204	213
23	60	64	132	140	99	95	107	100	213	221
24	73	66	150	140	110	102	126	122	282	260
25	59	50	132	126	109	101	116	116	234	214
Mean	70.5	69.5	139.0	139.2	98.0	99.2	108.5	109.3	222.0	224.0
SD	8.9	8.7	19.2	18.8	7.7	7.9	6.5	7.7	37.2	42.2
Range	87-52	88-54	172-100	178-100	108-80	111-81	121-96	124-96	323-166	358-158

Submaximum Mean Blood Pressure (SMBP)

Raw scores and descriptive statistics for SMBP are listed in Table 8. This subgroup had means of 108.5 mmHg for pre-testing and 109.3 mmHg for post-testing. A change of +.7% was calculated.

Submaximum RPP (SRPP)

Raw scores and descriptive statistics for SRPP are listed in Table 8. This subgroup had means of 222.0 mmHg for pre-testing and 224.1 mmHg for post-testing. A change of +.9% was calculated.

Group Comparisons

For descriptive and comparative purposes, Table 9 provides pre-test and post-test means for all dependent variables belonging to all subgroups. Table 10 provides percent changes from pre-test to post-test for all dependent variables belonging to all subgroups.

Inferential Statistics (t-tests)

The t-tests for independent groups were used to compare the experimental and control groups. Differences between pre-test means and post-test means were obtained for both experimental and control groups on all six dependent variables and then compared to one another. The following results for the respective dependent variables were obtained.

Weight. The t-test showed the difference between the experimental and control groups to be significant (t (98) = 2.8, p = .002).

Resting Heart Rate. The t-test showed the difference between the experimental and control groups to be significant (t (98) = 5.2, p = .000).

TABLE 9

Pre-Test & Post-Test Mean Scores for All Variables Belonging to All Four Subgroups

	Weight[a]		RHR[b]		SHR[c]		RMBP[d]		SMBP[e]		SRPP[f]	
Subject	pre	post	pre	post	pre	post	pre	post	pre	post	pre	post
EXHT[g]	73.0	71.2	70.4	67.2	150.2	145.1	101.2	98.6	119.8	114.6	265.4	245.3
CHT[h]	63.6	64.1	64.1	64.8	140.7	141.2	102.6	102.4	113.9	115.1	240.7	242.6
EXNT[i]	70.7	68.4	69.3	64.7	159.2	152.7	96.7	93.8	114.5	111.0	276.6	258.6
CNT[j]	67.0	66.6	70.5	69.5	139.3	139.2	98.2	99.2	108.6	109.3	222.0	224.1

a. Body weight in kilograms
b. Resting heart rate (bpm)
c. Submaximum exercise heart rate (bpm)
d. Resting mean blood pressure (mmHg)
e. Submaximum exercise mean blood pressure (mmHg)
f. Submaximum exercise rate-pressure product
g. Exercise subgroup with hypertension
h. Non-exercise subgroup with hypertension
i. Exercise subgroup normotensive
j. Non-exercise subgroup normotensive

Table 10

Percent Changes for the Means of all the Variables for All Four Subgroups

	WT[a]	RHR[b]	SHR[c]	RMBP[d]	SMBP[e]	RPP[f]
EXHT[g]	-2.5	-4.8	-3.6	-2.6	-4.6	-8.2
CHT[h]	+.7	+1.1	+.3	- .2	+1.0	+.8
EXNT[i]	-3.3	-7.1	-4.3	-3.1	-3.1	-7.0
CNT[j]	-.7	-1.4	-.1	+.9	+.7	+.9

a. Body weight in kilograms
b. Resting heart rate (bpm)
c. Submaximum exercise heart rate (bpm)
d. Resting mean blood pressure (mmHg)
e. Submaximum exercise mean blood pressure (mmHg)
f. Submaximum exercise rate-pressure product
g. Exercise subgroup with hypertension
h. Non-exercise subgroup with hypertension
i. Exercise subgroup normotensive
j. Non-exercise subgroup normotensive

Submaximum Heart Rate. The t-test showed the difference between the experimental and control groups to be significant (t (98) = 5.9, p = .000).

Resting Mean Blood Pressure. The t-test showed the difference between the experimental and control groups to be significant (t (98) = 3.8, p = .000).

Submaximum Mean Blood Pressure. The t-test showed the difference between the experimental and control groups to be significant (t (98) = 5.9, p = .000).

Submaximum Rate-Pressure Product. The t-test showed the difference between the experimental and control groups to be significant (t (98) = 7.6 p = .000).

Inferential Statistics (ANOVA)

With the use of two 2-way ANOVAS, the two main effects of exercise (pre-post scores) and hypertension within each two main groups of experimental and control were examined. The 2-way ANOVAS yielded the following results.

Weight. Table 11 lists the F values for the main effects of hypertension, pre-post scores, and the interaction between the two, for both experimental and control groups. Neither group yielded F values that indicated a statistically significant interaction.

Resting Heart Rates. Table 12 lists the F values for the main effects of hypertension, pre-post scores, and the interaction between the two, for both

TABLE 11

Two 2-way ANOVAS for the Body Weight

For the Determination of an Interaction Between the Two Main Effects of Hypertension and Exercise (Pre-Post Scores) Within Both Experimental and Control Groups

Experimental

Main Effect	SS	DF	MS	F	p
Hypertension	158.8	1	158.8	1.7	.197
Pre-Post Scores	104.0	1	104.0	16.1	<.001
Interaction	1.4	1	1.4	.2	.639

Control

Main Effect	SS	DF	MS	F	p
Hypertension	196.0	1	196.0	2.1	.152
Pre-Post Scores	.6	1	.6	.1	.718
Interaction	10.2	1	10.2	2.1	.152

TABLE 12

Two 2-way ANOVAS for Resting Heart Rate

Determining the Interaction Between the Two

Main Effects of Hypertension and Exercise

Within Both Experimental and Control Groups

Experimental

Main Effect	SS	DF	MS	F	p
Hypertension	84.6	1	84.6	.7	.42
Pre-Post Scores	384.2	1	384.2	70.7	<.001
Interaction	12.9	1	12.9	2.4	.013

Control

Main Effect	SS	DF	MS	F	p
Hypertension	756.2	1	756.2	5.8	.020
Pre-Post Scores	.5	1	.5	.1	.797
Interaction	18.5	1	18.5	2.5	.199

experimental and control groups. Neither group yielded F values that indicated a statistically significant interaction.

Submaximum Heart Rates. Table 13 lists the F values for the main effects of hypertension, pre-post scores, and the interaction between the two, for both experimental and control groups. Both groups had F values for interaction that was not statistically significant.

Resting Mean Blood Pressure. Table 14 lists the F values for the main effects of hypertension, pre-post scores, and the interaction between the two, for both experimental and control groups. Both groups had F values for interaction that was not statistically significant.

Submaximum Mean Blood Pressure. Table 15 lists the F values for the main effects of hypertension, pre-post scores, and the interaction between the two, for both experimental and controls. Both groups had F values for interaction that was not significant.

Submaximum Rate-Pressure Product. Table 16 lists the F values for the main effects of hypertension, pre-post scores, and the interaction between the two, for both experimental and control groups. Both groups had F values for interaction that was not significant.

Discussion

The training program for the present study was similar to many other programs reported in the literature (Vanhees, et al., 1982; May & Nagle, 1984; McGhee, et al., 1984). Research has shown that aerobic training programs utilizing large muscle groups, lasting at least three months, with a frequency of three days per week, and duration of 30 minutes at intensities of 65-80% of the maximum heart rate, are adequate

TABLE 13

Two 2-way ANOVAS for Submaximum Heart Rate for the Determination of an Interaction Between the Two Main Effects of Hypertension and Exercise (Pre-Post Scores) Within Both Experimental and Control Groups

Experimental

Main Effect	SS	DF	MS	F	p
Hypertension	1713.9	1	1713.9	2.3	.132
Pre-Post Scores	852.6	1	852.6	75.7	<.001
Interaction	11.6	1	11.6	1.0	.316

Control

Main Effect	SS	DF	MS	F	p
Hypertension	70.6	1	70.6	.1	.706
Pre-Post Scores	1.0	1	1.0	.1	.796
Interaction	1.9	1	1.9	.1	.719

TABLE 14

Two 2-way ANOVAS for Mean Blood Pressure for the Determination of an Interaction Between the Two Main Effects of Hypertension and Exercise (Pre-Post Scores) Within Both Experimental and Control Groups

Experimental

Main Effect	SS	DF	MS	F	p
Hypertension	501.8	1	501.8	5.1	.029
Pre-Post Scores	201.6	1	201.6	18.4	<.001
Interaction	.0	1	.0	.0	1.00

Control

Main Effect	SS	DF	MS	F	p
Hypertension	361.0	1	361.0	4.3	.044
Pre-Post Scores	4.0	1	4.0	.5	.463
Interaction	6.8	1	6.8	.9	.341

TABLE 15

Two 2-way ANOVAS for Submaximum Exercise Mean Blood Pressure for the Determination of an Interaction Between the Two Main Effects of Hypertension and Exercise (Pre-Post Scores) Within Both Experimental and Control Groups

Experimental

Main Effect	SS	DF	MS	F	p
Hypertension	488.4	1	488.4	3.6	.087
Pre-Post Scores	470.6	1	470.6	33.8	<.001
Interaction	20.2	1	20.2	1.4	.234

Control

Main Effect	SS	DF	MS	F	p
Hypertension	778.4	1	778.4	8.3	.006
Pre-Post Scores	24.0	1	24.0	4.0	.052
Interaction	1.2	1	1.2	.2	.657

TABLE 16

Two-way ANOVAS for Submaximum Exercise Rate-Pressure Product for the Determination of an Interaction Between the Two Main Effects of Hypertension and Exercise (Pre-Post Scores) Within Both Experimental and Control Groups

Experimental

Main Effect	SS	DF	MS	F	p
Hypertension	3733.2	1	3733.2	1.0	.328
Pre-Post Scores	9082.1	1	9082.1	100.1	<.001
Interaction	28.1	1	28.1	.3	.581

Control

Main Effect	SS	DF	MS	F	p
Hypertension	8667.6	1	8667.6	4.0	.051
Pre-Post Scores	98.0	1	98.0	1.0	.331
Interaction	.1	1	.1	.0	.976

to elicit beneficial training responses in physiological (e.g., heart rate and blood pressure) , and hemodynamic parameters (e.g., vascular resistance to blood flow and arterio-venous oxygen difference) in cardiac patients (Ogawa, et al., 1981). Anthropometric parameters (body weight) and cardiac structural changes (e.g., increased capillarization within muscle tissues) have also been influenced by exercise programs within the cardiac populations (Ehsani, et al., 1981). The results of this study stem from measuring indices representing physiological and anthropometric parameters (weight only). Therefore, hemodynamic and structural changes, if any, could not be identified in this study.

Body Weight (kg)

Both exercising subgroups (EXHT & EXNT) showed weight loss after training (–2.5% and –3.3%, respectively). The control subgroups showed conflicting results. The control hypertensive subgroups showed a small gain of +.7% and the control non-hypertensive subgroup showed a small reduction of -.7%. The *t*-test for independent groups showed the difference between the experimental and control groups to be statistically significant ($p < .003$). Two two-way ANOVAS within each group showed no interaction between hypertension and exercise ($p > .003$). Thus, there were no statistically significant differences between hypertensive and non-hypertensive subgroups within each main group when their pre-test and post-test scores were compared. The percentages of weight reduction for the experimental subgroups was similar to a study done by Stuart, et al. (1985), in which he reported a decrease of 3.62% after four months of aerobic training. He found this percent change to be significant ($p < .04$). McGhee,

et al. (1984) showed a similar statistically significant weight reduction ($p < .045$), while others did not (Ben Ari, 1986; Ogawa, et al., 1981). These inconsistencies may be due to the lack of control over caloric intake in most studies (Froelicher, 1983).

Resting Heart Rate

The changes of resting heart rate reported for both experimental subgroups (–4.8%, hypertensives and –7.2%, non-hypertensives) were larger than changes for controls (+1.1%, hypertensives and –1.4%, non-hypertensives). The t-test for independent groups showed the difference between the experimental and control groups to be statistically significant ($p < .003$). Two-way ANOVAS within each group showed no interaction between hypertension and exercise ($p > .003$). Thus, there were no statistically significant differences between hypertensive and non-hypertensive subgroups within each main group when their pre-test and post-test scores were compared. Stuart, et al. (1985) reported a 9.7% reduction of resting heart rates for his CAD subjects after four months of training ($p < .005$). Ehsani, et al. (1981) showed resting heart rates to be decreased ($p < .01$) after 12 months of aerobic training, but Ogawa and associates (1981) failed to demonstrate any significant changes at an alpha level of .05. Despite the inconsistencies reported above, resting bradycardia remains to be a rather consistent physiological finding (Froelicher, 1983). Lack of uniformity of training/testing protocols could influence the findings (Froelicher, 1983).

Submaximum Heart Rate

The differences in changes for submaximum exercise heart rates reported between the experimental subgroups (–3.6%, hypertensives and –4.3%, non-hypertensives) and the control subgroups (+.3%, hypertensives and –.1%, non-hypertensives) were considerable, with the same pattern as in resting heart rate. The t-test for independent groups showed the difference between the experimental and control groups to be statistically significant ($p < .003$). Two-way ANOVAS within each group showed no interaction between hypertension and exercise ($p > .003$). Thus, there were no statistically significant differences between hypertensive and non-hypertensive subgroups within each main group when their pre-test and post-test scores were compared. The results of this study are consistent with findings by Ogawa, et al. (1981), Froelicher (1984), McGhee, et al. (1984), and Pratt and associates (Pratt, et al., 1981). Submaximum exercise bradycardia is a consistent finding within the literature. According to Froelicher (1983), if a training program does not produce a decrease in submaximum exercise heart rate, then it is lacking in the proper intensity and duration, or the subject's extensive CAD is an impediment to adaptation. A decrease in submaximum exercise heart rate is of great consequence to CAD patients. Submaximum exercise heart rate is highly correlated with MV02 (May & Nagle, 1984). A reduction in submaximum exercise heart rate brings about a reduction in MV02 and thus decreases the stress (demand) on the heart and thus delays angina symptoms. This delay in angina allows the subjects to exercise for a longer period of time (Ehsani, 1987).

Resting Mean Blood Pressure

The resting mean blood pressure was reduced in both of the experimental subgroups (–2.6%, hypertensives and –3.1%, non-hypertensives). The control groups showed almost no changes (a gain in the non-hypertensives [+.9%] and a reduction in the hypertensives [–.2%]). The *t*-test for independent groups showed the difference between the experimental and control groups to be statistically significant ($p < .003$). Two-way ANOVAS within each group showed no interaction between hypertension and exercise ($p > .003$). Thus, there were no statistically significant differences between hypertensive and non-hypertensive subgroups within each main group when their pre-test and post-test scores were compared. It is difficult to quantitatively compare the results of the present study with others in this regard. Other researchers examined both components of blood pressure (i.e., the systolic and diastolic), rather than the mean figure to reflect blood pressure readings used in the current study. Ogawa, et al. (1981) reported a decrease in resting systolic pressure, but it was not significant. Diastolic pressure was not reported. Stuart and associates (1985) reported a significant decrease for systolic pressure ($p < .02$) with no such result for the diastolic pressure. Boyer and Kasch (1970) reported significant decreases for both systolic and diastolic pressures. The apparent inconsistencies could be explained if we realized that the diastolic readings are more reflective of the total peripheral blood pressure picture (Froelicher, 1983). Therefore, it is less vulnerable to an external stimuli such as exercise training.

Submaximum Exercise Mean Blood Pressure

Both experimental subgroups showed reductions in their submaximum mean blood pressures (–4.57% for the EXHT, and –3.09% for the EXNT), while the controls showed increases (1.04% for the CHT, and .69% for the CNT). The t-test for independent groups showed the difference between the experimental and control groups to be statistically significant ($p < .003$). Two-way ANOVAS within each group showed no interaction between hypertension and exercise ($p > .003$). Thus, there were no statistically significant differences between hypertensive and non-hypertensive subgroups within each main group when their pre-test and post-test scores were compared. Again it is hard to quantitatively compare these findings with others, as was the case with resting blood pressures. However, a reduction in submaximum exercise blood pressures has not been consistently reported. Lack of uniformity between research protocols, the absence of control groups, and the very complexity of the human blood pressure mechanism, both in terms of basic physiology and pathophysiology, contribute to difficulties in ascertaining the ability of aerobic exercise training to reduce blood pressure readings. Ogawa, et al. (1981) showed the systolic pressure to decrease from a mean of 147.9 ($p<.05$), with no report on the diastolic pressure. Ehsani, et al. (1985) showed statistically significant reductions for both systolic and diastolic pressures at submaximum exercise ($p < .001$). His strong showings could be attributed to the higher than usual intensity levels (i.e., 70-90% of maximum heart rate) utilized in his training program that lasted a full year. Stuart, et al. (1985) showed no significant changes for the systolic readings, but did so for the diastolic readings. It is usual for systolic pressures to show more

propensity for change than diastolic. However, Stuart and associates reported a less stringent alpha level to determine significance for the diastolic (i.e., .03) than used for the systolic (i.e., .001). Since diastolic blood pressure is reflective of the body's total vascular resistance to blood flow and thus more critical to the vascular system than the systolic pressure (Kannel, 1990), Stuart and associates used a less stringent alpha level, so that diastolic pressure could show a measure of statistical significance.

Submaximum Exercise Rate Pressure Product

Submaximum RPP, the last of the variables under examination in this study, did decrease in both experimental subgroups, and the decrease was of considerable magnitude (–8.20% for the EXHT, and –6.96% for the EXNT) when compared with the controls. The control groups showed the opposite effect and increased their RPP values by relatively small amounts (.79% for the CHT, and .91% for the CNT). The *t*-test for independent groups showed the difference between the experimental and control groups to be statistically significant ($p < .003$). Two-way ANOVAS within each group showed no interaction between hypertension and exercise ($p > .003$). Thus, there were no statistically significant differences between hypertensive and non-hypertensive subgroups within each main group when their pre-test and post-test scores were compared. A reduction of RPP after training is consistent with other research cited (Clausen, 1970; Clausen & Trap-Jensen, 1974; Debacker, 1974; May & Nagle, 1984; Ogawa, et al., 1981;). Effort angina always appears at the same RPP values in CAD patients (Ehsani, 1987), therefore, lowering RPP can delay the onset of angina pains, and consequently allow patients to exercise for a longer period of time. This

is a common objective within the medical community, and lowering RPP constitutes the quantitative explanation for increased exercise tolerance in CAD patients who undergo exercise training as part of the total rehabilitation program (Froelicher, 1983).

Discussion of the Hypotheses

This study had two hypotheses. The first stated that statistically significant physiological improvements on all six dependent variables would be obtained after a nine month exercise training program. The results from the t-tests comparing the differences in means between the experimental and control groups (regardless of hypertension) revealed statistically significant differences for all variables. The first hypothesis was accepted. The second hypothesis stated that there would be no differences between hypertensives and non-hypertensives when pre-test and post-test scores are compared on all six dependent variables. The two 2-way ANOVAS accounted for possible contrasts between hypertensive and non-hypertensive subgroups within the experimental and control groups. The resultant F values indicated a lack of interaction to exist between the two main effects of hypertension and exercise on all six dependent variables within both main groups. Thus, the second hypothesis was also accepted. Based on the above inferential data, the author contends that the participation of CAD subjects in structured exercise training programs similar to the YMCA program brings about beneficial physiological changes, and that these changes could be realized regardless of hypertension as a factor.

CHAPTER V

Summary, Conclusions and Recommendations

Summary

The purpose of this retrospective study was to determine the effects of a 9-month aerobic exercise program performed three days a week for 35 minutes a day at intensities of 60-85% of symptom-limited maximum heart rates, on changes in weight, resting and submaximum heart rates, resting and submaximum mean blood pressures, and submaximum RPP. The subjects forming the experimental group were 50 adult male CAD patients who were enrolled in the YMCA Cardiac Therapy Rehabilitation Program between the years of 1975 and 1985. They were divided equally into two subgroups of hypertensives and non-hypertensives. They were matched by 50 non-exercising CAD patients, forming the control group. This group was also equally divided into two subgroups of hypertensives and non-hypertensives.

Descriptive statistics clearly demonstrated decreases for all the dependent variables within the experimental group when the means for pre-tests and post-tests were compared. Results of the *t*-tests for independent groups yielded statistically significant differences between the experimental and control groups for all six variables, with the experimentals showing weight reduction and improvements on cardiovascular parameters both at rest and at submaximum exercise. The two 2-way ANOVAS indicated there was no interaction between the main effects of exercise and hypertension within each main group. Therefore, given the characteristics of these subjects and the training program,

hypertension is not an impediment to the attainment of cardiovascular benefits by CAD patients.

Conclusions

Since all dependent variables were significantly decreased after training for the experimental subgroups (both EXHT and EXNT), with no such results for the control subgroups, and since the differences between the experimental and control groups were statistically significant, it can be concluded that the changes in all mentioned dependent physiological variables could be attained after a 9 month jog-walk exercise training program. The lack of interaction between the two main effects of hypertension and exercise within each main group indicates that, given the characteristics of this study's subjects, study design, and training program, hypertension seems not to be an influential factor in the results obtained.

The results need to be viewed with caution, however, since the subjects were under constant influence of therapeutic drugs. Although research has clearly demonstrated that these medications do not interfere with training per se, they do, however, attenuate the response (McGhee, et al., 1984).

The descriptive results reported for the control subgroups showed these subjects show little change (relative to the experimental subgroups) from pre-test to post-test across all dependent variables. The medical therapy they received within that nine month period (medicine, nutritional counseling, and behavioral modification) from their personal physicians, can be assumed to have kept their disease state in check, with no

progression of the underlying pathological condition. May and Nagle (1984) used lack of change in sub-maximum RPP as evidence of no progression of CAD. As to why the control patients were not in any exercise program, the author speculates that their total health and level of CAD and hypertension served as a contraindication to participation in a structured exercise training program. Other factors, such as the cost to participate in YMCA-like programs, the convenience of not participating, and the lack of family support to participate in an exercise program, could be speculated to have some influence as well.

Recommendations

The following recommendations are suggested for researchers interested in training studies having pathophysiological ramifications.

1. The precise classification of subjects in terms of the magnitude of their CAD and severity of hypertension should be known. The author did not know, for example, if these subjects had one vessel, two vessel, or three vessel blockages in their coronary systems. The author did not know if their myocardial infarctions (MI) were anterior or posterior in relation to the anatomical orientation of their hearts. The author did not know the exact kind or amount of medication they were taking. All these factors, independently or in conjunction with one another, can produce unmistakable fundamental differences in one's response to a training program (Froelicher, 1983).

2. Dietary intakes in terms of the total caloric intake and composition of food should be controlled and differences accounted for. Diet over a length

of time could exert influences in one's response to training, especially within CAD subjects (Froelicher, 1983).

3. An account of the socio-economic status of subjects should be made. This is in the spirit of human psychology which contends that man is a multi-dimensional animal, and his reactions and responses both in terms of intellect and physiology, are governed by the totality of his environment. For example, white-collar workers may respond differently to an exercise training program of a considerable duration than might blue-color workers since they operate from totally different stress levels (Froelicher, 1983).

Based on the positive results of this study, which was in agreement with the literature cited, it is recommended that persons with coronary artery disease engage in a structured long-term exercise program to develop beneficial training effects within their cardiovascular systems. Exercise practitioners should be sensitive to the employment of proper exercise principles that could ensure the utilization of proper exercise intensity, duration, frequency and mode, if they are to impart beneficial changes on their subjects. Sedentary adults above the age of 40 should consult their physicians before participating in exercise programs.

REFERENCES

Bamrah, V., Ryan, P. & Ptacin, M. (1985). Value of exercise electrocardiography to predict additional jeopardized myocardial regions remote from site of previous MI. Clinical Cardiology, 8(2), 391-398.

Ben-Ari, E. (1986). Benefits of long-term physical training in patients after coronary artery bypass crafting- a 58 month follow-up and comparison with a non-trained group. Journal of Cardiopulmonary Rehabilitation, 6, 165-170.

Boyer, T. & Kasch. F. (1970). Exercise therapy in hypertensive men. Journal of American Medical Association, 211, 1668-1671.

Bruce, R. (1971). Exercise testing of patients with coronary heart disease. Annals of Clinical Research, 3, 323-332.

Clausen, J. (1976). Circulatory adjustments to dynamic exercise and the effects of physical training in normal subjects and patients with coronary artery disease. Progress in Cardiovascular Disease, 18, 459-495.

Clausen, J. & Trap-Jensen, J. (1970). Effects of training on the distribution of cardiac output in patients with coronary artery disease. Circulation, 42, 611-624.

Clausen, J. & Trap-Jensen, J. (1976). Heart rate and arterial blood pressure during exercise in patients with angina pectoris, effects of training and of nitroglycerin. Circulation, 53, 436-442.

Coote, J. & Hilton, S. (1971). The reflex nature of the pressor response to muscular exercise. Journal of Physiology (London), 215, 789-804.

Davis, J. & Convertino, V. (1975). A comparison of heart rate methods for predicting endurance training intensity. Medicine and Science in Sports, 7, 295-298.

Debacker, G. (1974). The influence of rehabilitation on the physical performance after M.I. Acta Cardiology (Brux), 29, 427-440.

Ehsani, A. (1978). Rapid changes in left ventricular dimensions and mass in response to physical conditioning and deconditioning. American Journal of Cardiology, 42, 52-56.

Ehsani, A. (1987). Cardiovascular adaptation to endurance exercise training in ischemic heart disease. Exercise and Sports Science Review, 87(15), 53-66.

Ehsani, A., Gregory, W., Heath, D., Hageberg, J., Sobel, B., & Hollowzy, J. (1981). The effects of 12 months of intense exercise training on ischemic ST-segment depression in patients with CAD. Circulation, 64, 1111-1123.

Fox, E., Bartels, R., Billings, C., O'Brien, R., Basom, R., & Mathews, D. (1975). Frequency and duration of interval training programs and changes in aerobic power. Journal of Applied Physiology, 38, 481-484.

Fox, E. & Mathews, D. (1981). The Physiological Basis of Physical Education and Athletics (3rd ed.). Chicago: Saunders College Publishing.

Frick, M. & Katila, M. (1968). Hemodynamic consequences of physical training after M.I., Circulation, 37, 192-202.

Froelicher, V. (1983). Exercise and the Heart (2nd ed.). Chicago: Yearbook Medical Publishers, Inc.

Froelicher, V. (1984). A randomized trial of exercise training in patients with coronary heart disease. Journal of American Medical Association, 252, 1291-1297.

Gary, F. & Berra, K. (1981). YMCA Cardiac Therapy. National Health Enchantment Program of the National Council of the Young Men's Christian Association of the U.S.A. Chicago: Raven Press.

Glass, G. & Stanley, J. (1970). Statistical Methods in Education and Psychology. New Jersey: Prentice-Hall, Inc.

Gobel, F. (1978). The rate-pressure product as an index of myocardial oxygen consumption during exercise in patients with angina pectoris. Circulation, 57, 549-556.

Hollowzy, J. (1971). Cardiac growth and respiratory enzyme levels in male rats subjected to a running program. American Journal of Physiology, 220, 1238-1241.

Hollowzy, J. (1975). Adaptation of skeletal muscle to endurance training. Medicine and Science in Sports, 7, 155-164.

Hollowzy, J. (1976). Adaptations of muscular tissue to training. Progress in Cardiovascular Disease, 18, 445-458.

Jablon, S. (1966). National Cancer Institute Monogram, 19, 445-465.

Kannel, W. (1990). Hypertension and the Risk of Cardiovascular Disease in, Hypertension, Pathophysiology, Diagnosis, and Management. New York: Raven Press.

Kitamura, K., Jorgensen, C. & Gobel, F. (1972). Hemodynamic correlates of oxygen consumption during upright exercise. Journal of Applied Physiology, 32, 516-522.

Lassen, N. & Larsen, O. (1970). Effects of training on the circulation in ischemic muscle tissue. In Larsen, O. & Malmborg, R., Editors. Coronary Heart Disease and Physical Fitness. Baltimore: University Park Press.

May, G., Nagle, F. (1984). Change in rate-pressure product with physical training of individuals with CAD. Physical Therapy, 64, 1345-1352.

McArdale, W. (1986). Exercise Physiology (2nd ed.). Philadelphia: Lea & Febiger.

McGhee, J., Siconolfi, S. & Bouchard, P. (1984). Propranolol therapy does not prevent an exercise training response in patients with CHD. Journal Cardiac Rehabilitation, 4, 445-449.

Ogawa, T. Vyden, J., Rose, H., Kanazawa, M., Yoshihiko, S., & Swan, H. (1981). Peripheral circulatory changes after physical conditioning in CAD patients. Journal of Cardiopulmonary Research, 1(4), 268-275.

Pattengale, P. & Hollowzy, J. (1967). Augmentation of skeletal muscle myoglobin by a program of treadmill running. American Journal of Physiology, 213, 783-785.

Patterson, D., Shephard, R. & Cunningham, D. (1979). Effects of physical training on cardiovascular function following M.I. Journal Applied Physiology, 47, 482-486.

Pick, R., Johnson, P. & Glick, G. (1974). Deleterious effects of hypertension on the development of aortic and coronary atherosclerosis in stumptaic macaques on an atherogenic diet. Circulation Research, 35, 472-482.

Pollock, M., Dimmick, J., Miller, H., Kendrick, Z. & Linnerud, A. (1975). Effects of mode of training on cardiovascular function and body composition of adult men. Medicine and Science in Sports, 7, 139-145.

Pratt, C., Welton, D., Squires, W., Kirby, T., Hartung, H., & Miller R. (1981). Demonstration of training effect during chronic beta-adrenergic blockade in patients with CAD. Circulation, 64(6), 1125-1129.

Priest, J., Oei, T., & Moorehead, W. (1982). Exercise-induced changes in common laboratory tests. American Journal of Clinical Pathology, 77(3), 285-289.

Rassmusen, B. (1975). Pulmonary ventilation, blood gases, and blood ph After training of the arm or the legs. Journal of Applied Physiology, 38, 250-256.

Report of the Third Joint National Committee on the Detection, Evaluation and Treatment of High Blood Pressure. (1984). Archives of International Medicine, 5, 144-169.

Sarnoff, S. (1958). Hemodynamic determinants of oxygen consumption of the heart with specific reference to the tension-time index. American Journal of Physiology, 192, 148-156.

Sonnenblick, E., Ross, J. & Braunwald, F. (1968). Oxygen consumption of the heart, newer concepts of it's multifactorial determinants. American Journal of Cardiology, 22, 328-336.

Stuart, R., Sanker, K. & Lundstorm, R. (1985). Does exercise training alter maximal oxygen uptake in CAD during long term beta-adrenergic blockade? Journal of Cardiopulmonary Rehabilitation, 5, 410-414.

Tortora, G. & Anagnostakos, N. (1981). Principles of Anatomy & Physiology (3rd ed.). New York: Harper & Row Publishers.

Vanhees, L., Fagard, R. & Amery, A. (1982). Influence of beta-adrenergic blockade on effects of physical training in patients with ischemic heart disease. British Heart Journal, 48, 33-38.

Wittie, J. (1980). The American Heart Association Handbook. New York: E.P. Dutton.

APPENDIX A

RONALD A. FREEMAN, M.D., INC.

2023 Forest Ave., Suite 3
San Jose, California 95128

Internal Medicine
Non-invasive cardiology
Cardiac Rehab. Consultant

Area Code 408
Telephone:
298-0433

San Jose State University
Graduate Studies Division
Human Subjects Review Committee

March 13, 1986
Re: Mahmoud Sadri

To Whom It May Concern;

I am the Medical Director of the YMCA cardiac rehab program at the central YMCA. I have had several discussions with Mr. Mahmoud Sadri regarding his research thesis. I have advised Mr. Sadri that I will give him the data from patients participating in the YMCA cardiac rehabilitation program as well as patients from my private practice. This information will be kept in the strictest of confidence and all patients will remain anonymous. I see no need to get a signed consent from each patient since the data will be pulled from the patients files and will not have any bearing on the patients medical treatment of further evaluations. In fact, it would be difficult in some cases to get signed releases since the data covered will be from the years 1978 through 1985. Many of these patients are no longer with the YMCA cardiac rehab program.

If you require any further comments regarding this matter, I would be happy to respond.

Sincerely;

Ronald A. Freeman, M.D.

YMCArdi Therapy Exercise Test Worksheet

Name _____ Date _____ YCT # _____ TEMP. _____
Age _____ Time _____ Test # _____ Rel. Num. _____
Physician Supervisor _____ Test Conducted by _____ DP ckd. by _____

METS	MPH	GRADE	TIME MINS	HR	BP	DP	VEB TYPES FREQ	ST ABNORMALITY LEADS MV	ANGINA	SX, SIGS, END PTS (CIRCLE
SUPINE REST										
	2	0%	1			X				
2	2	0%	3							
4	2	7%	6							
6	3	7.5%	9							
8	3	12.5%	12							
10	3.4	14.0%	15							
12	3.4	16.0%	18							
14	3.4	22.0%	21							
16	3.8	22.0%	24							

Mean Heart Rate = ___/min. (___METS); Mean Double Product = _____(_____METS)

Total Ex. Min's _____ RECOVERY

C V EXAM

PRE post

_____

_____

Murmur (Type)_____

Click_____

Symptoms & Signs: Endpoints should be circled VEB TYPES

1. Angina
2. Dyspnea
3. Fatigue
4. Claudication
5. Hypertension
6. Hypotension
7. Cerebral Sx
8. Arrythmias
9. ST Dep. 3 mv.
10. Other _____

I Isolated (Enter Freq/Min)
B/T Bigeminy/Trigeminy
P Pairs
M Multiform
VT Ventricular Tachycardia
VF Ventricular Fibrillation

MEDICATIONS

Drug	Dose	Hrs. Pretst.