THE INFLUENCE OF PHYSICAL CONDITIONING AND DECONDITIONING UPON CARDIAC STRUCTURE OF MALES AND FEMALES

A DISSERTATION

SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS

FOR THE DEGREE OF DOCTOR OF PHILOSOPHY

IN THE GRADUATE SCHOOL OF THE

TEXAS WOMAN'S UNIVERSITY

COLLEGE OF HEALTH, PHYSICAL

EDUCATION, AND RECREATION

BY

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DENTON, TEXAS

DECEMBER 1980

DEDICATED TO

The Al-Muhailan family: my mother, my wife, my son, my brothers Abdulla and Sulaiman, and other members of my family for their help, patience, encouragement, and support in helping me to achieve my career goal.

ACKNOWLEDGMENTS

The author would like to express his gratitude and appreciation to the director of this study, Dr. Joel Rosentsweig, for his guidance, counsel, thoughtful suggestions, and understanding. Gratitude also is expressed to Dr. Bernard Rubal for his encouragement, directions, and cooperation.

A special expression of appreciation is due to the dissertation committee, Dr. Marilyn Hinson, Dr. Barbara Gench, and Dr. Carolyn Rozier for their suggestions and recommendations during the preparation of this work.

The investigator would also like to thank Mrs. Laurie Hammett for her cooperation in typing this manuscript.

The participation of the subjects is also gratefully acknowledged.

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CHAPTER I

INTRODUCTION

Echocardiography (echo) was introduced in 1953 by Elder and Hertz (Roelandt, 1977) as a medical diagnostic tool. Ten years later Feigenbaum developed a technique to examine left ventricular function (Roelandt, 1977). Since then, echo usage has spread to many fields. Because it is noninvasive, it offers an unusual opportunity for the exercise physiologist and physical educator to better understand the effects of exercise upon the heart.

No study was found in the literature that examined the influence of conditioning and deconditioning upon the cardiac dimensions and function of males and females and compared them by means of echo. Only one echo study considered deconditioning (Ehsani, Hagberg, & Hickson, 1978). Ehsani measured the adaptation of left ventricular dimension using echo in six male long distance college runners after three weeks of detraining.

In the recent past, the effects of physical conditioning upon the cardiac structure in males and females have typically been determined by means of radiographic and electrocardiagraphic methods (Astrand, 1970). More

recently, investigators (DeMaria, Newmann, Fowler, & Masun, 1977; Zeldis, Morganroth, & Rubier, 1978) have been able to demonstrate the adaptation of the intrinsic dimensions of the heart to prolonged physical conditioning by using the sensitive, reproducible technique of echo on males and females.

The influence of deconditioning upon cardiovascular function has been studied by many investigators during the past two decades (Applegate & Stull, 1969; Cureton & Phillips, 1964; Fardy, 1969; Fringer & Stull, 1974). These investigators used parameters such as heart rate, blood pressure, carotid pulse, heart sound interval, and EKG as criterion measures to seek answers to questions raised concerning the effect of long term hospitalization, medical rehabilitation "drop-outs," aerospace prolonged flights, and off-season detraining upon the cardiovascular system of individuals involved.

Purpose of the Study

The purpose of the study was to determine, by echo, the cardiac structural response to physical conditioning and deconditioning of college males and females.

Statement of the Problem

The problem of the study was to investigate the influence of 30 min of jogging at 70% of maximal heart rate for

the first two weeks, then at 80% for 8 weeks, upon the heart structural changes of male and female subjects. These results were compared with data collected from the same subjects after 10 weeks of deconditioning. The study was conducted during the spring and summer of 1980 in the Exercise Physiology Laboratory at the Texas Woman's University, Denton, Texas. The subjects were 20 volunteers, 10 males and 10 females. Their ages ranged from 19 to 31 years. A conclusion was drawn with respect to cardiac structural changes as a result of training and deconditioning.

Rationale for the Study

The effects of aerobic conditioning and deconditioning upon the structure and function of the human heart following a period of physical conditioning remain unknown. Contradictory findings have recently been presented (DeMaria et al., 1977; Wolfe, Cunningham, Rechnitzer, & Nichol, 1979; Zeldis et al., 1978) regarding the effect of endurance conditioning programs upon the heart structure. The completed research (DeMaria et al., 1977; Zeldis et al., 1978) indicated that changes in heart structure could be determined by echo after 30 to 40 min a day of jogging at 60 to 80% of maximal oxygen uptake, 3 days a week for 10 weeks.

Lack of exercise has been named as a major factor in many skeletal and cardiac diseases. Kraus and Raab (1961) stated that,

> hypokinetic diseases, which are attributable in a major or minor degree to the generally prevailing lack of exercise. These diseases, disorders, aches and pains concern chiefly the muscular and cardiovascular systems, metabolism and emotional patterns. They cause a premature reduction of earning power, early invalidism and widespread mortality from functional and degenerative cardiovascular derangements. (p. 173)

In addition, one of the major problems in the rehabilitation of the postmyocardial infraction patient is "dropping out" from the exercise program (Andrew, Goldberg, Sahn, Schy, & Wujcik, 1979). Therefore, it is important to better understand the beneficial effects of exercise upon the heart and the potential negative effects of stopping exercise.

Definitions and/or Explanations

For the purpose of clarification, the following terms are defined:

1. Ao (aorta): An echogram recording of the aortic root and valves. The transducer head should be aimed from

the fourth intercostal space (mitral valve position) to superior and medial direction toward the heart base (Roelandt, 1977).

2. Conditioning: A physical training program consisted of jogging for 2 weeks, for 30 min at 70% of the maximal heart rate found on the last minute of the first maximal treadmill performance; and then at 80% of the maximal heart rate for eight weeks. The jogging was performed every other day (seven times every two weeks) for the ten weeks.

3. Deconditioning: A ten-week period of relative rest, with no exercise or hard activity. The subject performed the regular daily activities that he/she customarily performed before the conditioning period.

4. Echocardiogram (echo): A graphic display that is obtained from the application of ultrasonic techniques (Roelandt, 1977).

5. EF (ejection fraction): A measure used to estimate the absolute volume of the left ventricle. The EF is the result of a ratio between the stroke volume (the difference between the left ventricle end-diastolic and end-systolic (volumes) and the left ventricle end-diastolic volume. Ejection Fraction (EF) (%) = $(LVIDd^3 = LVIDs^3)/LVIDd^3 \times 100$ (Roelandt, 1977).

6. LAD (left atrial dimension): A measure obtained by the ultrasound beam from the echocardiogram transducer head. It begins when the beam leaves the left ventricle and the posterior leaflet disappears from the tracing (Chang, 1976).

7. LDH (lactic dehydrogenase): A serum enzyme present in most tissues. It catalyzes the interconversion of lactic and pyruvic acids (Hunter & Critz, 1971).

8. LVIDd (left ventricular internal dimension at the end of diastole): A distance measured in millimeters between the anterior edge of the left ventricle endocardial surfaces of the interventricular wall to the left ventricular posterior wall echo, at the Q-wave onset (Sahn, DeMaria, Kisslo, & Weyman, 1978).

9. $LVIDd^3$ (left ventricular volume at the end of diastole): A measurement obtained as the cubic LVIDd (Roelandt, 1977).

10. LVIDs (left ventricular internal dimension at the end of systole): A measure in millimeters of the shortest distance between the anterior edge of the echogram of the left ventricular endocardial surface of the interventricular septum and the left ventricular posterior wall (Roelandt, 1977).

11. LVIDs^3 (left ventricular volume at the end of systole): A measure obtained as the cubic LVIDs (Roelandt, 1977).

12. PWT (left ventricular posterior wall at the end of diastole): A measure in millimeters of the distance between the anterior edges of the endocardial and pericardial echograms of the left ventricular posterior wall at the electrocardiogram Q-wave (Sahn et al., 1978).

13. SV (stroke volume): The result of LVIDd^3 minus LVIDs^3 (Roelandt, 1977).

Hypotheses of the Study

The following hypotheses were examined at the .05 level of significance:

1. There is no significant difference in the cardiac dimensions prior to conditioning, following conditioning, and following deconditioning programs, as measured by echo.

2. There is no significant difference between sexes in cardiac dimensions as measured by echo.

3. There is no significant interaction between trials and sexes in cardiac dimensions as measured by echo.

Delimitations of the Study

The delimitations for the study included the following:

1. The subjects were 24 college students, 12 males and 12 females, ranging in age from 19 to 31 years. Four subjects--two males and two females--were unable to complete this investigation, thereby reducing the number of subjects to twenty.

2. The kind, amount, and quality of the subjects' nutritional intakes were not regulated.

3. The daily activity of the subjects other than the exercise prescription was not monitored.

4. The health status of the subjects during the conditioning-deconditioning program was not specifically determined. Medical approval prior to beginning the program was obtained and medical supervision was available during the testing.

Chapter II presents the review of literature which was found pertinent to this investigation.

CHAPTER II

REVIEW OF RELATED LITERATURE

Introduction

The literature reviewed illustrated that varying adaptations of cardiac dimensions and functions occur with different periods and types of physical conditioning and deconditioning. Many different types of measurements have been made to study cardiac activity. This chapter is divided into three major categories pertinent to the study. The first category deals with the studies concerning the influence of conditioning upon the heart dimensions and functions by means of echo; the second category comprises those studies which specifically relate to deconditioning. The final category includes those studies that are related to the effects of exercise on serum enzymes. The literature reviewed is presented in ascending chronological order.

Studies of Echocardiography

Smithen, Wharton, and Sowton (1972) studied the ventricular wall maximal velocity and excursion by echo at rest and immediately following maximal erect exercise.

Eleven normal volunteers and six patients complaining of chest pain were the subjects of the study.

The eleven normal volunteers ranged in age from 20 to 48 years. Movement of the posterior wall of the ventricle was measured in a supine resting position and immediately following a maximal bicycle ergometer exercise. The maximal heart rate was increased by 10 beats per min and stopped at 160 beats per min. The Eskoline 20 Diagnostic Ultrasonoscope was used to obtain the echo measures.

Smithen and his colleagues found that immediately after the maximal erect exercise, the posterior wall velocity which was measured as the maximal slope of the wall movement from the end-diastolic position to the end-systolic position increased 159% while the posterior wall excursion which was measured as the maximal amplitude of movement anteriorly increased by 60%. The wall velocity and excursion returned to resting values several minutes after exercise. The six patients showed a linear reduction in maximal posterior wall velocity and posterior wall excursion with the increased heart rate. The investigators concluded that the alterations in the left ventricular function could be reflected by changes in the posterior wall movement and was independent of heart rate.

In 1975, Hirshleifer, Crawford, O'Rourke, and Karliner used echo to evaluate the effect of acute alterations

in heart rate and systemic arterial blood pressure due to drug treatment on left ventricular performance measurements in 25 subjects in the Cardiovascular Division of the Department of Medicine, University of California, San Diego, California. The volunteer subjects were 10 women and 15 men ranging in age from 21 to 29 years. The Picker Ultrasmoscope and a Honeywell Visicorder Oscillograph, Model 1856, were used to perform the echos on the supine or partial left lateral decubitus positioned subjects. Heart rate, systemic arterial pressure, and a baseline echo were obtained after an intravenous infusion of 5% dextrose and water. A second echo was obtained after the intravenous administration of atropine to raise the subject's heart rate as many as 30 beats per min. Phenylephrine was then administered to raise the systolic arterial pressure as much as to 40 mm Hg. When this level of systolic pressure had been maintained for at least ten minutes, a third echo was performed. The ejection fraction normalized mean rate of circumferential fiber shortening and mean posterior wall velocity were calculated from the end-diastolic dimension, end-systolic dimension, and the ejection time of the left ventricle measurements. The investigators used Student's t-test for paired data for statistical analysis.

Hirshleifer and his colleagues found a significant increase in heart rate and systemic systolic arterial pressure following the administration of atropine. This in turn increased the normalized mean rate of circumferential fiber shortening and the normalized posterior wall velocity. As the heart rate increased, the filling time decreased, which caused a significant decrease in the end-diastolic dimension. The administration of phenylephrine caused a significant systolic arterial pressure increase and a slight insignificant heart rate decrease. This, in turn, caused a significant decline in normalized mean rate of circumferential fiber shortening, in mean normalized posterior wall velocity, and in the ejection fraction. The end-diastolic dimension showed a significant increase. The investigators concluded that consideration must be given to the heart rate and systemic arterial pressure levels when performing left ventricular echo during surgical procedures in order to assess the intervention effects.

Longo, Guzman, and Triebwasser (1975) presented normal values and formulas for echo studies of adults. The information was collected from 33 studies, which were performed on adults ranging in age from 18 to 64 years. Unfortunately, these values were averaged for both sexes rather than reported independently.

The investigators reported that right ventricular internal dimension (RVID) was between 1.1 and 1.9 cm, the LVID was from 3.7 to 5.3 cm, and the LAD was from 1.8 to 3.9 cm. The left ventricular posterior wall end diastolic thickness (PWT) was from 0.6 to 1.0 cm, end-systolic thickness was from 0.8 to 1.6 cm. Mean velocity was from 3.3 to 6.7 cm per second, normalized mean velocity was from 0.8 to 1.1 per second, and mean circumferential fiber shortening was from 1.1 to 1.8 cm. The interventricular septum wall thickness was found to be between 0.7 and 1.1 cm, and velocity was from 0.8 to 1.1 cm/sec. The aortic root parameters were between 2.0 and 3.7 cm, and the aortic valve opening was found to be from 1.6 to 2.6 cm. The mean rates of cusp opening and closing movements were 36.9 and 29.3 cm/sec, respectively. The EF slope and total excursion for the mitral valve ranged from 8.0 to 14.0 cm/sec and 2.0 to 3.0 cm, respectively.

Longo and his colleagues presented ten formulas to calculate left ventricular volumes for small and large volumes, left ventricular posterior wall velocity, left ventricular mean velocity of circumferential fiber shortening, left ventricular muscle volume and mass, interventricular septal wall velocity, and left or right shunt flow in a ventricular septal defect. The investigators also listed 31 echo abbreviations.

Morganroth, Maron, Henry, and Epstein (1975) compared the left ventricular end-diastolic volume, the left ventricular mass, and the posterobasal left ventricular wall of athletes participating in isotonic exercises with those of athletes participating in isometric exercises and also with sedentary subjects at the University of Maryland during the 1974-1975 academic year. The participants were 15 swimmers, 15 long-distance runners, 12 wrestlers, and 16 sedentary subjects; 10 long-distance runners and 4 shot putters from the International Track Association, Los Angeles, California, also served as subjects. All the subjects were white males whose ages ranged from 18 to 24 years. The Aerotech Gamma Transducer and Ekoline 20A Ultrasonic Unit with a Honeywell 1856 Visicorder were utilized to obtain resting echo readings from the subjects. The ventricular septum and the posterobasal left ventricular wall were recorded by the T-scan technique. The left ventricular internal dimension at the end of distole was recorded to estimate the left ventricular end-diastolic volume, while the method of Troy, Pombo, and Rackley was used to calculate the left ventricular mass.

Morganroth and his colleagues found that the wrestlers and shot putters had a greater left ventricular mass (330 and 348 g) than did the swimmers and runners (308 and 302g), while the control subjects showed the smallest mass

(211 g). The left ventricular end-diastolic volume was significantly greater in swimmers and runners (191 and 160 ml) when compared with that of wrestlers and shot putters (110 and 122 ml). The control subjects again demonstrated the smallest volume (101 ml). The isometrically trained participants had a significantly larger posterobasal left ventricular wall (13 and 14 mm), while the isotonic activity participants showed "normal" wall thickness (equal to or below 12 mm). In conclusion, the investigators noted that, when evaluating an individual who is participating in competitive athletics, an incorrect diagnosis of heart disease may result since the cardiac dimensions of trained athletes may fall outside the range observed in individuals classified as nonathletes.

Rost, Schneider, and Stegmann (1975) compared the echo measurements of 23 patients, without regard to their illness, to those of 10 subjects under 40 years of age and 10 highly trained endurance athletes at the Cardiology Department of the University Clinic for General Medicine, Wurzburg, Germany. Rost and his coworkers used Kretz-Technik echo apparatus to measure the telediastolic volume (TDV), telesystolic volume (TSV), stroke volume (SV), output fraction (OF), left ventricular muscle mass, maximum movement of mitral value, maximum angle of the value aperture, posterior wall amount of movement (dH), and speed

of movement in all three groups. The 23 patients were also measured by the indicator-dilution method in order to validate the echo measurements. The ten highly trained athletes were measured by echo at rest as well as under 50-watt and 150-watt exertion. The 10 subjects under 40 years of age received ultrasound measurements only.

The investigators found that the movement of the posterior heart wall and the densitometrically calculated cardiac output showed a correlation of 0.85. The trained subjects had greater diastolic diameter by 1.1 cm, greater telediastolic volume by 63%, total decrease in the size of ventricular diameter by 2 mm, decrease in posterior wall total movement by 1 mm, and decrease in cardiac output at rest by 47 ml when compared with subjects who were nonathletes.

The ventricular hypertrophy in 42 professional basketball athletes was evaluated by electrocardiograph (EKG), vectorcardiograph, phonocardiograph, and echo. The measurements were compared with 10 sedentary subjects over a three-year period (1973-1976) in the study by Roeske, O'Rourke, Klein, Leopold, and Karliner (1976).

The 42 professional basketball players, who ranged in age from 21 to 31 years, were compared with 10 subjects who comprised a control group. The subjects were matched for age, sex, height, weight, and body surface area. The

electrocardiograms were recorded from 12 leads with the subject in supine resting position. Right ventricular hypertrophy was diagnosed from the EKG by Sokolow and Lyon's criteria; the left ventricular hypertrophy was diagnosed by the Romhilt-Estes "point-score" system. The vectorcardiograms (VCG) were recorded by a Hewlett-Packard Vector Programmer Model 1503. The right ventricular hypertrophy was diagnosed from the VCG by Chou's criteria, while the left ventricular hypertrophy was diagnosed by Pipberger's criteria. The indirect carotid arterial tracing was performed with a recording of the heart sounds using an Elena-Schonader Mingograph-34 with a Siemens-Elena E 117E transducer. The echos were obtained from the ten athletes and ten control subjects using a Picker Echoline instrument with a Honeywell strip chart recorder. The Feigenbaum dimension criteria were employed to measure the right ventricular end-diastolic dimension, left ventricular septal thickness, left ventricular end-diastolic dimension, and posterior left ventricular wall thickness. The investigators analyzed the data by Chi-square and made comparisons utilizing Student's t-test whenever appropriate.

Roeske and his colleagues found that 69% of the professional basketball players showed right ventricular hypertrophy by EKG and 43% by VCG. Left ventricular

hypertrophy was found by EKG in 26% and by 29% by VCG in the 42 athletes. From the phonocardiographic studies all the athletes showed a third heart sound, while 56% showed a fourth heart sound. The results of the echo study demonstrated that 50% of the athletes tested exhibited a right ventricular end-diastolic dimension of 23 mm or more while 40% showed an increased left ventricular end-diastolic dimension. The left ventricular septal thickness was equal to or greater than 14 mm and the left ventricular posterior wall thickness was increased in 60% of the athletes. When the heart dimensions of the athletes were compared with those of the control subjects, the athletes demonstrated a significantly greater LVIDd, RVIDd, PWT, and SV than did the control subjects. The investigators concluded that "physiological hypertrophy" caused the right and left ventricular enlargement, which was proposed to be a useful mechanical adaptation in the athlete.

Allen, Goldberg, Sahn, Schy, and Wojcik (1977) investigated the cardiac dimensions as measured by echo of 77 children. All were members of a swim team. Measurements were compared with the 5th and 95th percentiles from another study. The data were correlated, also, with a medical and activity history questionnaire answered by the parents of the students, and with an estimate of the championship ability and training level as determined by

the swimming coach. Of the 77 subjects, 45 were male and 32 were females; ages ranged from 5 to 17 years. The subjects swam 2 to 6 miles (2 to 4 hours) per day, 6 days a week with a mean length of participation of 27 months. The questionnaires completed by the parents concerned each child's medical history, duration of participation on the swim team, and participation on other competitive athletic teams. The ranking of championship ability by the coach ranged from 0 (poor) to 9 (excellent). Echos were performed on all subjects with a Smith Kline 20A Echocardiograph and recorded on a Honeywell 1956 ultraviolet recorder. The measurements were interpreted independently by at least two investigators.

Allen and his colleagues found that the right ventricular anterior wall, interventricular septum, left ventricular posterior wall, right ventricular cavity, aortic root dimension, and aortic intercusp separation exceeded the 95th percentile of a previous study in more than 80% of the subjects. The mean left ventricular internal dimension at diastolic and the left atrial internal dimension for swimmers was similar to the 50th percentile for normals. No significant correlation was found between the echo dimensions and the coach's estimate of championship ability. In conclusion, the investigators explained that quantitative differences existed between the echos of children who

participated in extensive physical training such as swimming and those of nonathletic children.

In 1977, DeMaria, Newmann, Fowler, and Masun (1977) studied the influence of an eleven-week physical conditioning program upon the mass and performance of the cardiac ventricles of 26 volunteers from the Sacramento Police Academy, Sacramento, California. The subjects were 15 males and 11 females and ranged in age from 20 to 34 years.

The investigators obtained echo and electrocardiogram (EKG) recordings from the subjects and carried out a maximal treadmill test prior to the eleven-week physical conditioning program. The Ekoline 20-A, Smith-Kline Instruments echograph was used to record the heart dimensions, while the EKG was recorded from twelve electrodes. The Bruce protocol treadmill test was performed on a Quinton instrument, Model 18-49C, treadmill, where the subjects ran to maximal exertion. Heart rate, blood pressure, and a maximal oxygen consumption estimate were obtained during the maximal treadmill test by the Bruce formula. The investigators used the Student's t-test for paired data to evaluate the statistical differences between the pre and post physical conditioning program data. The protocol of the physical conditioning consisted of a walk-jog-run program at 70% of predicted maximal heart rate for a duration of 1 hour, 4 days a week. The subjects

were involved in self-defense activities on other days for 1 hour.

DeMaria and his coworkers found that the eleven-week physical conditioning program influenced changes in cardiac structures. A significant increase was found in left ventricular intercavity size, muscle mass, and greater contractile pattern recorded directly by echo and indirectly by EKG. There was also an increase of the shortening fraction of the left ventricle and stroke volume due to an increase in the end-diastolic dimension and a decrease in the end-systolic dimension of the left ventricle. In conclusion, DeMaria and his colleagues found an increase of the left ventricular cavity and wall which they felt enhanced cardiac performance.

Gilbert, Nutter, Felner, Perkins, Heymsfield, and Schlant (1977) compared the cardiac dimensions and function of 20 long distance runners and 26 sedentary subjects by means of echo during the fall of 1976. The 20 long distance runners were males ranging in age from 20 to 39 years while the control group consisted of 26 sedentary males who ranged in age from 22 to 26 years. The endurance athletes were members of the Atlanta Track Club and had been engaged in training for at least one year during which they averaged 242 miles a month. The sedentary subjects did not participate in any aerobic activities such

as jogging or swimming, but were normally active in school or business.

The Bruce protocol was used to determine each subject's physical work capacity, and the Tissot Gasometer was used to measure the total expired air volume. This volume was then analyzed for carbon dioxide and oxygen content by infrared and Beckman paramagnetic instruments, respectively, to determine oxygen consumption. A .1 mm blood sample was obtained by a finger stick to measure serum lactata by the Strom method at rest, immediately after the treadmill test, and 5 min following the treadmill test. Prior to the treadmill test, a supine echo measurement was performed on all subjects using C-100 Unirad Ethoscope with 5/8 inch transducer. The Tektronix strip chart recorder, model 174, was used to obtain the M-mode heart scan recording. The echo measurements included the following items: (a) right ventricular internal diameter at end of diatole (RVIDd); (b) LVIDd, (c) LVIDs, (d) PWT, (e) IST, (f) $LVIDd^3$ and $LVIDs^3$, (g) SV, (h), EF, (i) percent shortening of left ventricular internal diameter, (j) maximal systolic posterior wall velocity, (k) mean normalized rate of left ventricular circumferential shortening, (l) left ventricular myocardial mass, (m) aortic diameter at diastole and systole, and (n) LAD.

Gilbert and his colleagues found that the long distance runners performed for a significantly longer time on the treadmill than did the sedentary subjects. The athletes also showed a significantly greater maximal oxygen uptake, PWT, $LVIDd^3$, left ventricular mass, RVIDd, internal diameter shortening percentage, and EF than did the sedentary subjects. Other diameters did not show a significant difference between the two groups. The investigators concluded that the effect of hypertrophy induced by long term training on the ventricle function is unknown.

Knapp and Brinkbus (1977) investigated the left ventricular internal dimensions at rest and following exercise in trained and untrained subjects in Heidelberg Medical University, Heidelberg, Germany. The subjects were 33 sedentary individuals (18 males and 15 females) and 31 athletes (14 males and 17 females). All were college students. Echo recordings were performed at rest and following submaximal treadmill exercise on the ventricular dimensions. Relative cross-sectional area differences (RCD) and relative velocities of Contraction (RVC) were also determined.

Knapp and Brinkbus found a significant increase in ventricular volume among the athletes. The RCD and EF showed a slight change with physical training, but RCD increased significantly after exercise among athletes. The

investigators concluded that the degree of the myocardial lesion in the left ventricular hypertrophy cases can be determined from the quantitative results of RCD and RVC without the necessity of taking the patient's ability to perform physical exercise into account.

Underwood and Schwade (1977) investigated the cardiac function of 20 world class distance runners, 8 college distance runners, and 10 non-athletes by echo or VCG, and cardiac intervals. The study was performed in the Medical City Dallas Hospital, Dallas, Texas, in 1977.

The investigators recorded still VCG loops in the frontal, left sagittal, and transverse planes using the Frank lead reference system on an Instant Vectrocardiogram-1B. A Kent Cambridge, Type 01077, recorder was used to record phonocardiograms by applying the transducer in the second right, second left, and fourth left intercostal spaces at the edge of the sternum. Heart rate, PQ interval, QRS interval, isovolumic contraction time (ICT), isovolumic relaxation time (IRT), left ventricular ejection time (LVET), left ventricular ejection time index (LVETI), pre-ejection period (PEP), pre-ejection period index (PEPI), and the ratio of PEP/LVET were all calculated for each subject. A modified Ekoline 20A ultrasonic unit and a Smith Kline Instruments, Model 0-14, transducer were used to record the echos. The measurements of LVIDd,

LVIDs, $LVIDd^3$, and $LVIDs^3$ were obtained from the left ventricle, and SV and EF were calculated. The aortic root diameter in diastole (AoRDd) and in systole (AoRDs), and the left atrial diameter in distole (LADd) and in systole (LADs) were also measured. The thickness of the left ventricular posterior wall was measured at systole (WTS) and at diastole (WTd). The method of Cooper, O'Rourke, and Karliner was used to determine the circumferential fiber shortening rate and the Troy, Pombo, and Rackley method was used to calculate the left ventricular mass.

Underwood and Schwade found that systolic time intervals were normal and nonspecific ST vectors were common in all groups. The two groups of runners demonstrated a longer isovolumic relaxation time than did the control group. The athletes also showed a right atrial hypertrophy 35% greater and a left ventricular hypertrophy 30% greater than those of the control subjects. In addition, they exhibited a significantly greater LVIDd, EDV, SV, LAD, and LV mass than did the control group. The world class athletes showed a greater aortic root diameter in systole than the college runners and the control subjects. No significant differences were found in EF, posterior wall excursion, posterior wall velocity, or mean velocity of circumferential fiber shortening among all groups. The investigators concluded that the athletes had larger

hearts, placing them at the upper "normal" border line, while a few showed abnormally large measurements.

In 1977, Zoneraich, Rhee, Zoneraich, Jordan and Appel performed a battery of noninvasive graphic techniques--i.e., echo, apexcardiogram, carotid pulse, EKG, VCG, phonocardiogram, systolic time interval and treadmill stress testing--on 12 marathon runners at The State University of New York at Sonny Brook, Jamaica, New York.

The subjects in this study were marathon runners who had completed a marathon run in less than 3 hours and had an average age of 33.8 years and nonathletic subjects. Their training level averaged 75.8 miles per week. The EKG was measured by 12 leads while the VCG were recorded at frontal, horizontal, and right sagittal planes using Hart Electronics PV-5 VCG, and a Frank lead system. A Cambridge MCIV Multichannel Recorder was used to record phonocardiograms, carotid pulses, apexcardiograms and EKG. The systolic time intervale--i.e. electromechanical systolic index (Q-SI), left ventricular ejection time index (LVETI), preejection period (PEP) and PED/LVET ratio--were also recorded on the Cambridge Multichannel MCIV. The Ekoline 20 ultrasonic unit was used to perform 8 echo measurements on the 12 marathon runners and 20 nonathletic subjects matched for age, weight, and body surface.

Zoneraich and his coworkers noted that almost all marathon runners demonstrated abnormal EKG and vector-cardiophic measures at rest. Sinus bradycardia, sinus arhythmia, and ST-segment elevation greater than 1 mm were the common abnormalities found. There was no down-slope ST depression exceeding 1 mm during the treadmill exercise test among the 12 marathon runners. The marathon runners showed significantly greater left ventricular end-diastolic and end-systolic volumes, stroke volume, posterior left ventricular wall thickness, and left ventricular mass than did the nonathletes; however, ejection fraction and percentage of internal diameter shortening did not show significant differences. The athletes also showed a significantly greater total electromechanical interval and preejection period than did the nonathletes; the left ventricular ejection time index and PEP/LVET ratio did not show a significant difference. In conclusion, the investigators explained that the left ventricle's major response to a significant load is dilation, followed by hypertrophy, accompanied by an increase in cardiac size and length, which in turn allow a greater cardiac performance for a given work load.

Ehsani, Hagberg, and Hickson (1978) examined the influence of physical conditioning and deconditioning upon the left ventricular dimension of college students by

echo. The study was performed at the Department of Preventive Medicine of Washington University, School of Medicine in Saint Louis, Missouri.

The Saint Louis University swimming team, which consisted of 7 males and 1 female whose ages ranged between 17 to 19 years, served as the training group. They trained 2 hours a day, 6 days a week, for 9 weeks after they had been inactive for 2 to 7 months. The detraining group was the cross-country team of Washington University, which consisted of 6 males aged 18 to 22 years. The detraining duration was 3 weeks and occurred after 3 months of training during which the runners had covered 10 miles a week.

The Ekoline 20 ultrasonic unit with a Cambridge strip chart recorder and a 2.25 Megahertz, 10 cm diameter transducer, was used to measure the left ventricular end-diastolic volume, left ventricular mass, stroke volume, and ejection fraction before, during, and after training for the swimmers and before, during, and after detraining for the cross-country runners. The swimmers' maximal oxygen uptake ($maxVo_2$) was obtained while the subjects were performing maximally on a bicycle ergometer by a Perkin-Elmer mass spectrometer model MGA-1100, with PDP-12 computer for immediate data analysis. A maximal treadmill performance

was used to determine the maxVo_2 uptake for the long-distance runners.

In response to 9 weeks of physical conditioning by swimming, the subjects' left ventricular end-diastolic dimension, volume and mass, and resting stroke volume index were increased significantly after the first week of training; an increase which was maintained throughout the conditioning period. The posterior wall thickness increased gradually but was not significantly different from the pretraining values until the fifth week. The maxVo_2 showed a significant increase from the pretraining value after the third week. The maxVo_2 increase was 7% by the end of the first week and continued to increase until it reached 15% at the end of the 9-week conditioning period. There was no significant change in ejection fraction and body weight.

The left ventricular end-diastolic dimension, volume and estimated mass, posterior wall thickness, and stroke volume decreased significantly following 3 weeks of detraining after 3 months of cross-country training. Following the first week of deconditioning, an 11% decrease of the maximal oxygen uptake occurred; this did not change for the remaining 2 weeks. Heart rate and total body weight increased but not significantly. The investigators explained that with endurance training the

adaptive changes in the ventricular dimension and walls occurred rapidly and was lost rapidly with cessation of the training. The unaffected ejection fraction in both cases indicated that the left ventricular mechanical performance might not be affected.

The hearts of 12 long distance runners were evaluated and compared with those of a sedentary group by means of EKG, vectorcardiographic, radiologic, and echo recording. The study was performed by Parker, Londeree, Cupp, and Dubiel (1978) in the Division of Cardiology of the University of Missouri, Columbia, Missouri.

The 12 long distance runners were all males; 6 were from the University of Missouri cross-country team and 6 belonged to the Columbia Track Club. Their ages ranged from 19 to 40 years and the maximal oxygen uptake averaged 73.7 mg/kg/min. The age matched control group consisted of 12 sedentary males, whose maximal oxygen uptake averaged 40.7 ml/kg/min. The runners averaged 10 miles a day at a speed of 6 minutes per mile.

The left ventricular hypertrophy diagnosis was determined from EKG recordings using Romhilt's criteria, and the height of R wave in lead V_1. Right ventricular hypertrophy was diagnosed from the 12-lead EKG. The Frank lead system VCG was performed to obtain the diagnosis of ventricular hypertrophy. The radiographic measurements of

the heart were obtained by X-ray films for 10 athletes and 7 sedentary individuals. The echo measurements of $LVIDd^3$, $LVIDs^3$, SV, EF, diastolic ISP/PWT, mean rate of shortening of the circumferential fibers, LV mass, and cardiac output were obtained using an Ekoline 20 ultrasonic unit.

Parker and his colleagues found that four runners showed left ventricular hypertrophy when examined by EKG and VCG; three runners had right ventricular hypertrophy diagnosed by VCG. The runners' hearts showed significant adaptations to training as determined by echo measurement. The thickness ratio of the interventricular septum to the posterior wall was not different from that of the control group.

Zeldis, Morganroth, and Rubier (1978) studied, by means of echo, the heart's structural response to prolonged dynamic conditioning in 10 female athletes from The University of Pennsylvania; they compared their findings with previous results gathered from males. The study was conducted at the hospital of The University of Pennsylvania Medical School, Philadelphia, Pennsylvania.

The subjects were 10 female athletes on the varsity field hockey team of The University of Pennsylvania and a control group of 25 age-matched, non-athletic female students from the same school. The 10 athletes had been

active in team sports for five years, and at the time of the study were actively training. Electrocardiograms were recorded with a 12-lead system while the echos were obtained by the Ekoline 20-A Smith-Kline Instruments ultrasonic unit and a Honeywell 1856 Visicorder strip recorder, using the T-scan technique. The left ventricular internal dimension, posterior wall thickness, and interventricular septum were measured at the R-wave onset by each of three investigators. The Bruce exercise protocol was used to determine maximal exercise performance in all of the athletes and in five members of the control group.

Zeldis and his coworkers found that the athletes' left ventricular mass and resting cardiac output were significantly greater than those of the non-athlete control group. No significant differences were found between the right ventricular dimensions, aortic root, or left atrium between the two groups; a high correlation (.92) was reported between the maxVo_2 and the LVIDd index, indicating agreement with results obtained in previous studies.

The investigators explained that the increases in the left ventricular dimension and the stroke volume were a result of prolonged conditioning and that these increases might be important to optimal athletic performance. The LVIDd values obtained from the subjects in this study were lower than those of males reported in

previous studies. Zeldis and his coworkers concluded that prolonged physical conditioning brought about positive adaptive changes in the female college athletes' left ventricular structures when compared with female nonathletes.

The relationship between myocardial contractility and the left ventricular end-diastolic volume in college athletes and nonathletes was studied by Crawford and Alexander (1979). The data were collected in the Exercise Physiology Laboratory and the Division of Cardiovascular Disease Echocardiography Laboratory at Howard University, Washington, D.C.

The subjects were 56 black male college students, 44 of whom were athletes (basketball, baseball, and football players), with ages ranging from 17 to 22 years. The remaining 12 subjects were nonathletes, aged 18 to 28 years. A precordial EKG and left ventricular dimensions were recorded by a Smith-Line ultrasonascope model 20A with a Honeywell recorder, model 1856. The echo measurements obtained from all subjects were LVIDd, LVIDs, LVID duration of shortening, shortening fraction of LVID, $LVIDd^3$, $LVIDs^3$, SV, EF, the cardiac index, and the normalized velocity of circumferential fiber shortening (Vcf).

The athletes were found to have greater cardiac output (+200 ml), SV, cardiac index, LVIDd, LVIDs (+10 mm), and faster shortening velocity when compared to nonathletes.

The correlation coefficient of Vcf and SV in the non-athletes was r = .94; in the athletes it was r = .93. An inverse linear relationship was found between the systolic volumes and velocities of fiber shortening in athletes and non-athletes. The investigators concluded that with large left ventricular dimension and volume in athletes, the large stroke volume was ejected by normal velocity of myocardial fibers shortening.

Crawford, White, and Amon (1979) investigated the left ventricular dimensions in diastole and systole and function by M-mode echo during 15 and 50% maximal handgrip isometric exercise and supine and upright position exercises in 23 men and 4 women. The study was conducted at the Cardiology Division, Department of Medicine, University of Texas Health Science Center in San Antonio, Texas.

The 27 subjects' ages ranged from 19 to 36 years. They had varying physical fitness levels but none was a trained athlete. The M-mode echo recordings were obtained using a Picker Echoview 80-C with an Irex Continutrace 101 recorder. The 27 subjects performed the maximal isometric handgrip exercises while in a supine position until the point of fatigue. An echo and blood pressure were obtained during the last 30 sec of each isometric effort. Ten of the 27 subjects received 1.5 mg of atropine and phenylephrine intraveneously on another day in order to raise the

blood pressure 40 mm Hg above the resting level. This was followed by echo recordings to investigate the subjects' response to the acute pressure loading. The Monark bicycle ergometer was used to test 12 of the 27 subjects in an upright position, where the subjects performed against 150 kilopond-meters (kpm), increased by 150 kpm every 3 min. The Quinton Uniwork Ergometer, Model 844, was used to test 20 of the 27 subjects who performed against 200 kpm per minute; the load was increased by 100 kpm every 3 min. The subjects' blood pressure and echos were recorded prior to the exercise and during the last 30 sec of each exercise. The end-diastolic dimension, end-systolic dimension, and ejection time of the left ventricle were obtained; then the percentage of left ventricular dimension shortening (%D) and the normalized mean rate of left ventricular dimension shortening (Vd) were calculated.

Crawford and his associates found that for the 15% and 50% of maximal hand grip exercises a significant increase in heart rate and systolic blood pressure occurred, while left ventricular dimension at diastole remained unchanged, but the percentage of left ventricular shortening and the normalized mean rate of left ventricular dimension shortening were significantly reduced. The administration of atropine and phenylephrine did not change the left

ventricular dimension at diastole but significantly increased it at systole while the %D and the Vd decreased significantly. During the twelfth minute of the supine bicycle exercise and the ninth minute of the upright bicycle exercise, heart rate, systolic blood pressure, and percentage of left ventricular dimension shortening rose significantly, while the end-diastolic dimension of the left ventricle did not change and the end-systolic dimension decreased significantly. The investigators noted that at 50% of the maximal isometric handgrip exercise the stroke volume was noted to decrease when compared with the bicycle ergometer exercises. The heart rate increase during the dynamic exercise was greater than during the isometric exercise. The blood pressure showed a greater increase during the isometric exercise than the increase in heart rate which may cause a decrease in the left ventricular performance due to the increase in afterload stress.

Grayevskaya, Goncharova, Kalugina, and Timonova (1979) investigated the cardiac dimensions of three different kinds of athletes by echo. The investigation was performed at the National Research Institute of Physical Culture in Moscow, U.S.S.R.

The 196 male athlete subjects were divided into three groups. Group I consisted of 88 endurance athletes (skiers, long distance runners, rowers), Group II included

64 athletes participating in different sport games (i.e. ice hockey, soccer, and handball) defined as speed-endurance activities that demanded maximum intensity, and Group III consisted of 44 refined technique athletes (gymnastics and figure skating). A group consisting of 31 sedentary men served as a control. The average age for members of Groups I and II was 22 years; Group II and the control group averaged 24 years of age. The left ventricular posterior wall at systole and diastole, left ventricular dimension at end of diastole, interventricular septum thickness, heart mass, and stroke volume were measured by the Aloka-80 echo apparatus.

Grayevskaya and his colleagues found that Group I subjects showed significant increases in heart wall thickness and heart mass compared to the other athletes and the control groups. The sport games athletes (Group II) showed a significant increase in left ventricular cavity expansion due to maximal work intensity which in turn demanded a substantial rise in systolic outpout for urgent blood circulation. Group III did not show significant differences from the control group when tested for heart wall and left ventricular volume.

The investigators noted that a high positive correlation existed between sport experience and heart wall thickness for beginners only; therefore, the heart hypertrophy

developed mainly during the early stages of training. They noted that endurance athletes' heart mass would be approximately 165 g and their left ventricular cavity volume 180 cm^3. Group II athletes usually illustrated a left ventricular cavity volume of 210 cm^3; if the volume is greater than this medical attention should be provided.

Ikaheimo, Palatsi, and Takkunen (1979) compared the heart dimensions of athletic sprinters, endurance runners, and sedentary subjects at the Oulu University Central Hospital in Finland. The investigators performed echo and and EKG measurements on all subjects. The subjects were 12 male endurance runners, 10 male sprinters, and 13 sedentary males; the mean ages were 29, 23.9, and 24.4 years, respectively. The echo measurements of the mitral valve, aortic root, left atrium, and the left ventricular dimensions were obtained with a Picker Echoview 80C ultrascope and a Honeywell LS-GA ultraviolet strip chart recorder. Electrocardiograms were recorded with an Elma-Schonander EKG. All measurements were obtained while the subjects were in a supine position at rest. The endurance runners had been involved in long distance running for 9 years and the sprinters for 8 years.

From the EKG recording, the investigators found that the endurance runners had a significantly larger heart size than the sprinters; the sprinters in turn, had a

significantly larger heart size than the control subjects. The echo recordings showed that the endurance runners had a significantly larger aortic root diameter and left atrial diameter than did the sprinters and the control group; the sprinters did not differ significantly from the control group in these measures. In addition, the endurance runners had a significantly greater left ventricular end-diastolic diameter (and respective volume), left ventricular outflow tract diameter, left ventricular posterior wall thickness, interventricular septal thickness, and left ventricular mass than did the sprinters and the control group. The sprinters had a significantly thicker left ventricular posterior wall and interventricular septum, a greater left ventricular end-diastolic diameter, and a greater left ventricular end-systolic diameter than did the sedentary men.

Ikaheimo and his coworkers concluded that in comparing the echo and EKG of endurance runners, sprinters, and sedentary males, the training method might have some effect on the function and dimensions of the heart. They noted that intensive sprint training would cause the left ventricle to dilate and its wall to increase in thickness, but would cause no significant effect upon the ventricle's function. On the other hand, endurance training caused dilation, better systolic emptying, and hypertrophy of the

left ventricle; all of the above might be due to the decrease of left ventricle compliance. The left atrium dilated in endurance runners, but did not change significantly in the sprinters.

Laird, Fixler, and Huffines (1979) evaluated the cardiovascular response to submaximal isometric handgrip exercise in 10 female and 22 male adolescents using echo in the Department of Pediatrics, University of Texas Health Science Center, Dallas, Texas. Echo was performed to determine the left ventricular function and systolic time intervals.

The investigators used an Ekoline 20 ultrasonoscope with Ekoline 21 strip chart recorder to determine the echo recording at rest and at the third minute of the isometric contraction. At the fourth minute the systolic time intervals were determined from the aortic valve echo. The submaximal isometric handgrip was performed at 25% of maximum voluntary contraction using a Stoelting handgrip dynamometer. Arterial blood pressure was also recorded during the isometric performance from the non-exercising arm while the subject was in supine position or at a slight left lateral decubitus position. The end-diastolic diameter and the end-systolic diameter were measured at the third minute in order to calculate stroke volume, cardiac index, shortening fraction, and mean velocity of

circumferential fiber shortening (Vcf). The systemic vascular resistance was estimated by the cardiac index and calculated mean blood pressure.

Laird and his colleagues found nonsignificant change in the left ventricular diastolic and systolic dimensions during the submaximal handgrip isometric exercise despite the significant heart rate, systolic, diastolic, and mean blood pressure elevation during exercise when compared with resting values. The stroke volume did not show a significant difference; therefore, the increase in the cardiac index was due to the increase in heart rate. There were no significant changes in the shortening fraction and mean velocity of circumferential fiber shortening; the left ventricular ejection time index, which was calculated using normal pediatric subjects data, increased significantly. The investigators concluded that the sustained submaximal isometric handgrip exercise was a safe technique for adolescents and was especially useful in left ventricular function assessments in young patients with left heart abnormalities.

The left ventricular dimension and function of well trained, moderately trained, and untrained school children were investigated by Lengyel and Gyarfas (1979) using echo. The investigation was performed at the Hungarian Institute of Cardiology in Budapest.

The subjects for the study were assigned to three groups. Group I consisted of 9 well trained swimmers who averaged 18 hours of training a week; Group II included 9 moderately trained children who performed 8.2 hours of moderate exercise a week, and Group III comprised 10 untrained subjects who exercised in school gymnastics 3 hours a week.

A resting 12-lead EKG was recorded to determine left ventricular and left atrial hypertrophy. A Picker Echoview 10 was used to measure LVIDd, LVIDd corrected for body surface area, LV diameter percent shortening (to measure LV contractility), mean of circumferential fiber shortening, interventricular septum, PWT, left ventricular mass, LA, and LA corrected for body surface area.

Lengyel and Gyarfas found that LVIDd, left ventricular diameter percent shortening, and mean percentage of circumferential fiber shortening did not significantly change. The resting heart rate was significantly lower in Group I than in Group II and lower in Group II than in Group III. The left ventricular end-diatolic index and the interventricular septal thickness were significantly higher in the well trained group when compared with the untrained group. The LA, LA index, and left ventricular mass were significantly greater in the well-trained group than in the moderately trained subjects. The posterior wall thickness

and resting heart rate exhibited the only significant differences between the moderately trained and untrained groups.

Wolfe, Cunningham, Rechnitzer, and Nichol (1979) studied the endurance training effect on the dimensions of the left ventricle in healthy males by echo. The study was performed in 1978 on 12 members of the faculty and staff at the University of Western Ontario, Canada. The echo recording was performed on the control and training groups prior to the endurance program, after 3 months of training, and again after 6 months of training to determine any changes in the left ventricular structures due to the training.

There were 12 subjects in the training group (mean age 36.8 years) and 10 in the control group (mean age 34.8 years). The members of the training group had not engaged in regular exercise during the previous 5 years; the control group included one former long-distance runner, 4 "habitual joggers," and 5 sedentary males. After the training group subjects received an echo recording, they began the training program which consisted of jogging for 10 minutes at 60% of maximal heart rate (as determined for maximal exercise performed on a bicycle ergometer) for 4 days a week. The maximal oxygen intake was predicted by a submaximal bicycle ergometer performance. The intensity

of the exercise was increased to 70% of maximal heart rate after 2 weeks, and the jogging time was increased to 30 min. a day. The members of the control group were asked to continue their "usual" activity status with no restrictions. The investigators used a Unirad, Model 100, to trace M-mode heart dimension and a Honeywell 1856 for paper recording. Three and six months after the first test, an echo, predicted maximal oxygen intake, a resting electrocardiogram, and blood pressure were obtained from all the subjects.

Wolfe and his colleagues found a significant increase in the predicted maximal oxygen uptake and a significant decrease in the resting heart rate after a training program documented by echo. Qualitatively small increases were found in the LVIDd and calculated LVEDV, but these were not significant. When the investigators compared the results with the significant left ventricular changes resulting from 11 weeks of training as documented by DeMaria, it was claimed that the greater ages of the subjects might have had an influence upon the effect of this training. In addition, the initial level of the aerobic fitness of the subjects in this study (42.4 ml/kg/min.) was higher than that of the subjects in DeMaria's study (35.5 ml/kg/min.).

Cohen, Gupta, Lichstein, and Chadda (1980) examined the cardiac dimension and function of 30 professional ballet dancers by phonocardiography, EKG, and echo. The study was conducted at the Metropolitan Opera House, Lincoln Center, New York City. The subjects were 15 male and 15 female ballet dancers. All were members of the American Ballet Theatre and ranged in age from 18 to 32 years. Eight males and seven females served as a sedentary control group.

From the standard 12-lead EKG, right and left ventricular hypertrophy were diagnosed by the Sokolow and Lyon Criteria and Simonson Criteria, respectively. The Irex System II echo was used to determine the interventriculas septal thickness, posterior wall thickness, left ventricular end-diastolic dimension, and volume and left ventricular mass measurements. The Irex System II multiparameter module and transducers were used to obtain phonocardiogram and carotid pulse recordings.

From the phonocardiographic recording, a third heart sound was found in 83% of the male and 50% of the female dancers, while a fourth heart sound was found in 42% of the male dancers only. One-fourth of the dancers demonstrated low-intensity grade murmurs. The EKG findings revealed sinus bradycardia in one-third of the men and three-fourths of the women. Only two male dancers showed

left ventricular hypertrophy and none of the subjects showed a right ventricular hypertrophy.

Significant adaptation to training was found in the dancers' interventricular septum, posterior wall thickness, left ventricular end-diastolic dimension, volume, and mass when compared to the control subjects. The male dancers demonstrated greater values of the above measurements than did the female dancers. The stroke volume and index were significantly greater among all dancers when compared to the control group, and the female dancers showed a greater shortening fraction and ejection fraction than did the sedentary females. No significant difference was found in velocity of circumferential fiber shortening and the dimensions of the left atriam, aortic root, and right ventricle. The stroke volume index for male dancers correlated significantly with work intensity, while the female dancers' left ventricular mass index significantly correlated with ballet hours and work intensity.

The investigators explained that because ballet consists of isometric and isotonic exercises, the dancers in this study demonstrated an increase in left ventricular cavity and wall thickness. Finally, it was concluded that the ballet dancers' cardiac changes were related to the intensity and amount of training. These changes were

similar to the "athletic heart" response to prolonged exercise which could be mistakenly diagnosed as abnormal.

In 1980, Longhurst, Kelly, Gonyea, and Mitchell used echo to compare the left ventricular masses and volumes of long-distance runners with those of competitive and amateur weight lifters. The study was performed at the Harry S. Moss Heart Center and Pauline and Adolph Weinberger Laboratory for Cardiopulmonary Research, Department of Internal Medicine, University of Texas Health Science Center in Dallas, Texas. The subjects consisted of 12 distance runners, 17 competitive weight lifters, 7 amateur weight lifters, and 23 males acting as an untrained control group, who were assigned to heavy and light subgroups.

The DuBois nomogram was used to estimate the subjects' body surface, while the Wilmore-Behnke method was used to calculate lean body mass. Systolic and diastolic blood pressures were measured by a standard sphygmomanometric method, while the MFE four-channel recorder, model M24-CAHA, was used to obtain the resting EKG and heart rate. The modified method of Sanne was used to estimate the maximal oxygen consumption by performance duration while the subjects were exercising on Monark bicycle ergometer.

The resting M-model echo was obtained while the subject was in 30° left lateral decubitus position using the Unirad Series C Echocardiograph. The standard and Penn

conventions methods were employed to determine the interventricular septal thickness at diastole, the left ventricular internal diameter, the posterior wall thickness, and the left ventricular mass, while the cube method was used to obtain the left ventricular end-diastolic volume.

Longhurst and his colleagues found that the competitive and amateur weight lifters body surface area and weight and lean body weight were significantly higher than those of the marathon runners and the control group. The runners showed a significantly lower resting heart rate and higher estimated maximal oxygen consumption than the weight lifters and the control group.

The interventricular septal thickness was not significantly different between the athletic groups and the control subjects, but the thickness of the posterior wall was significantly greater in the competitive weight lifters when compared with the other subjects. The marathon runners showed a greater left ventricular end-diastolic dimension and left ventricular mass when related to body weight, body surface area, or lean body mass than did other subjects. The competitive weight lifters showed significantly greater posterior wall thickness than the heavy control group, but exhibited no difference compared to the other groups.

The left ventricular enlargement and function of young and middle-age professional bicyclists were examined by echo and compared with measurements of sedentary individuals. The investigation was performed in the Department of Internal Medicine of the Kyoto University, Kyoto, Japan, by Nishimura, Yamada, and Kawa (1980).

The subjects were 60 well trained male professional bicyclists ranging in age from 20 to 49 years. They were assigned to 3 groups according to ages. Group I, aged 20 to 29 years, had been exercising for 5 years; Group II, aged 30 to 39 years, had exercised for 14 years; and Group III, aged 40 to 49 years, had exercised for 27 years. Exercise frequencies were 4, 3, and 2 hours a day, five days a week, for Groups I, II, and III, respectively. The control subjects were 35 matched-age sedentary men. The Aloka SSD90 instrument with a 2.25 MHZ transducer and an ECO-125A strip-chart recorder was used to obtain echo recordings of IVS, PWT, LVIDd, $LVIDd^3$, LVIDs, $LVIDs^3$, LV mass, aortic root, LA, EF, and SV. Nishimura and his colleagues found that the subjects in Group III showed an increase in IVS and PWT thickness, LV mass, and LA dimension and reduced EF, and mean velocity of circumferential fiber shortening (Vef) when compared with other athletes and the control group.

All athletes demonstrated significantly greater LVIDd, $LVIDd^3$, $LVIDs^3$, LV mass, SV, and LA than did subjects who were not athletes. The Vcf was reduced slightly in Groups I and II when compared to the control group; this difference might be due to lower heart rate, the investigators explained. The conclusion of the investigators was that the enlargement of the left ventricle is an important adaptation to exercise in young athletes.

Studies of Deconditioning

Cardiovascular respiratory deconditioning was investigated by means of prolonged bed rest (Birkhead, Haupt, Issekutz, & Rodahl, 1963; Birkhead, Haupt, & Myers, 1964), prolonged chair rest (Lamb, Johnson, Stevens, & Welch, 1964), and space cabin confinement (Lamb, Johnson, & Stevens, 1964). The subjects in these investigations consisted mainly of young healthy males whose activity and, for some, food intake, were controlled throughout the study period.

These studies demonstrated that the subjects' cardiorespiratory decondition was due to simple inactivity rather than the weightlessness which was created by gravity elimination. The deconditioning programs produced significant decreases in blood volume, hemoglobin, hematocrit, exercise and orthostatic tolerance, treadmill exercise

endurance, and maximal oxygen uptake; the urinary calcium excretion significantly increased. Birkhead (1963) explained that the hypodynamic humoral action caused by prolonged bed rest was the result of the reduction in arterial and venous sympathetic activity. Lamb (1964) noted that there was no significant difference between the influence of prolonged bed rest and prolonged chair rest in producing a cardiovascular decondition status.

Cureton and Phillips (1964) evaluated the effect of 8 weeks of training, 8 weeks of detraining, and 8 weeks of retraining upon the physical fitness of 6 middle-aged men. The study was performed in the Physical Fitness Research Laboratory of the University of Illinois, Urbana, Illinois.

The 6 subjects and 4 control individuals who participated in this study were "out of condition" males; they ranged in age from 28 to 47. These 6 subjects performed one hour of moderate exercise (30 min. of jogging and 30 min. of handball), 6 days a week for 8 weeks, followed by a detraining program for 8 weeks. A retraining period of 8 weeks followed the detraining period, which was similar to the first eight weeks but consisted of hard exercise. At the beginning and completion of each eight-week period, the investigators evaluated abdominal girth, body weight, surface area, and total body fat (by skinfolds and underwater weighing), step-test, mile run time, and treadmill

performance. They also evaluated oxygen uptake, heart size (by Liljestrand's equation), ECG changes, cardiovascular fitness (via Brachial Pulse Wave), serum cholesterol, and peripheral resistance.

Cureton and Phillips found a significant reduction in abdominal girth, body weight, surface area, and total fat after each of the eight weeks of training, while a significant increase occurred in these measures after the detraining period. The training session significantly improved the subjects' mile run and treadmill run times, and the step-test recovery heart rate, while the detraining period caused the return of their performance to the pre-training level. The increase in gross oxygen uptake following the 8 weeks of hard exercise showed a significantly higher value than after the first 8 weeks of moderate training. The detraining period reduced the oxygen uptake significantly. Heart size was found to decrease with training and increase with detraining. The R and T waves from the precordial EKG waves and the brachial pulse wave increased with training and decreased with detraining, while serum cholesterol and peripheral resistance were found to decrease after each training program and increase after detraining.

The influence of deconditioning for 10 weeks upon cardiac actions and heart rates of a college freshmen

basketball team and a 7 week deconditioning program upon heart rate and oxygen uptake of a high school female track team were investigated by Trigueiro (1965) and Everts (1970), respectively. Both studies were submitted as master's theses at the University of California at Santa Barbara. The subjects in these investigations were tested every 2 weeks during the deconditioning program for the recovery heart rates following a 5 min. step test and the maximal oxygen uptake from either a maximal bicycle ergometer or a treadmill performance.

In testing the cardiac functions, both investigators reported a significant decrease in physical working capacity of the subjects when tested by maximal performance on the treadmill or the bicycle ergometer. The recovery time following a step-test showed an increase. Everts noted that breathing efficiency was gradually reduced as the deconditioning period lengthened. Trigueiro claimed that the 7 week detraining program caused small but significant decreases in maximal oxygen uptake. He concluded in his study that the physiological adaptation following deconditions in college basketball players would not necessarily occur in a time span equal to that of the physical conditioning program.

Leon and Bloor (1968) conducted an investigation on the influence of daily and intermittent exercise and its

cessation upon the ventricular mass and the cross-section of the coronary and extracoronary arteries lumen. The study was performed at the Department of Cardiorespiratory Disease, Walter Reed Army Medical Center, Washington, D.C.

Young male rats (N = 125) were randomly assigned to three groups. Group I swam in a 30 gal. water tank for 1 hour daily, for 10 weeks; Group II swam 1 hour, twice a week for 10 weeks; and Group III served as a control group which did not exercise. The rats were deconditioned for 14 to 42 days after the ten-week conditioning program. A number of animals were randomly selected and weighed, heparinized, and sacrificed after 10 weeks of conditioning, 14 days of deconditioning, and 42 days of deconditioning. The ventricular dry weight was obtained after the heart was opened and left to dry for 3 to 4 hours. A planimeter and projection of constant magnification made with a camera lucida were used to determine the right and left coronary artery cross-sectional areas .5 mm from the origin. The Zeen method was used to obtain the extracoronary collateral arteries cross-sectional areas, while the capillary-fiber ratio was determined by counts of capillaries and myocardial fibers from a midportion of both ventricles, stained with hematoxylin and eosin.

After the ten week conditioning program, Group I significantly decreased in weight. A significant increase

occurred in ventricular dry weight by 117%, mean cross-sectional area of the coronary arteries by 127%, and cross-sectional area of extracoronary collateral artery area by 145% when compared to the control group. In addition, scattered zones of focal necrosis were found by histological examination in some daily exercise animals. Group II did not show significant change in ventricular dry weight or in cross-sectional area of the coronary artery, but demonstrated an increase of 192% of extracoronary collateral arteries cross-sectional areas wehn compared with the control group. The histological examination showed no myocardial lesion in Group II. Both exercising groups exhibited increased capillary-fiber ratio.

The 14 days of deconditioning produced a significant gain in body weight in Groups I and II, and a rapid decrease in ventricular dry weight and coronary artery cross-sectional area. The extracoronary collateral arteries cross-section in Group II remained significantly greater than in the control group for 24 days of deconditioning after which it returned to preexercise levels. The investigators noted that the increased vascularization, without hypertrophy of the heart, produced by exercise might be a protective value against coronary heart disease in humans.

Saltin, Blomquist, Mitchell, Johnson, Wildenthal, and Chapman (1968) designed a study to evaluate the effect of prolonged bed rest and heavy physical training upon body anthropometric and cardiorespiratory function in healthy college students. The study was performed at the Pauline and Adolph Weinberger Laboratory for Cardiovascular Research, Department of Internal Medicine, University of Texas Southwestern Medical School, Dallas, Texas.

The subjects were 5 male college students; 2 were active in college sports and 3 were considered sedentary. The ages of the subjects ranged from 19 to 21 years. The subjects were measured for anthropometric and cardio-respiratory function before and after a 20-day period of bed rest and then following a 55-day physical conditioning program. The training program consisted of running from 2.5 to 7 miles discontinuously; then, interval exercise was performed by running from 2 to 5 min., 4 to 10 times, with 3 min. of rest. A continuous training program included performance to exhaustion which lasted longer than 10 min.

The anthropometric measurements consisted of specific gravity obtained by the body density method, and the lean body mass. The Prentice method was used to determine total body water. The Bergstrom needle technique was performed to obtain muscle biopsies for elecron microscopy to determine cross-sectional area blood vessels number,

size of blood vessels and capillary basement membrane thickness. The respiratory values included timed volume, residual volume, diffusion capacity of the lung for carbon monoxide, and pulmonary capillary blood flow. The cardiovascular parameters tested were total heart volume, cardiac output, heart rate, arterial pressure, and oxygen uptake. The blood pH, oxygen pressure, carbon dioxide pressure, hematocrit hemoglobin and lactic acid, and serum cholesterol and triglyceride were also determined.

Saltin and his colleagues found that after the 20 days of bed rest, there was a significant decrease in lean body mass, total body water, intercellular fluid volume, plasma volume, red cell mass, total diffusion capacity, capillary blood volume, resting total heart volume, submaximal cardiac output and stroke volume, and maximal oxygen uptake. There was no significant change following bed rest in the quadriceps muscle biopsy parameters, lung capacity, forced vital capacity, membrane diffusion capacity for carbon monixide, peripheral resistance and A-V oxygen difference.

The 55-day training period caused no significant difference in lung volumes, membrane diffusion capacity, and submaximal cardiac output. There was significant increase in pulmonary capillary blood volume, total diffusing capacity, red cell mass, maximal oxygen uptake, stroke

volume, and total heart volumes. The investigators concluded that bed rest and physical conditioning had a direct effect on myocardial function.

The influences of 2, 4, and 6 weeks of deconditioning following 6 weeks of conditioning upon cardiovascular endurance were examined in 47 young and adult females by Applegate and Stull (1969). The investigation was performed at the Department of Physical Education of the University of Maryland, College Park, Maryland.

The 47 volunteer subjects were freshmen students at the University of Maryland. They ranged in age from 18 to 30 years. All subjects were involved in the conditioning program which consisted of two "all-out" bouts on a Crescent Monark bicycle ergometer, twice a week, for 6 weeks. The first training session was used as the pretraining test, while the twelfth and final session served as the post-training test. The cardiovascular endurance score was the work done during the two bouts in kilogram-meters. Following the 6 weeks of conditioning, the subjects were assigned randomly to three deconditioning groups for 2, 4, and 6 weeks. A third test was performed to determine the deconditioning effect after each detraining program to determine if any endurance was lost.

Applegate and Stull found that 6 weeks of physical training caused a significant improvement in cardiovascular

endurance in the 47 subjects. In comparing the post-training and post-deconditioning data, the three groups did not experience any alteration in cardiovascular endurance as a result of 2, 4, or 6 weeks of rest. The investigators explained that the amount of cardiovascular endurance gained was influenced by the training interval length. They also concluded that 2, 4, and 6 weeks of deconditioning following a 6-week period of conditioning did not cause a loss of cardiovascular endurance.

The influence of 10 weeks of soccer training followed by 5 weeks of detraining was investigated by Fardy (1969). Eleven soccer players and 5 control students from the University of Illinois served as subjects. The 11 subjects ranged in age from 19 to 21 years and were engaged in regular rigorous exercise of 20 min. of calisthenics, 30 min of soccer drills, and 80 min. of game situation practice. The training season lasted for 10 weeks. For this study, the testing was performed prior to the training season, 5 weeks after the beginning of training, 10 weeks after training, and after 5 weeks of detraining. The measurements taken in this investigation were heart rates during a 5-mile standardized work task and "all-out" bicycle ergometer time. The Q to first heart sound time, isovolumetric contraction period (ICP), and the tension period of the left ventricle (TP) were recorded on a

Sanborn 4-challel Direct-writing Poly-Viso Cardiette, Model 64. The Parkinson-Cowan, Type CD-4, and the Hans-Rudolph high speed respiratory valve were used to record and measure the peak oxygen intake and peak ventilation. The tests were administered four times; at rest, during, and at one and one-half min. after a submaximal bicycle ergometer exercise for five minutes at 5500 ft. lb./min., and again at 2, 5, 10, and 15 min. following an "all-out" bicycle ergometer exercise at a resistance of 9000 ft. lb./min.

The investigator noted that the heart rate decreased significantly for each minute following five weeks of training and increased significantly for each minute following 5 weeks of detraining. Significantly shorter ICPs and TPs were found after the 5 weeks of training, and significantly longer values were found following detraining at 2 min following the "all-out" ride. The peak oxygen intake, peak ventilation, and ergometer ride duration increased significantly after 5 weeks of training and decreased significantly following 5 weeks of detraining. The control group did not show any significant difference throughout the study. Fardy concluded that 5 weeks of detraining were sufficient to produce a reversal of improvement in cardiovascular fitness.

Teenage females physiological responses to three months of detraining following track season training were evaluated by Drinkwater and Horvath (1972). The investigation was performed at the Institute of Environmental Stress of the University of California, Santa Barbara, California.

The subjects in this study were 7 female track athletes, ranging in age from 14 to 17 years. The testing was performed during the final month of the track season and again after three months of detraining. The resting systolic and diastolic blood pressure ventilation, oxygen uptake, and sitting heart rate were measured. Blood samples were obtained prior to the maximal treadmill exercise test and 4 min. following the test to determine hemoglobin, hematocrit plasma protein, and lactate values. A maximal treadmill exercise was performed by all subjects to determine maximal oxygen uptake, maximal heart rate, and maximal ventilation. The Heath Servo-Recorder, Model EUW-20A, was used to record ventilatory volumes and respiratory rates. The Servomex A.O. 137 oxygen analyzer and the Beckman LB-1 CO_2 gas analyzer were used to analyze the expired air. The oxygen debt, oxygen pulse, and energy expenditure were also determined.

Drinkwater and Horvath found that 3 months of detraining significantly decreased maximal oxygen uptake, maximal

ventilation, oxygen pulse, energy expenditure, and oxygen debt for the 7 teenage female athletes. No significant differences were found between the post-track season training and 3 months of detraining measurements of systolic and diastolic blood pressures, lactate level, hemoglobin, hematocrit, and plasma protein values when measured before and after the 4 min post-test values. The change in the maximal heart rate was not significant. The investigators concluded that three months of detraining were sufficient to cause deterioration in cardiorespiratory fitness.

Knuttgen, Nordesjo, Ollander, and Saltin (1973) evaluated the influence of three types of interval training upon the physical working capacity of 37 young military personnel. The effect of detraining upon 23 of the same subjects followed. The study was done in the Department of Clinical Physiology, University Hospital, Uppsala, Sweden.

The subjects in this study were from the First Swedish Communications Regiment, with a mean age of 20 years. The first group consisted of 20 subjects who performed exercise for 15 sec and then rested for 15 sec, for 15 min per day, 3 days a week, for 2 months. Group II included 9 subjects who performed exercise for 3 min and rested for 3 min for 15 min a day, 3 days a week, for 2 months. The third group consisted of 8 subjects who

performed no exercise for the first month, then exercised for 3 min. and rested for 3 min., for 15 min. a day, 5 days a week for 1 month. Ten subjects from Group I, 7 from Group II, and 6 from Group III were chosen as a subgroup to be tested again 8 months following the testing program. The training consisted mainly of high intensity running, which totaled approximately 4 km per session. Each subject performed submaximal and maximal exercise on the Elema-Scholander bicycle ergometer before the training program, a month after training began, and 2 months after training began. The 23 subjects who served as the subgroup for the investigation were tested after 8 months of deconditioning. During testing, the EKG was used to determine the maximal heart rate, while the Riva-Rocci technique was used to determine the blood pressure. The expired air was analyzed for oxygen and CO_2 content in order to calculate maximal oxygen uptake. The lactate concentration was determined by a colorimetric technique from a fingertip blood sample. The method of Borg was used to determine perceived exertion.

Knuttgen and colleagues found that submaximal and maximal heart rates decreased significantly in all groups following the three training programs. Maximal oxygen uptake was also significantly higher in all subjects after the 3 training programs. The concentration of blood

lactate was lower after 1 and 2 months of training for the group which performed 15-sec. interval training but the 3-min. interval training group (Group II) showed a higher post-training blood lactate concentration than before training. After 8 months of deconditioning, all subjects in the three subgroups demonstrated physical condition decline as illustrated by lower maximal oxygen uptake and higher blood lactate concentration (similar to pre-training values). The maximal heart rate in the third subgroup returned to the initial rate which was recorded before training, but the first and second subgroups showed a significantly lower maximal heart rate than that initially recorded. The investigators concluded that the discontinuation of physical training for 8 months by a communications regiment in a military life situation was sufficient to cause the loss of a high level of fitness.

Fringer and Stull (1974) evaluated the changes in selected cardiorespiratory parameters of young and adult female subjects due to 10-week training period followed by 5- and 10-week detraining periods. The investigation was performed in the Department of Physical Education of the University of Maryland, College Park, Maryland.

The subjects in the study were 44 female college students who ranged in age from 17 to 28 years. The subjects were tested on a Crescent Monark bicycle ergometer

before the 10 weeks of training, following the training period, and following the 5 or 10 weeks of detraining. The subjects performed against a resistance of 360 kgm/min. for the first 2 min. The work load increased by 180 kgm/min. every 2 minutes until the subject was unable to continue the pedaling rate of 60 rpm. The second session was performed 2 days after the first session in which the subject warmed up by pedalling 6 min. against 360 kilometer per minute, then rested for 8 min., then continued performing against an increased resistance of 180 kgm/min for another 6 min. The test was terminated when the subject was unable to maintain pedalling at 60 revolutions per minute. The heart rate was recorded during the last 5 sec. of both sessions by a Beckman Type R Dynograph. During the intermittent work, an expired air sample was collected for the last 3 min. of work and then analyzed by a Beckman Model E-2 Oxygen Analyzer and a Beckman Model LB-1 Gas Analyzer for oxygen and carbon dioxide content. The subjects trained on the bicycle ergometer by performing a continuous "all-out" ride against an initial resistance of 360 kgm/min. which was increased by 180 kgm/min every 2 min. until exhaustion. This training regime took place twice a week for 10 weeks. After the training program the subjects were tested again, then assigned randomly to either the 5- or the 10-week deconditioning group. The

cardiorespiratory parameters measured were rest and maximal heart rate, maximal oxygen uptake, maximal pulmonary ventilation, maximal oxygen pulse, ventilation equivalent for oxygen during maximal exercise, and work performed during the "all-out" exercise test.

Fringer and Stull found that maximal heart rate and total work output during the continual work test increased significantly from pretraining to posttraining, but did not change significantly after either 5 or 10 weeks of detraining. A significant increase was found in maximal heart rate, maximal pulmonary ventilation, maximal oxygen uptake, and maximal oxygen pulse from pretraining to posttraining on the intermittent work test; the maximal ventilation equivalent for oxygen decreased significantly. No significant changes occurred in resting heart rate or body weight. When the investigators compared the pretraining and posttraining values, a significant increase in resting heart rate and maximal ventilation equivalent was found. The maximal oxygen uptake, maximal pulmonary ventilation, and maximal oxygen pulse increased significantly, while maximal heart rate and body weight did not show significant changes. When the Tukey test was used to locate the significant difference between the two deconditioning time periods, it was found that the 5-week deconditioning period held significantly higher maximal

oxygen uptake and maximal oxygen pulse, and also showed a significantly lower maximal ventilation equivalent for oxygen than did the 10-week deconditioning period. Maximal and resting heart rates, maximal pulmonary ventilation, and body weight did not produce any significant differences. The investigators concluded that the longer the detraining period for an individual, the greater the individual's loss of cardiorespiratory fitness.

Penny and Wells (1975) investigated the effect of detraining upon the heart rate, blood pressure, serum lactate levels, and cholesterol levels. The study was performed at the Middle Tennessee State University during the academic year of 1975.

The subjects were 6 physically trained senior varsity football players (3 linemen and 3 backs), and 6 sedentary college students who comprised the control group. The Monark bicycle ergometer was used at 1200 kiloponds per minute to test the subjects' performance for 6 min. immediately following the football season, and at the end of 3, 6, and 9 weeks of deconditioning. Serum lactate, cholesterol, and triglyceride levels were determined from a venous blood sample drawn 5 min. after the pedalling session. Blood pressure and rectal temperature were recorded immediately after the 6 min. of performance. The heart rate was recorded at the end of each minute of

performance. The serum cholesterol, lactate, and triglyceride levels were analyzed by the procedure of Pearson and associates.

No significant differences were found between the football players and the control group in rectal temperature, blood pressure, recovery heart rate, and serum cholesterol. The serum lactate level for the four measurements and the exercise heart rate for the initial measurement were significantly lower in the football players. The investigators postulated that the reduced heart rate at the ninth week for the athletes may have been due to the participation of these athletes in physical education activity classes.

Stremel, Convertino, Bernawer, and Greenleaf (1976) examined the cardiovascular effects of three different 14-day activity programs while the subjects were at bed rest. The study was performed at the NASA-Ames Research Center and the Human Performance Laboratory, University of California, Davis, California.

The subjects in this study were 7 volunteer college men, with a mean age of 20 years. All subjects were placed on a nutritionally balanced diet of 3000 kilocalories per day, and exercised on the Monark bicycle ergometer at 50% of their maximal oxygen uptake for 60 min. daily for the first two weeks of the 15-week study. The ambulatory control period was repeated again, but for three weeks

each between three two-week periods of restricted bed rest. During the bed rest periods, the subjects were asked to perform (a) no exercise, (b) 21% of maximal static leg extension for 1 min. and repeated every other minute using Elgin exercise tables for 30 min. in the morning and 30 min. in the afternoon, or (c) leg dynamic exercise for 30 min. in the morning and 30 min. in the afternoon at 68% of maximal oxygen uptake using the Collins ergometer at 60 rpm. The three routines were randomly selected for each subject.

Two days before, and at the end of each period of bed rest, a maximal exercise test was performed on the Collins ergometer with the subject in a supine position. This was done in order to determine his maximal heart rate, maxVo_2, and the exercise test duration. A Parkinson-Cowan high velocity meter was used to measure the expired gas volume; the Beckman E2 oxygen analyzer and the Godart Capnograph CO_2 analyzer were used to determine the contents of the expired air. The modified Evans blue dilution method was used to obtain the plasma volume before and after each period of bed rest.

The investigators found that for maximal exercise, the maxVo_2 was reduced when the post and prebed rest values were compared for the subjects who performed no exercise, static exercise, and dynamic exercise. The average maximal heart rate increased significantly for the

three bed rest regimens. A significant decrease occurred in the duration of the maximal exercise following the no exercise bed rest only but the heart rate remained unchanged following the static and dynamic exercise bed rest periods. No significant change was found in maximal ventilation for the three regimens. Plasma volume decreased significantly with no exercise and static exercise during bed rest. The investigators explained that the deconditioning process caused cardiovascular hydrostatic pressure reduction, decreased energy expenditure, reduced pressure on the bones, and actual confinement itself. They also noted that the prolonged bed rest deconditioning reduced tissue exchange and utilization of oxygen; the central nervous system stimulation on vascular reactivity caused increased atrophy of the cardiac and skeletal muscles. A decrease in plasma volume and muscular tone occurred which caused an impaired venous return. Cardiac output was reduced as a result. The investigators concluded that these central (heart) and peripheral changes, brought about by deconditioning, caused the impairment in maximal working capacity.

Miyashita, Haga, and Mizuta (1978) investigated the effect of 15 weeks of physical training followed by 6 months of detraining upon the physical working capacity of 11 sedentary Japanese middle-aged and old men. The study was

conducted at the Laboratory for Exercise Physiology and Biomechanics, Faculty of Education, University of Tokyo.

The 11 subjects' ages ranged from 35 to 54 years. Maximal oxygen uptake was determined before and after the training program, and again following detraining. A modified Balke protocol was used to test the subjects. Douglas bags were used to collect the expired gas and the Scholander microanalyzer was used to analyze it. The CO_2 rebreathing method was used to determine the cardiac output. The maximal pulmonary ventilation, respiratory frequency, tidal volume, maximal cardiac output, arteriovenous oxygen difference, maximal heart rate, stroke volume, and ventilatory equivalent for oxygen were also measured. The subjects trained on a treadmill 3 days a week by walking for 10 min. a day at a grade determined by the intensity of performance required to achieve 80% of maxVo_2. The duration of the training was 15 weeks, after which the subjects were tested on the selected parameters; they were then deconditioned for six months during which time they did not exercise but engaged only in normal, sedentary activities similar to the lifestyle they had followed before the training. The third testing period was conducted immediately following the 6-month deconditioning program.

Miyashita and associates found a significant increase in maximal oxygen uptake (11.7%) after the training program which was related to increases in maximal ventilation (15.1%), tidal volume (14.8%), maximal cardiac output (9.9%), stroke volume (7.1%), the maximal heart rate (2.7%), and performance duration on the treadmill (22.7%). Following the 6-month deconditioning period, maximal oxygen uptake decreased significantly (-7.2%) and was related to decreased in the maximal ventilation (-.13.9%), respiratory frequency (-8.0%), tidal volume (-6.8%), cardiac output (-6.9%), stroke volume (-3.9%), maximal heart rate (-3.5%), and treadmill performance duration (-13.5%). Body weight was not significantly different while the arterio-venous oxygen difference increased after the training period by 2.7% but did not change after detraining. The investigators concluded that 6 months of detraining was long enough to produce a decrease in cardiovascular function in middle-aged and old Japanese subjects.

Wyatt and Mitchell (1978) investigated the influence of physical conditioning and deconditionning upon the circumflex branch of the coronary artery and the left ventricular mass in dogs. The study was performed at the Department of Internal Medicine and Physiology at the University of Texas Southwestern Medical School, Dallas, Texas.

The investigators used 4 female and 4 male dogs for this study. The dogs were maintained in cages for 6 weeks prior to the conditioning program. The physical conditioning program consisted of performance on the treadmill at 39 to 52% of maximal heart rate for 7 dogs and at 72% for 1 dog. A speed of 4 to 8 miles per hour (mph) was used for 30 min. for the first week, then increased to 1 hour a day, 5 days a week for 12 weeks. The deconditioning period comprised rest in the cages for 6 weeks.

The measurements of the right coronary artery diameter and the myocardiac distribution and size were taken before and after the conditioning program and after the deconditioning period, while the dogs were anesthetized. The circumflex coronary artery diameter was obtained by a coronary arteriogram from a Number 8 French NIH catheter. The myocardial capillary distribution and size were determined from a biopsy sample obtained from the right ventricular septal wall. The sample was studied under an electronic microscope to determine the number and the diameter of the blood vessels, and the basement membrane widths were obtained by electron microscopy.

Wyatt and Mitchell found an increase of the circumflex coronary artery dimensions, left ventricular mass, and myocardial capillary density in the dogs following the physical conditioning. The six-week deconditioning period

caused a significant decrease in the circumflex branch of the coronary artery dimensions and myocardial capillary density. The investigators noted that regular exercise may be required to increase vascular dimensions. Regular exercise, in turn, increased the maximal circumflex artery blood flow and maintains an increase in myocardiac artery density.

Studies of Exercise Effect on Enzymes

The influence of exercise upon levels of the serum enzymes creatine phosphokinase (CPK), lactic dehydrogenase (LDH), and glutamic oralacetic transaminase (GOT) in trained and untrained subjects were studied by a number of investigators (Fowler, Chowdhury, Pearson, Gardner, & Gratton, 1962; Misner, Massey, & Williams, 1973; Sanders & Bloor, 1975). The serum enzymes from venous blood samples were obtained prior to (resting values) and immediately after maximal performance on the treadmill or bicycle ergometer. The enzymes, CPK, GOT, and LDH, exist in the liver, kidneys, pancreas, skeletal and cardiac muscles. Following heavy exercise, there is elevation of these enzymes in the blood stream, probably due to increased muscle cell membrane permeability (Fowler et al., 1962).

In a study of endurance runners (Sanders & Bloor, 1975), it was found that CPK levels were greater following

the maximal performance than prior to it, and were considered to be a sensitive index of exercise stress in well-trained individuals, while LDH levels did not change following exercise. No relationship was found between post-exercise enzymes and maximal heart rate, and rectal temperature. When the serum enzymes levels of trained subjects were compared with those of nontrained individuals, the greatest increments were found in the latter group following maximal performance. The GOT levels increased linearly with exercise intensity, while LDH did not increase until the subjects performed severe exercise.

The effect of physical conditioning on serum enzyme levels in man was investigated by Hunter and Critz (1971). The subjects were 12 males students at the University of Western Ontario, Canada; the subjects ranged in age from 20 to 27 years. Maximal oxygen uptake ($maxVo_2$), physical working capacity at a heart ratio of 150 beats per minute (PWC_{150}); and the Harvard step test were administered before the physical training program to determine the subjects' current levels of cardiorespiratory fitness. Blood samples were obtained before and after the $maxVo_2$ and the PWC_{150} to determine changes in the plasma enzymes CPK, GOT, and LDH. The purpose of the study was to determine the relationship between the changes in enzymes and training.

The physical conditioning program consisted of a 30 min. performance at a heart rate of 150 beats per minute on the bicycle ergometer, 3 days a week for 10 weeks. At the end of the ten-week training program, the three cardiorespiratory fitness tests were repeated and blood samples were obtained before the maxVo_2 and physical working capacity at 170 beats per minute (PWC_{170}) tests. The methods of Rosalki, Karmen, and Babson and Phillips were used to determine the activities of CPK, GOT, and LDH, respectively, in the plasma.

Hunter and Critz found a significant increase in the mean maximal oxygen uptake, in physical work capacity, and in the Harvard step test after the completion of the training program. They also found that the increase of the CPK, GOT, and LDH levels immediately after the maxVo_2 were significantly higher, 36%, 100%, and 9%, respectively, prior to the physical conditioning program than after it. The CPK, GOT, and LDH values dropped after conditioning to 11%, 65%, and 0%, respectively. Prior to the conditioning, the untrained subjects had an increase of plasma CPK (18.5%) and GOT (55%) after a 30 min. submaximal bicycle ergometer performance. The same subjects exhibited no change in plasma CPK and a 17.5% increase in GOT when they repeated the submaximal test after the physical training program. It was also found that a significant reduction in maximal

heart rate and maximal systolic blood pressure resulted from the physical conditioning program.

The authors explained that maximal performance produced some of the increase in hematocrit as a result of the decrease in plasma volume which, in turn, explained the prephysical conditioning increases in plasma LDH after maxVo_2 and CPK after a submaximal performance. The training appeared to be the cause of the increase of postphysical conditioning plasma CPK after the maxVo_2 test and in the GOT after the submaximal performance. Hunter and Critz emphasized that exercise intensity was the dominant factor and more important than exercise duration in provoking an increase in the plasma enzymes studied. The authors claimed that training increased the mitochondria number and size in the skeletal muscle cells. The increase in mitochondria increased adenosine triphosphate production and helped increase cell membrane integrity; therefore, an afflux of enzymes associated with exercise was reduced. Hunter and Critz concluded that the responses of the postexercise plasma CPK and GOT levels provided a useful index of the physical training, but that LDH levels did not.

Chapter III presents the procedures followed in the development of the study.

CHAPTER III

PROCEDURES FOLLOWED IN THE DEVELOPMENT OF THE STUDY

Introduction

The purpose of this investigation was to determine, by means of echocardiography (echo), the cardiac structural response to physical conditioning and deconditioning in college male and female students. The aortic root, right atrium, and left ventricular dimensions were obtained in 10 male and 10 female students prior to and following 10 weeks of physical conditioning; following a 10-week deconditioning program the dimensions were assessed once again. Other measures of conditioning and cardiac response to exercise were taken. The differences in cardiac dimensions among the three trials, the differences between sexes, and the interaction between sex and trials were examined.

Preliminary Procedures

Before definite procedures for the investigation were determined, all available sources of literature pertinent to the investigation were studied. A complete review of the literature revealed that no study duplicated the

specific design of the present study. A tentative outline was developed and revised in accordance with suggestions made by the committee members. The approved outline was presented to the Provost of the Graduate School in the form of a prospectus. Finally, permission to conduct the experiment was secured from the Human Subjects Review Committee.

Selection of Subjects

The subjects in this investigation were 10 male and 10 female volunteer college students, ranging in age from 19 to 31 years. The subjects were enrolled at the Texas Woman's University and North Texas State University, both in Denton, Texas, during the 1979-1980 academic year. No subjects had heart disease as determined by 12 lead EKG, an echo, and a medical history form. Three female and 2 male volunteers were rejected by the investigator due to abnormal EKG, blood pressure, and/or echo recording. At the beginning of the investigation subjects participated in an orientation session designed to inform them of their role in the investigation. A health questionnaire and a consent form were presented to each subject. Those who answered three or more items on the medical questionnaire positively were rejected from the study; a form with two positive answers was reviewed by the adviser of the study for dispensation.

Grouping of Subjects

The two groups in this study were 10 male and 10 female volunteer subjects. Each subject was tested three times by a voluntary maximal treadmill performance test; a blood serum enzymes test prior to and following the treadmill test; and by echo, prior to conditioning, after conditioning, and following the deconditioning program. Each subject participated, therefore, in three trials; the first of these trials was considered to be the control or baseline value for each subject. At the beginning of the conditioning program, each group was comprised of 12 subjects but with the loss of two males and two females, each group included, finally, ten subjects.

Selection and Description of Conditioning and Deconditioning Programs

The intensity, duration, and frequency of the physical conditioning program were chosen because of their effectiveness in increasing an individual's cardiac dimensions, function, and general fitness (DeMaria et al., 1978). No source was available to quantify the difference between male and female cardiac dimensions adaptation to 10 weeeks of deconditioning. Conditioning programs of many different lengths of time have been used. Ten weeks of conditioning were arbitrarily selected because it has been successfully used by other investigators (Fardy, 1969; Fringer et al.,

1974). Some investigators (Cureton et al., 1960, Ehsani et al., 1978; Fardy, 1969; Fringer et al., 1979) reported that 3 to 10 weeks of deconditioning were sufficient in duration to reduce cardiovascular fitness.

Physical Conditioning Program

The physical conditioning program in this investigation consisted of 30 min. jogging at 70% of maximal heart rate in the first two weeks, then increasing the performance to 80% of maximal heart rate in the remaining 8 weeks of the training program. The subject performed every other day, for a total of 7 times every 2 weeks. The subject was instructed to jog for 10 min., then to walk and measure the heart rate by placing the right index and middle fingers on either the carotid artery or the anterior surface of the opposite wrist (carpal tunnel). The heart rate was recorded for 30 sec. and multiplied by two. If the heart rate of the subject was higher or lower than 70% (in the first two weeks), or 80% (in the remaining eight weeks) of the maximal heart rate, the subject was advised either to decrease or increase his performance, respectively, in order to be at the exercise level prescribed. Exercise summary sheets were given to each subject to record the date, time, duration, distance of the exercise, and average exercise heart rate. In case of an injury, the subject was

provided with laboratory time to exercise on a bicycle ergometer at the same duration, intensity, and frequency as the jogging training program. During the course of the conditioning program the investigator jogged at least once with each subject in order to answer questions and maintain standardization of procedure during the exercise session.

Physical Deconditioning Program

During the ten week deconditioning program the subjects were asked to perform only the regular daily activities that they customarily performed before the conditioning period. The investigator urged the subjects to discontinue participation in strenuous informal or any type of formal exercise.

Procedures Followed in the Collection of Data

Prior to (Trial 1) and following the ten weeks of conditioning (Trial 2) and then following the ten weeks of deconditioning programs (Trial 3), each subject was measured for body weight, resting heart rate, maximal heart rate, maximal oxygen uptake ($maxVo_2$), preexertion serum enzymes, postexertion serum enzymes, and echo recordings. During each of these testing sessions quantitative data were collected on each subject according to the procedure described in the following paragraphs.

For each test, a timetable was developed to take all measurements in a relatively short time span; therefore, almost all of the maximal treadmill exercise tests were carried out in one day. A medical doctor was present during the testing sessions. The EKG and echo recordings were made on the day prior to the exercise test. Instructions explaining the procedure to be used were given to each subject before any test was carried out. The conditioning and deconditioning programs were described orally by the investigator. Two assistants, one male and one female, were trained to assist with the testing of the subjects.

The EKG to evaluate any cardiac electro-activity abnormality was performed for each subject at the Cardiovascular-Respiratory Research Laboratory of the Texas Woman's University prior to the physical conditioning program. The resting 12-lead EKG was recorded while the subject was in a supine position, using the EKG coupler (RG-11) manufactured by Beckman Instruments, Inc., Schiller Park, Illinois. All EKGs were checked by a cardiovascular physiologist for unrecognized abnormalities.

Systolic and diastolic blood pressure readings were recorded by cuff sphygmomanometer while the subject was in a sitting position. The criteria for acceptance of subjects for participation was a clear and normal echo, normal blood pressure, and normal EKG. Based on these criteria,

two female subjects were not allowed to participate in this investigation; the echo tracing of one illustrated abnormal mitral leaflet, while difficulty was encountered in obtaining a clear and complete tracing from the second. One male was rejected from the study due to his abnormally high blood pressure. Two individuals failed to meet the EKG criteria to participate; one male exhibited right ventricular hypertrophy and right axis deviation, and one female showed six premature ventricular contractions in a one-minute interval.

The resting echo test was performed on each subject at the Cardiovascular-Respiratory Research Laboratory of the Texas Woman's University. Instructions utilized included the IREX SYSTEM II which included a strip-chart recorder, a monitor oscilloscope, and a main control panel. The transducer used was the IREX echocardiographic 13 mm diameter transducer manufactured by IREX Medical Systems, Upper Saddle River, New Jersey. The subject's echo was recorded while in a supine position or in a slight-left lateral decubitus position in order to enhance good quality tracings. Four EKG electrodes were placed on the upper arms and the lower legs and then connected to the IREX SYSTEM II to record the EKG simultaneously with the echo.

The echo transducer was placed at the left edge of the sternum on the third, fourth, or fifth intercostal

space. A coupling gel was applied to the transducer head to ensure an air-free medium between it and the subjects' skin. To identify the aorta, the aorta valve, and the left atrium, the transducer head was tilted toward the patient's right shoulder until the nearly parallel moving echoes of the two aortic walls were recorded. As the transducer moved slightly inferiorly and laterally, the ultra-sound beam penetrated the chest wall, anterior heart wall (AHW), right ventricle (RV) cavity, interventricular septum (IVS), left ventricle (LV) cavity, anterior mitral value leaflet (AML), left atrium (LA) cavity, and left atrium posterior wall (LAPW). As the transducer head moved farther inferiorly and laterally, the sound beam traversed the AHW, RV cavity, IVS, LV cavity, and left ventricular posterior wall (LVPW). The recordings of clear, good quality tracing echos were made while the heart was contracting and relaxing, unobstructed by intervening lung tissues. The transducer head was kept perpendicular to the long axis of the heart throughout that recording.

The measurement of the aortic root diameter was explained by Roelandt (Roelandt, 1977) as extending from the anterior aortic wall (AAOW) leading echo to the posterior aortic wall (pAoW) echo at the end of diastole. The left atrial dimension was measured during ventricular systole. This measure was the distance between the interior edge

of the pAoW and the anterior surface of the LAPW (Roelandt, 1977). The PWT was measured in millimeters, as the distance between the anterior edges of the endocardial and pericardial echograms of the left ventricular posterior wall during the EKG Q-Wave. The interventricular septal thickness (IVS) was measured in millimeters as the distance between the inner surfaces of the right and left sides of the septum at the onset of the right and left sides of the septum at the onset of the EKG Q-Wave (Sahn et al., 1978). The LVIDd was measured as the distance between the edge of the LV endocardial surfaces of the IVS to the left ventricular posterior wall echo at the EKG Q-Wave onset, while the LVIDs was considered to be the shortest distance between the anterior edge of the echogram of the maximal contracted left ventricle (Roelandt, 1977). The left ventricular volume at the end of diastole ($LVIDd^3$) and at the end of systole ($LVIDs^3$) was obtained as the cubic LVIDd and LVIDs, respectively. The stroke volume was the difference between $LVIDd^3$ and $LVIDs^3$ while the ejection fraction was derived from the ratio between the stroke volume and the left ventricular end-diastolic volume (Gilbert et al., 1977; Roelandt, 1977). All cardiac dimension echos were measured and interpreted independently by two individuals (B. Rubal, Ph.D., Ph.D., and A. Al-Muhailani, M.S.). If the difference between their two measurements varied more than 5% a

new measurement was performed by a third qualified individual (Bonnie Hamerly, diagnostic ultrasonic technician).

A blood sample of 10 cc was drawn by a certified medican technician after the subject had been in a fasting state for at least 12 hours. This was done at the Medical Laboratories, Inc., of Denton, Texas. The subject was asked not to engage in strenuous activity or exercise for 48 hours before the blood sample was taken so that resting baseline levels of the serum enzymes CPK, LDH, and GOT could be obtained.

On the day following the echo recording, all subjects performed a maximal treadmill exercise to determine maximal oxygen uptake. The room barometric pressure and temperature in centigrade were recorded every hour during the testing day, and the room air was analyzed for oxygen percentage using the Applied Electrochemistry oxygen analyzer Model S-3A.

Calibration of the treadmill speed and elevation grade was performed at least once prior to the testing days at Trial 1, Trial 2, and Trial 3 according to the Quinton Treadmill Instruction Manual. The manual was also used to calibrate the cardiac tachometer. The oxygen and carbon dioxide analyzers were calibrated on each testing day with known percentage calibration gases according to the S-3A Oxygen Analyzer Operation and Service Manual from Applied

Electrochemistry, Inc., at Sunnyvale, California, and the Beckman Model LB-1 Medical Gas Analyzer Instruction Manual published by Spinco Division of Beckman Instruction, Inc., of Palo Alto, California.

The subject's height in inches and weight in pounds were obtained immediately before the treadmill performance test. The Health-O-Meter Scale, manufactured by Continental Scale Corporation, Chicago, Illinois, was used. These values were converted to centimeters and kilograms. The subject then was prepared for 12-lead EKG electrode placement. The five sites used were the right and left deltoid fossas, over the right and left anterior-superior of the iliac crest; and on the fourth or fifth intercostal space, anterior to the midaxillary line. The electrodes were connected to the Quinton Exercise Cardiac Monitoring System, Model 621, and the Cardio Tachometer, Model 609, which recorded the subject's heart rate and the heart's electrical activity from the V_5 configuration. The speed and elevation of the treadmill were controlled automatically by a programmed Bruce exercise testing protocol by Quinton Programmed Exercise Control, Model 643. The subject performed on an electrically-powered treadmill controlled by the Quinton Treadmill Controller. The treadmill system, tachometer, and exercise controller were

manufactured by the Quinton Instruments Company, Seattle, Washington.

The subject's resting heart rate and systolic and diastolic blood pressures were recorded following 15 min. of rest in a sitting position. The resting EKG and blood pressure readings were examined by the investigator for abnormalities such as depressed S-T segment and/or high blood pressure. The proper usage of the mouthpiece was demonstrated and was then connected to the Tissot tank by a tube 28 mm in diameter and 6 ft. in length. The proper way of getting on and off the running treadmill was also demonstrated by the investigator. The Bruce protocol was followed for the treadmill test (Bruce, 1971). The subject started the maximal treadmill exercise test by walking at 1.7 mph on 10% grade elevation. The speed and elevation were increased automatically by 0.8 mph and 2%, respectively, every 3 min. At the last 15 sec. of each three-minute stage, the subject's heart rate and EKG were recorded. The subject was asked to put the mouthpiece and nose clip on when the heart rate reached 130 beats per minute; the expired air was then collected for 1 min. and flushed out. A recording of the initial level of the Tissot 350 liter chain-compensator gasometer was then made.

The voluntary maximal treadmill exercise test was terminated following one minute of expired air collection. Collection began at a signal from the subject, indicating that he/she had reached a fatigue state. At the same time, the EKG, exercise duration, and maximal heart rate were recorded during the last 30 sec. of performance. The subject was then asked to take off the mouthpiece and nose clip and to remain on the treadmill while its speed was reduced to 1.7 mph and its elevation to 0% in order to cool down until the heart rate dropped to approximately 10 beats above the resting value. The EKG and heart rate were recorded during the last 15 sec. of every minute of recovery. After recovery the subject was asked to get off the treadmill and sit quietly while the postexertion blood pressure was recorded.

The second level of the Tissot gasometer was recorded and a sample of the expired air was drawn by a small tube for analysis. At this point, the EKG electrodes were disconnected from the subject; he/she was asked not to take a hot or cold shower for the next 5 hours, and to report to the Medical Laboratories, Inc. three hours following the termination of the maximal treadmill exercise so that a 10 cc blood sample could be drawn in order to determine the postexertion levels of CPK, LDH, and GOT. The maximal oxygen uptake calculation was made according to

the Calculations for Gas Analysis form from the manual Experiments and Demonstrations in Exercise Physiology (Rosentswieg, 1979).

Treatment of the Data

The procedures outlined below include the treatment of the data and selection of statistical techniques. Descriptive statistics were obtained for each of the variables under each condition. Two-way analysis of variance with repeated measures on three treatments was computed to determine whether any significant difference existed between the male and female echo measurements elicited by conditioning and deconditioning programs, and to determine whether any significant differences existed between the preconditioning, postconditioning, and post-deconditioning treatments. The interactions between the sexes and treatment were also examined. If significant differences were found at the .05 level, Tukey's test was applied as a subsequent test to determine where the significant differences occurred. The data were organized into appropriate tabular and graphic forms. The data were analyzed at the Computer Center of the Texas Woman's University by the Analysis of Variance and Covariance including Repeated Measures Program (BMDP2V) created at the University of California at Los Angeles.

Preparation of the Final Written Report

The written report was developed by the investigator upon the completion of the statistical treatment of the data. The report consists of a discussion of the study's implications and the conclusion of the study. Recommendations for further studies, a bibliography, and an appendix were the final sections of the report to be developed.

The analysis of the data and findings are presented in Chapter IV.

CHAPTER IV

PRESENTATION OF THE FINDINGS

In Chapter IV, the results of the statistical analysis are presented. The purpose of the present investigation was to determine, by echocardiography, the cardiac structural response to physical conditioning and deconditioning in college male and female students.

The data were treated statistically by a two-way analysis of variance with repeated measures in order to examine the differences among the three trials, between the sexes and their interactions. The Tukey test was used as the subsequent test for making mean comparisons when significant F ratios were found in the analysis of variance. These data are presented under the following headings: Descriptive Data Related to Status, Descriptive Data Related to Dependent Factors of the Study, and Statistical Analysis of Data.

Descriptive Data Related to Status

Table 1 reveals descriptive statistics regarding the age, weight, and height of the 20 subjects. The female subjects ranged in age from 19 to 31 years with a mean of 25.5 years; the male subjects ranged in age from 21

to 30 years with a mean of 26.5. The standard deviation and standard error of the mean for the females' age were found to be 1.22 and .38 years greater, respectively, than the males'. The weight of the female subjects ranged from 51.4 to 72.9 kg with a mean of 60.0 kg and a standard deviation of 6.75 kg, while the male subjects ranged in weight from 55.5 to 112.3 kg with a mean of 76.7 kg. The large standard deviation of 18.1 kg for the male subjects revealed that the subjects were heterogeneous with respect to weight. The height of the female subjects ranged from 151.3 to 170.5 cm, with a mean of 164.4 cm and a standard deviation of 6.13 cm; the male subjects showed a range of 159.0 to 188.0 cm, with a mean of 173.6 cm and standard deviation of 9.23 cm.

Descriptive Data Related to Dependent Factors of the Study

The results of the descriptive data of resting and maximal heart rates, maximal oxygen uptake, treadmill performance time, and body weight for female and male subjects during the three trials (T1, T2, and T3) are shown in Table 2. These performance measures were studied to validate the conditioning-deconditioning programs.

The mean resting heart rates for female and male subjects in T1 were 74.8 and 75.1 beats per minute (bpm),

Table 1

Descriptive Data for Ten Female and Ten Male Subjects on Age, Weight, and Height

Variable[a]	Range (min-max)	Mean	SD	SE_M
Female				
Age	19-31	25.5	4.24	1.34
Weight	51.4-72.9	60.0	6.75	2.13
Height	151.3-170.5	164.4	6.13	1.94
Male				
Age	21-30	26.5	3.03	.96
Weight	55.5-112.3	76.7	18.10	5.72
Height	159.0-188.0	173.6	9.23	2.92

[a]Age was measured in years; weight in kg; height in cm.

respectively. These decreased to 66.6 and 65.6 bpm after conditioning, then increased to 69.2 and 75.5 bpm following deconditioning. The females' mean maximal heart rates in T1 and T2 (190.8 and 187.8 bpm) were slightly higher than the males' (184.2 and 183.8 bpm). This was expected. Heart rates during T3 were very similar for both sexes. The standard deviation for the males' maximal heart rate during T1 was twice the amount of the females' which suggests that the males were heterogeneous with respect to their maximal heart rate.

The mean maxVo_2 for the females increased from 38.01 ml/kg/min. in T1 to 46.02 ml/kg/min. in T2; it then decreased to 37.77 ml/kg/min. in T3. The males' maxVo_2 was 41.59 ml/kg/min. in T1 and increased to 50.09 ml/kg/min. in T2. It then fell to 42.95 ml/kg/min. at T3. This implies that the conditioning program produced the desired results and that the deconditioning period was successful in achieving a lowering of physiological function back to the initial levels for these subjects. The treadmill performance time for female and male subjects changed in manner similar to the maxVo_2. There was an increase from T1 to T2 (8.51 to 11.09 min. for females, and 9.09 to 10.81 min. for males); there was then a decrease in T3 (9.3 min. for females, and 9.4 min. for males).

Table 2

Descriptive Data of Resting and Maximal Heart Rates, Maximal Oxygen Uptake, Treadmill Performance Time and Body Weight During Three Trials

Variable[a]	Range of Scores			Mean			S.D.			SE_M		
	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3
Female												
Resting H. R.	40 (54-94)	39 (49-88)	42 (46-88)	74.80	66.60	69.20	13.36	13.75	12.28	4.23	4.35	3.88
Max. H. R.	28 (180-200)	28 (172-200)	25 (175-200)	190.80	187.80	189.00	7.08	7.02	7.63	2.24	2.22	2.41
Max. VO_2	17.6 (28.8-46.4)	21.7 (38.7-60.4)	11 (32.6-43.6)	38.01	46.02	37.77	6.52	7.56	4.71	2.06	2.39	1.49
Treadmill Time	3.2 (7.0-10.2)	5.1 (8.2-13.3)	3.7 (8.0-11.7)	8.51	11.09	9.30	1.49	1.19	1.15	.47	.38	.36
Body Weight	21.5 (51.4-72.9)	18.1 (51.4-69.5)	22.3 (50.4-72.7)	60.04	59.25	59.72	6.75	6.12	7.06	2.14	1.94	2.23
Male												
Resting H. R.	40 (54-94)	33 (50-83)	35 (55-90)	75.10	65.60	75.50	14.10	11.38	12.95	4.46	3.60	4.09
Max. H. R.	50 (150-200)	32 (170-202)	15 (185-200)	184.20	183.80	190.10	14.62	9.54	5.30	4.63	3.02	1.68
Max. VO_2	26.6 (30.0-50.6)	12.5 (42.8-55.3)	11.6 (34.9-46.5)	41.59	50.09	42.95	6.95	5.41	3.89	2.20	1.71	1.23
Treadmill Time	5.5 (5.5-11.0)	3.7 (9.7-13.4)	2.7 (8.5-11.2)	9.09	10.81	9.42	1.16	1.29	1.57	.37	.41	.49
Body Weight	56.8 (55.5-112.3)	57.7 (53.2-110.9)	61.8 (52.3-114.1)	76.73	76.26	76.29	18.11	18.37	19.07	5.73	5.81	6.03

[a]Resting H. Rate measured by beats per minute; Max. H. R. measured by beats per minute; Max. VO_2 measured by ml/kg/min. Treadmill time measured by min.; Body weight measured by kg.

The resting and postexercise blood serum enzymes (CPK, GOT and LDH) data are presented as units per liter (U/L) in Tables 3 and 4. The resting CPK mean values were greater for the male subjects during the three trials (81.5, 66.7, and 69.5 U/L) when compared with those of the female subjects (48.4, 47.1, and 46.9 U/L). The large standard deviations and standard errors of the means for both sexes indicates they were heterogeneous and unreliable estimates of the population means with respect to resting CPK values. The postexercise CPK mean values showed a slight to moderate increase from the resting CPK values; the same relationship was maintained between sexes. Between trials the CPK values tended to increase from T1 to T2, then to decrease again for T3.

The LDH enzyme showed a large reduction at postexercise for both sexes. The mean resting value for females decreased 39.2 U/L from T1 to T2, then increased 46.5 U/L in T3. The postexercise LDH for the females decreased 20.9 U/L after conditioning, then increased 23.9 U/L after deconditioning. The male resting LDH values decreased 63.5 U/L after conditioning then increased 47.7 U/L after deconditioning. The postexercise value decreased 7.9 U/L after conditioning then increased 7.7 U/L after deconditioning. The trend of the changes in blood enzymes

Table 3

Descriptive Data of Resting, Post Exercise of Serum Enzymes of the Female Subjects During Three Trials

Variable[a]	Range of Scores			Mean			SD			SE_M		
	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3
Resting												
CPK	53 (20-78)	67 (19-86)	67 (19-86)	48.4	47.1	46.9	23.1	21.5	15.0	7.3	6.8	4.7
GOT	26 (6-32)	17 (9-26)	13 (3-21)	15.5	14.6	14.2	8.0	4.8	6.6	2.5	1.5	2.1
LDH	75 (140-215)	77 (85-162)	148 (130-278)	174.1	134.9	181.4	25.4	28.9	42.6	8.0	9.2	3.5
Post Exercise												
CPK	71 (57-128)	61 (51-112)	39 (63-102)	49.3	78.9	55.2	16.4	52.5	15.9	5.2	16.5	5.0
GOT	9 (11-20)	14 (9-23)	7 (19-26)	14.0	15.2	15.8	2.5	4.5	4.3	0.8	1.4	1.4
LDH	173 (93-266)	81 (119-200)	107 (141-248)	179.5	158.6	182.5	38.0	27.5	29.8	12.0	8.71	9.4

[a]CPK measured in Units per Liter (U/L); GOT measured in Units per Liter (U/L); LDH measured in Units per Liter (U/L).

Table 4

Descriptive Data of Resting, Post Exercise and Serum Enzymes

of the Male Subjects During Three Trials

Variable[a]	Range of Scores			Mean			SD			SE_M		
	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3
Resting												
CPK	83 (53-136)	39 (46-85)	61 (34-95)	81.5	66.7	69.5	25.5	10.9	16.7	8.1	3.5	5.3
GOT	57 (10-67)	28 (10-38)	24 (3-27)	21.8	16.9	15.8	16.4	8.2	7.2	5.2	2.5	2.3
LDH	18 (157-175)	70 (105-175)	114 (130-244)	192.7	129.2	176.9	36.2	22.0	38.0	11.5	6.9	12.1
Post Exercise												
CPK	49 (20-69)	161 (29-190)	56 (26-82)	83.4	86.7	79.4	19.1	21.6	14.5	6.0	6.8	4.6
GOT	39 (11-50)	20 (13-33)	13 (12-25)	20.2	17.5	18.4	11.5	5.8	4.7	3.6	1.8	1.5
LDH	276 (75-351)	81 (139-220)	119 (131-260)	176.0	169.1	176.8	85.7	21.7	42.9	27.1	6.9	13.6

[a]CPK measured in Units per Liter (U/L); GOT measured in Units per Liter (U/L); LDH measured in Units per Liter (U/L).

supported the underlying concept; i.e., that positive changes occur with training and the process reverses with deconditioning.

Tables 5 and 6 are a presentation of the descriptive data for the three echo measurements for female and male subjects, respectively. The mean of the aortic diameter (Ao) for the female subjects changed from 2.25 cm in T1 to 2.16 cm in T2, then to 2.15 cm in T3. In the male subjects, it increased from 2.68 cm in T1 to 2.84 cm in T2. This diameter was maintained in T3. Throughout the three trials the mean values for the left atrium diameter (LAD) were higher among the males than the females. There was a slight increase for the female subjects and a slight decrease for the male subjects after the conditioning program. There was a larger decrease following the deconditioning program for both sexes than occurred as a result of training.

In all trials the left ventricular posterior wall thickness (PWT) was greater in males than in the female subjects. Following conditioning, the mean PWT increased by .09 cm for females and by .06 cm for males; it then decreased following deconditioning by .04 cm for females and by .06 for males. The interventricular septum (IVS) thickness mean changes at T1, T2 and T3 were similar to those of the PWT; it showed an increase in thickness

Table 5

Descriptive Data of the Echocardiographic Parameters of the Female Subjects During Three Trials

Variable[a]	Range of Scores			Mean			SD			SE_M		
	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3
Ao	1.0 (1.7-2.7)	0.6 (1.9-2.5)	0.5 (2.0-2.5)	2.25	2.16	2.15	.28	.19	.18	.09	.06	.06
LAD	1.3 (2.4-3.7)	1.1 (2.6-3.7)	1.2 (2.5-3.7)	3.12	3.14	2.96	.39	.42	.44	.12	.13	.14
PWT	0.2 (0.5-0.7)	0.4 (0.6-1.0)	0.3 (0.5-0.8)	.60	.69	.65	.09	.12	.12	.03	.04	.04
IVS	0.2 (0.5-0.7)	0.4 (0.6-1.0)	0.4 (0.5-0.9)	.59	.71	.65	.09	.12	.13	.03	.04	.04
LVIDd	1.4 (4.2-5.6)	1.9 (4.1-6.0)	2.0 (4.0-6.0)	4.70	4.87	4.82	.47	.55	.58	.15	.18	.18
LVIDs	1.0 (2.7-3.7)	1.3 (2.2-3.5)	1.6 (2.7-4.3)	3.15	3.15	3.36	.37	.50	.46	.12	.16	.14
$LVIDd^3$	101.4 (74.2-175.6)	147.1 (68.9-216.0)	152 (64.0-216.0)	106.72	119.62	116.54	33.69	42.75	44.88	10.65	13.52	14.19
$LVIDs^3$	31 (19.7-50.7)	53.4 (10.6-64.0)	35.2 (19.7-54.9)	32.48	33.34	39.93	11.73	14.85	17.49	3.70	4.69	5.53
SV	83.7 (44.3-125.0)	105 (47.0-152.0)	102.3 (34.2-136.5)	74.22	86.31	76.61	24.07	30.30	31.19	7.93	9.58	9.86
EF	21.9 (56.9-78.8)	27.9 (59.6-87.5)	26.3 (50.6-76.9)	69.32	72.43	65.16	7.15	7.30	9.57	2.26	2.31	3.03

[a]Ao, LAD, PWT, IVS, LVIDd, and LVIDs are measured in cm; $LVIDd^3$ and $LVIDs^3$ are measured in cm^3; SV is measured in ml; EF is measured as a percentage.

Table 6

Descriptive Data of the Echocardiographic Parameters of the Male Subjects During Three Trials

Variable[a]	Range of Scores			Mean			SD			SE_M		
	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3
Ao	0.8 (2.3-3.1)	1.1 (2.4-3.5)	1.2 (2.1-3.3)	2.68	2.84	2.84	.23	.32	.31	.07	.10	.10
LAD	1.0 (3.0-4.0)	1.0 (3.1-4.1)	1.1 (2.8-3.9)	3.48	3.47	3.39	.41	.41	.41	.13	13	.13
PWT	.3 (0.6-0.9)	.3 (0.7-1.0)	.1 (0.7-0.8)	.75	.81	.75	.08	.09	.05	.03	.03	.02
IVS	.2 (0.7-0.9)	.3 (0.7-1.0)	.1 (0.7-0.8)	.78	.84	.74	.06	.09	.05	.02	.03	.02
LVIDd	1.3 (4.7-6.0)	1.3 (5.0-6.3)	1.8 (4.4-6.2)	5.37	5.70	5.16	.47	.38	.59	.15	.12	.19
LVIDs	1.5 (3.1-4.6)	1.3 (3.2-4.5)	2.0 (2.5-4.5)	3.81	3.93	3.62	.46	.48	.62	.15	.15	.19
$LVIDd^3$	112.2 (103.8-216.0)	125.1 (125.0-250.1)	203.1 (35.2-238.3)	158.00	187.47	142.37	40.52	36.85	50.71	12.82	11.65	16.04
$LVIDs^3$	67.5 (29.8-07.3)	55.2 (35.9-91.1)	75.5 (15.6-91.1)	57.49	63.08	49.82	20.84	21.51	24.24	6.17	6.80	7.66
SV	69.6 (66.9-136.5)	69.9 (89.1-159.0)	100.8 (58.0-158.8)	100.51	124.40	92.75	29.94	21.51	33.55	9.47	6.80	10.61
EF	32.7 (41.5-74.2)	24.4 (54.0-78.4)	26.4 (50.3-76.7)	63.67	67.02	65.45	9.29	7.14	10.52	2.94	2.26	3.33

[a]Ao, LAD, PWT, IVS, LVIDd, and LVIDs are measured in cm; $LVIDd^3$ and $LVIDs^3$ are measured in cm^3; SV is measured in ml; EF is measured as a percentage.

following conditioning of .12 cm for females and .06 cm for males and then a decrease after deconditioning of .06 cm for females, and .10 for males. The male subjects had a thicker septum under each condition measured.

The left ventricular internal dimension at diastole (LVIDd) was found to be larger in male than female subjects in all trials. There was an increase of .17 cm for females and .33 cm for males after conditioning and then a decrease for both sexes occurred after deconditioning. The male subjects showed a larger mean left ventricular internal dimension at systole (LVIDs) when compared to the same dimension for the female subjects. There was no change in the females' LVIDs after conditioning, but there was an increase after deconditioning; whereas, the males' LVIDs increased by .33 cm and decreased by .54 cm following conditioning and deconditioning, respectively. The volumes of the left ventricle at diastole and systole ($LVID^3$) were found to be greater in male subjects in all trials when compared to those of the female subjects. The $LVIDd^3$ increase after conditioning in females was greater than the decrease after deconditioning. The decrease in $LVIDd^3$ for males following deconditioning was greater than the increase following conditioning. The $LVIDs^3$ mean in females increased only by .86 cc following conditioning

and continued to increase by 6.59 cc following deconditioning; the males' $LVIDs^3$ increased by 5.59 cc after conditioning, and decreased by 13.25 after deconditioning.

The stroke volume (SV) of both sexes increased and decreased following conditioning and deconditioning, respectively. The mean values for the females' stroke volume increased after conditioning and this was greater than the decrease after deconditioning, whereas the males' stroke volume decrease after deconditioning was greater than the increase after conditioning. The ejection fraction (EF) mean value for the female subjects was greater than those of males by 5.65 and 5.41% at T1 and T2, respectively, while the males showed a greater ejection fraction than the females at T3 by 29%. The ejection fraction changes following conditioning and deconditioning were similar to those of SV.

Statistical Analysis of Data

Cardiovascular, Weight, and Serum Enzymes

Heart Rates, Performance Time, Weight and Serum Enzymes

The statistical analysis of ten week programs of conditioning and deconditioning on resting and maximal heart rates, treadmill performance time, body weight and blood serum enzymes of college females and males are presented

in Table 7. A study of Table 7 reveals there was no significant interaction between sexes and trials, but there were significant F ratios at the .05 level between males and females when total body weight, resting CPK, and post exercise CPK were compared. Resting heart rate, treadmill performance time, and the resting LDH differences were found to be significant between trials at the .05 level of significance. There were no significant differences at the .05 level, either between groups or between trials in maximal heart rate, resting GOT, postexercise GOT, postexercise LDH, CPK difference, GOT difference, and LDH difference.

MaxVo2

Table 8 represents the statistical analysis of the influence of physical conditioning and deconditioning upon the maximal oxygen uptake of 20 college females and males. It should be noted that there was no significant interaction between trials by sexes. A significant F ratio was found between trials at the .05 level. Figure 1 and Table 9 indicate that for the females maxVo_2 at T2 was significantly greater than at T1 or T3. There was no significant difference between T1 and T3. The same finding was observed for the male subjects.

Table 7

Analysis of Variance on Heart Rates, Performance Time, Body Weight and Serum Enzymes

Measure	F Ratio (Between Groups)	p	F Ratio (Between Trials)	p	F Ratio (Group X Trials Interaction)	p
Resting Heart Rate	.15	.700	4.98**	.021	.91	.411
Maximal Heart Rate	1.26	.276	1.12	.338	1.22	.308
Treadmill Performance Time	1.00	.755	21.63**	.000	.81	.455
Total Body Weight	7.33*	.014	.64	.536	.08	.922
Serum Enzymes						
Resting						
CPK	17.26*	.001	1.28	.291	.86	.431
GOT	1.13	.303	1.43	.253	.62	.545
LDH	.07	.799	21.60**	.000	1.25	.299
Post Exercise						
CPK	6.75*	.018	3.16	.055	1.63	.210
GOT	2.75	.116	.18	.834	1.14	.330
LDH	.00	.980	1.46	.246	.38	.688

*$F_{.05}(1,18) = 4.41$

**$F_{.05}(2,36) = 3.32$

Table 8

Analysis of Variance of Maximal Oxygen Uptake

SV	df	SS	MS	F	p
Between					
Group	1	274.69	274.69	3.68	.071
S (group) error	18	1342.71	74.59		
Within					
Trials	2	851.58	425.79	26.16**	.000
Trials X Groups	2	6.70	3.35	.21	.815
TxS (group) error	36	585.95	16.27		

*$F_{.05}(1,18) = 4.41$

**$F_{.05}(2,36) = 3.32$

There were no significant differences found between the female and male subjects maxVo_2 at the .05 level. Table 9, the Tukey test, indicates that there was a significant difference between maxVo_2 of females and males at T2 and T3 but not between females and males at T1. Figure 1 illustrates the mean response in maxVo_2 during conditioning and deconditioning which indicates a similar increase and decrease in both sexes.

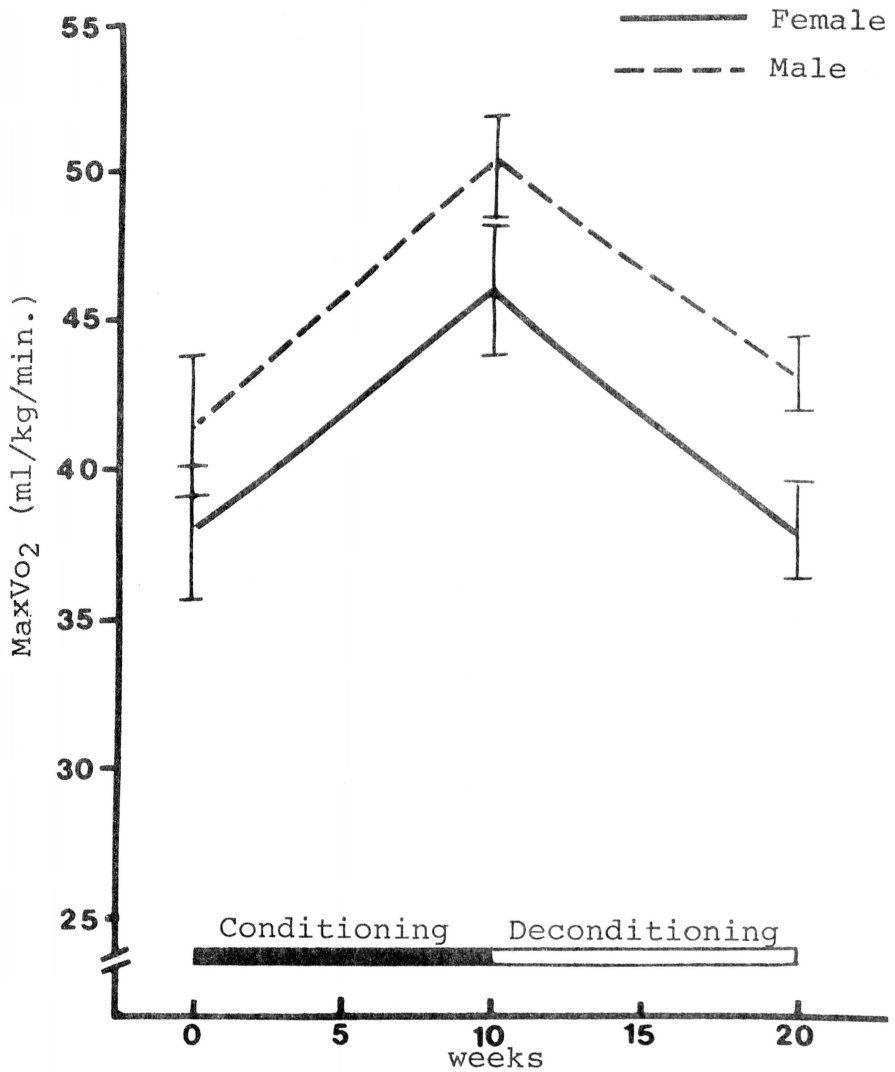

Figure 1. Maximal oxygen uptake (mean ± SE) response during conditioning and deconditioning

Table 9

Matrix of Mean Differences for Maximal Oxygen Uptake

	Females			Males		
	T1	T2	T3	T1	T2	T3
Females T1	--	8.01*	.76	3.58	12.08*	4.94*
Females T2		--	8.25*	4.43*	4.07	3.07
Females T3			--	3.82	12.32*	5.18*
Males T1				--	8.50*	1.36
Males T2					--	7.14*
Males T3						--

*$C_{.05}(6,36) \pm 4.43$

Echocardiographic Measurements

Aortic Diameter

Table 10 illustrates the statistical analysis of the changes in the aortic diameter after conditioning and deconditioning in 20 female and male college students. There was a significant interaction between sexes and trials in the aortic diameter at the .05 level. From Tables 10 and 11 it was noted that there was a significant difference between the female subjects' means and the male subjects' means in all trials. The F ratio for the difference between

trials did not meet the criterion to reach a significant level.

Table 10

Analysis of Variance on the Aortic Diameter

SV	df	SS	MS	F	p
Between					
Group	1	5.400	5.400	39.68*	.000
S (group) error	18	2.449	.136		
Within					
Trials	2	.014	.007	.23	.797
Trials X Groups	2	.217	.108	3.46*	.042
TxS (group) error	36	1.128	.031		

*$F_{.05}(1,18) = 4.41$

**$F_{.05}(2,36) = 3.32$

There was a significant difference observed in Table 10 between the females and the males in the aortic diameter at the .05 level. The reader should interpret this with caution since the aortic diameter for the males increased (T1 to T2) but not significantly. The aortic diameter for the females decreased from T1 to T2 but again, not significantly. This is illustrated in Figure 2. Both males and females remained unchanged from T2 to T3.

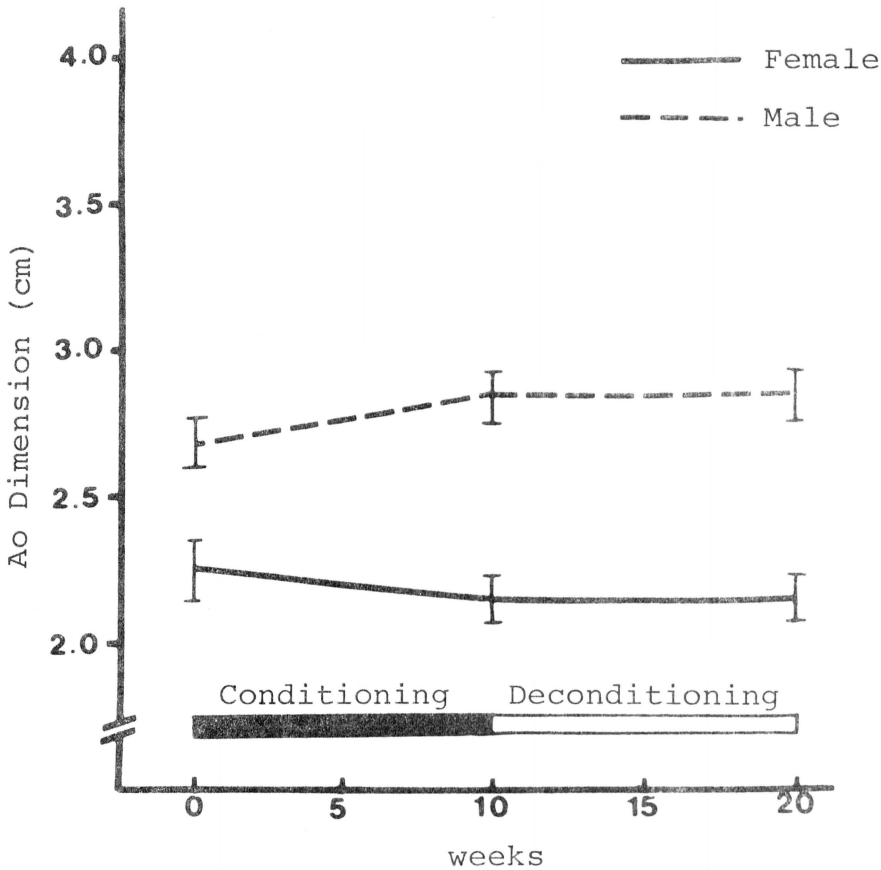

Figure 2. Aortic dimension (mean ± SE) response during conditioning and deconditioning

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significant difference between the left atrial diameter of female and male subjects at the .05 level. The finding was supported by the significant differences between the female's and male's LAD means at trials 1 and 3 as shown in Table 13. Figure 3 illustrates the LAD means in the three trials for both sexes. The graph shows similar changes for both sexes during the three trials.

Table 12

Analysis of Variance on Left Atrial Diameter

SV	df	SS	MS	F	p
Between					
Group	1	2.090	2.090	5.74*	.028
S (group) error	18	6.553	.364		
Within					
Trials	2	.217	.108	1.49	.238
Trials X Groups	2	.026	.013	.18	.835
TxS (group) error	36	2.616	.072		

*$F_{.05}(1,18) = 4.41$

**$F_{.05}(2,36) = 3.32$

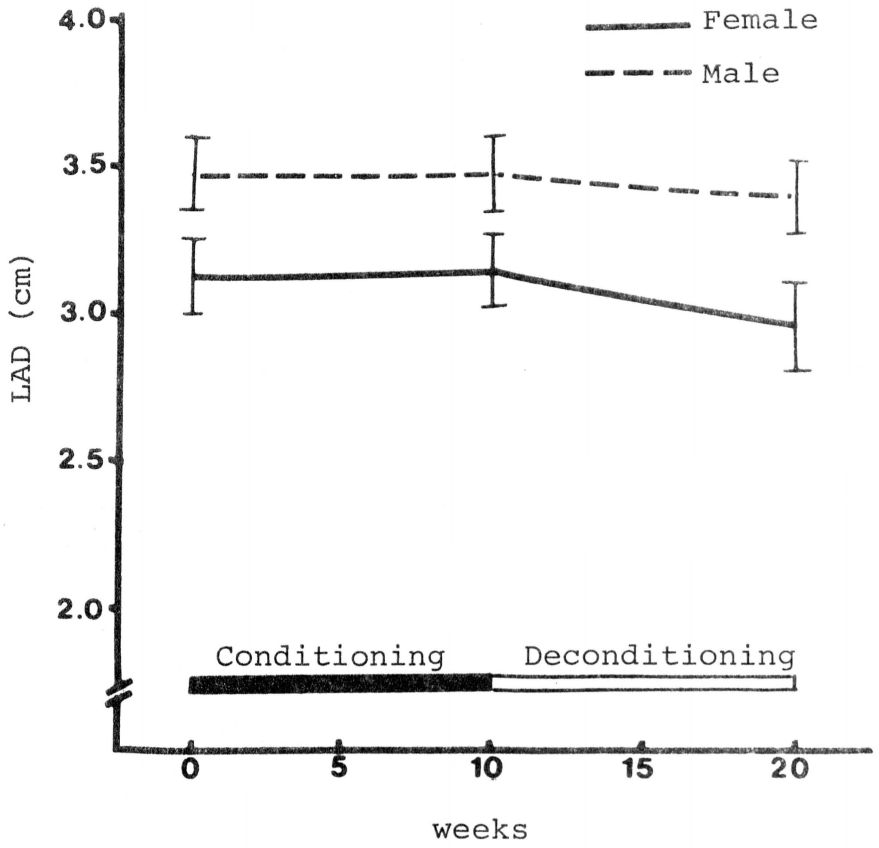


Figure 3. Left atrial diameter (mean ± SE) response during conditioning and deconditioning

Table 13

Matrix of Mean Differences for

Left Atrial Diameter

	Females			Males		
	T1	T2	T3	T4	T5	T6
Females T1	--	.02	.16	.36*	.35	.27
Females T2		--	.18	.34	.33	.25
Females T3			--	.52*	.51*	.43*
Males T1				--	.01	.09
Males T2					--	.08
Males T3						--

*$\underline{C}_{.05}(6,36) = \pm .36$.

Posterior Wall Thickness

Table 14 illustrates the analysis of variance of PWT for female and male subjects during the three trials. There was no significant interaction noted between sexes and trials when PWT was tested, where there was a significant difference between trials at the .05 level. From Table 15, which illustrates the PWT mean differences, significant differences between the paired means may be found. Although a significant difference between the sexes may not be determined by the paired means the compound effect of sex on the PWT exists. When the means were collapsed over

sexes a significant difference was found between the first and second trials. This supported an increase as a result of conditioning. No other merged means indicated significance. Figure 4 graphically depicts this finding. Using conservative degrees of freedom the F ratio remains significant, therefore, no test of compound symmetry was performed. Table 15 illustrates where the significance actually occurred when comparing the sexes at each trial condition.

Table 14

Analysis of Variance of Posterior

Wall Thickness

SV	df	SS	MS	F	p
Between					
Group	1	2.2817	2.2817	12.93*	.002
S (group) error	18	3.1767	.1765		
Within					
Trials	2	.5833	.2917	5.99**	.006
Trials X Groups	2	.0633	.0317	.65	.528
TxS (group) error	36	1.7533	.0487		

*$F_{.05}(1,18) = 4.41$

**$F_{.05}(2,36) = 3.32$

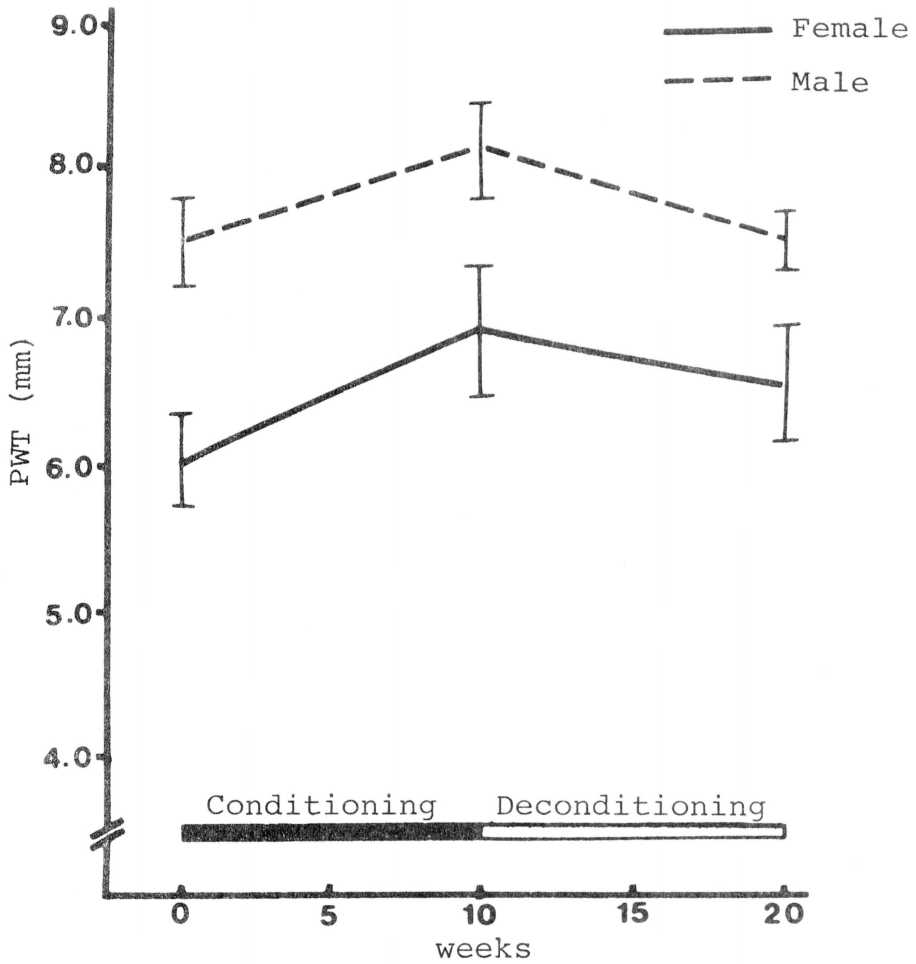


Figure 4. Posterior wall thickness (mean ± SE) response during conditioning and deconditioning

Table 15

Matrix of Mean Differences for

Posterior Wall Thickness

	Females			Males		
	T1	T2	T3	T1	T2	T3
Females T1	--	.09	.05	.05	.21	.15
Females T2		--	.04	.06	.12	.06
Females T3			--	.10	.16	.10
Males T1				--	.06	.00
Males T2					--	.06
Males T3						--

*$C_{.05}(6,36) = \pm .297$

Interventricular Septum Thickness

Table 16 illustrates the analysis of variance on the IVS changes due to conditioning and deconditioning in 20 college female and male students. Table 16 shows no significant interaction between sexes and trials when IVS was measured. The F ratio between trials, however, was found to be significant at the .05 level. On Table 17 the mean differences are not found. The significant F ratio indicates that the differences exist, most likely in a compound manner, between the sexes.

Table 16

Analysis of Variance on Interventricular Septum Thickness

SV	df	SS	MS	F	p
Between					
Group	1	2.535	2.5350	15.75*	.001
S (group) error	18	2.890	.1609		
Within					
Trials	2	.760	.3800	6.54**	.004
Trials X Group	2	.280	.1400	2.41	.104
TxS (group) error	36	2.090	.0580		

*$F_{.05}(1,18) = 4.41$

**$F_{.05}(2,36) = 3.32$

The males' interventricular septal thickness was significantly greater than the females' as illustrated in Table 16, and in Figure 5. The graphs were found to change in similar fashion for the two sexes; as they increased in T2 then decreased in T3 with the variation in the degree of change for each sex the crucial factor. The Tukey test for paired means failed to indicate where the significance exists. When the means were merged for the sexes a significant difference was found for the initial and post training conditions. Using conservative degrees of

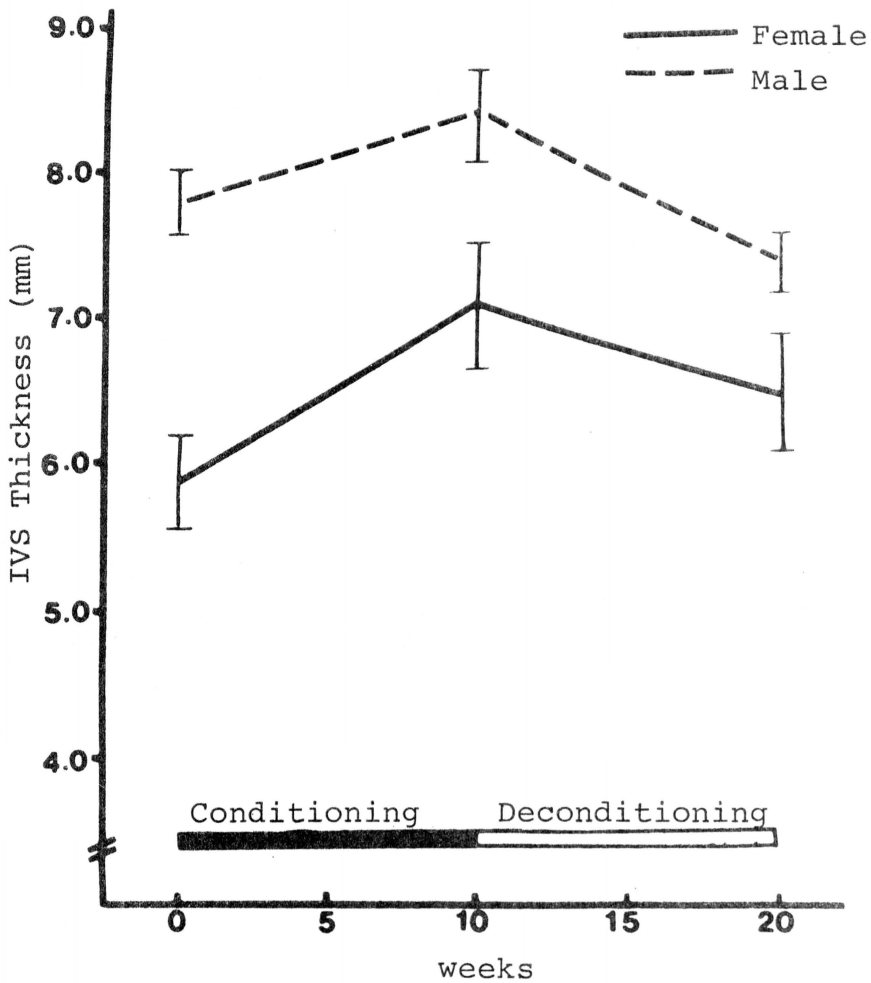


Figure 5. Interventricular septal thickness (mean ± SE) response during conditioning and deconditioning.

freedom the F ratio remained significant, therefore, no test of compound symmetry was performed.

Table 17

Matrix of Mean Differences for Interventricular Septum Thickness

	Females			Males		
	T1	T2	T3	T1	T2	T3
Females T1	--	.12	.06	.19	.23	.15
Females T2		--	.06	.07	.11	.03
Females T3			--	.03	.07	.09
Males T1				--	.04	.04
Males T2					--	.08
Males T3						--

*$C_{.05}(6,36) = \pm .324$

Left Ventricular Dimension at Diastole

The analysis of variance and the matrix of mean differences on the LVIDd are presented in Tables 18 and 19, respectively. A significant interaction between sexes and trials of LVIDd at the .05 level of significance may be seen from Table 18. Table 19 illustrates the LVIDd values for the female subjects were significantly different from the males. A significant F ratio between trials was found

for the males' LVIDd. The differences were between T1 and T2, and T2 and T3.

Table 18

Analysis of Variance on Left Ventricular Dimension at Diastole

SV	df	SS	MS	F	p
Between					
Group	1	5.642	5.642	7.95*	.011
S (group) error	18	12.770	.709		
Within					
Trials	2	1.010	.505	11.87**	.000
Trials X Group	2	0.624	.312	7.34**	.002
TxS (group) error	36	1.532	.042		

*$F_{.05}(1,18) = 4.41$

**$F_{.05}(2,36) = 3.32$

The between groups F ratio and significant mean differences between sexes in Tables 18 and 19 as well as the graphs in Figure 6 indicate that the males' LVIDd was significantly greater than those of the females in all conditions. The main effects of the trials cannot be interpreted because the significant F ratio is a result of the changes in the male subjects only.

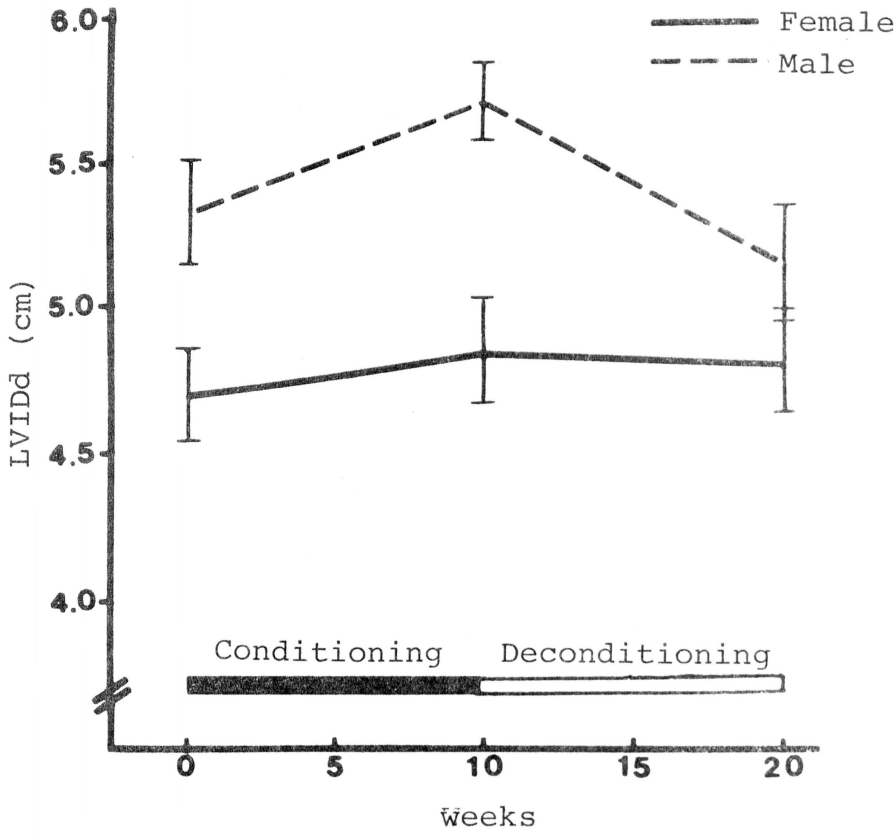


Figure 6. Left ventricular internal dimension at diastole (mean ± SE) response during conditioning and deconditioning

Table 19

Matrix of Mean Differences for Left Ventricular Internal Dimension at Diastole

	Females			Males		
	T1	T2	T3	T1	T2	T3
Females T1	--	.17	.12	.67*	1.00*	.46*
Females T2		--	.05	.50*	.83*	.29*
Females T3			--	.55*	.88*	.34*
Males T1				--	.33*	.21
Males T2					--	.54*
Males T3						--

*$\underline{C}_{.05}(6,36) = \pm .276$

Left Ventricular Internal Dimension at Systole

An analysis of variance of the influence of conditioning-deconditioning programs upon the left ventricular internal dimension at systole for 20 college female and male subjects is illustrated in Table 20. Studying Table 20 reveals that there was a significant interaction between sexes and trials at the .05 level. The male subjects' LVIDs showed no significant change from T1 to T2, whereas

it decreased from T2 to T3, but insignificantly. The female subjects' LVIDs did not significantly change through the three trials (see Table 21 and Figure 7).

Table 20

Analysis of Variance on Left Ventricular Internal Dimension at Systole

SV	df	SS	MS	F	p
Between					
Group	1	4.816	4.816	8.57*	.009
S (group) error	18	10.116	.562		
Within					
Trials	2	.041	.020	0.28	.761
Trials X Group	2	.741	.370	4.93**	.013
TxS (group) error	36	2.704	.075		

*$F_{.05}(1,18) = 4.41$

**$F_{.05}(2,36) = 3.32$

The between trials F ratio in Table 20 did not reach significance at the .05 level. It was also noted that there was a significant difference between the LVIDs of female and male subjects at T1 and T2.

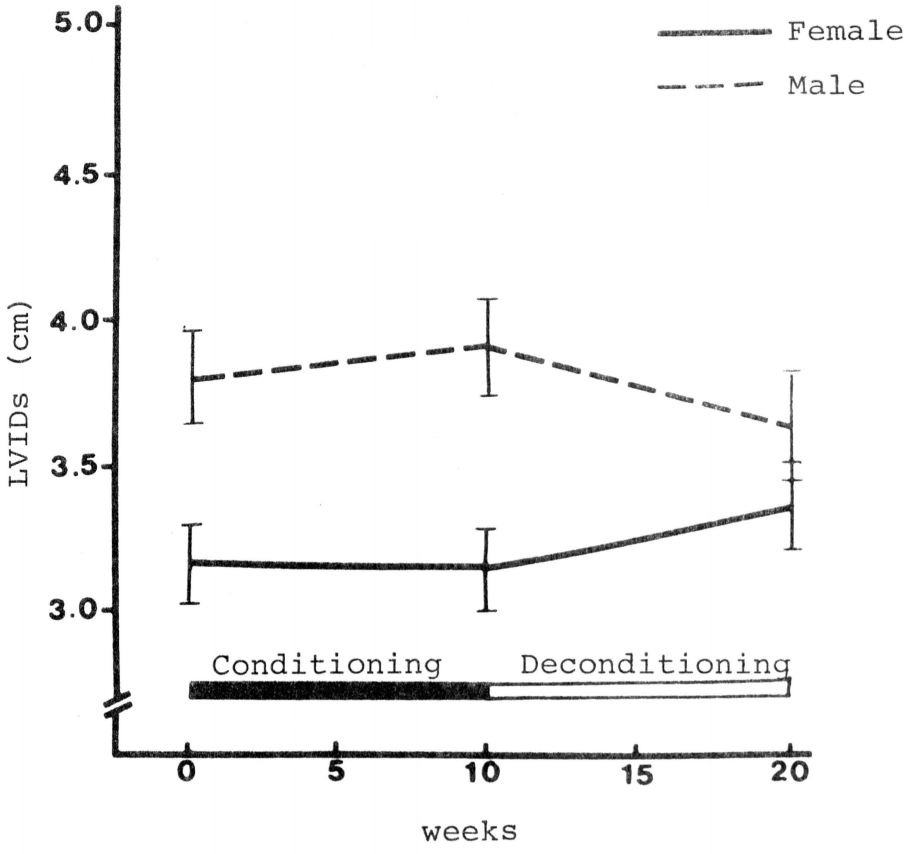


Figure 7. Left ventricular internal dimension at systole (mean = SE) response during conditioning and deconditioning

Table 21

Matrix of Mean Differences for Left Ventricular Internal Dimension at Systole

	Females			Males		
	T1	T2	T3	T1	T2	T3
Females T1	--	0	.21	.66*	.78*	.41*
Females T2		--	.21	.66*	.78*	.41*
Females T3			--	.45*	.57*	.26
Males T1				--	.12	.19
Males T2					--	.31
Males T3						--

*$\underline{C}_{.05}(6,36) = \pm .369$

Left Ventricular Volume at Diastole

Table 22 illustrates the analysis of variance of the effect of conditioning-deconditioning programs upon the $LVIDd^3$ of 20 college female and male students. It may be noted that there was a significant interaction between sexes and trials when measuring the left ventricular volume at diastole of 20 female and male college students at T1, T2 and T3.

Table 22

Analysis of Variance on Left Ventricular Volume at Diastole

SV	df	SS	MS	F	p
Between					
Group	1	35022.2	35022.3	7.52*	.013
S (group) error	18	83851.0	4658.4		
Within					
Trials	2	6917.1	3458.6	11.22**	.000
Trials x Group	2	4479.9	2239.9	7.27**	.002
TxS (group) error	36	11098.3	308.3		

*$F_{.05}(1,18) = 4.41$

**$F_{.05}(2,36) = 3.32$

From a study of the mean of $LVIDd^3$ graph changes in Figure 8 and the matrix mean differences in Table 23, it appears that there was greater increase and decrease in the males' $LVIDd^3$ from T1 to T2 and from T2 to T3, respectively, than in the females. The male values were significant although this was not found for the female subjects. The significant difference between trials was supported by a significant F ratio in Table 22. Using conservative degrees of freedom the F ratio remained significant, therefore, no test of compound symmetry was performed. There was also a

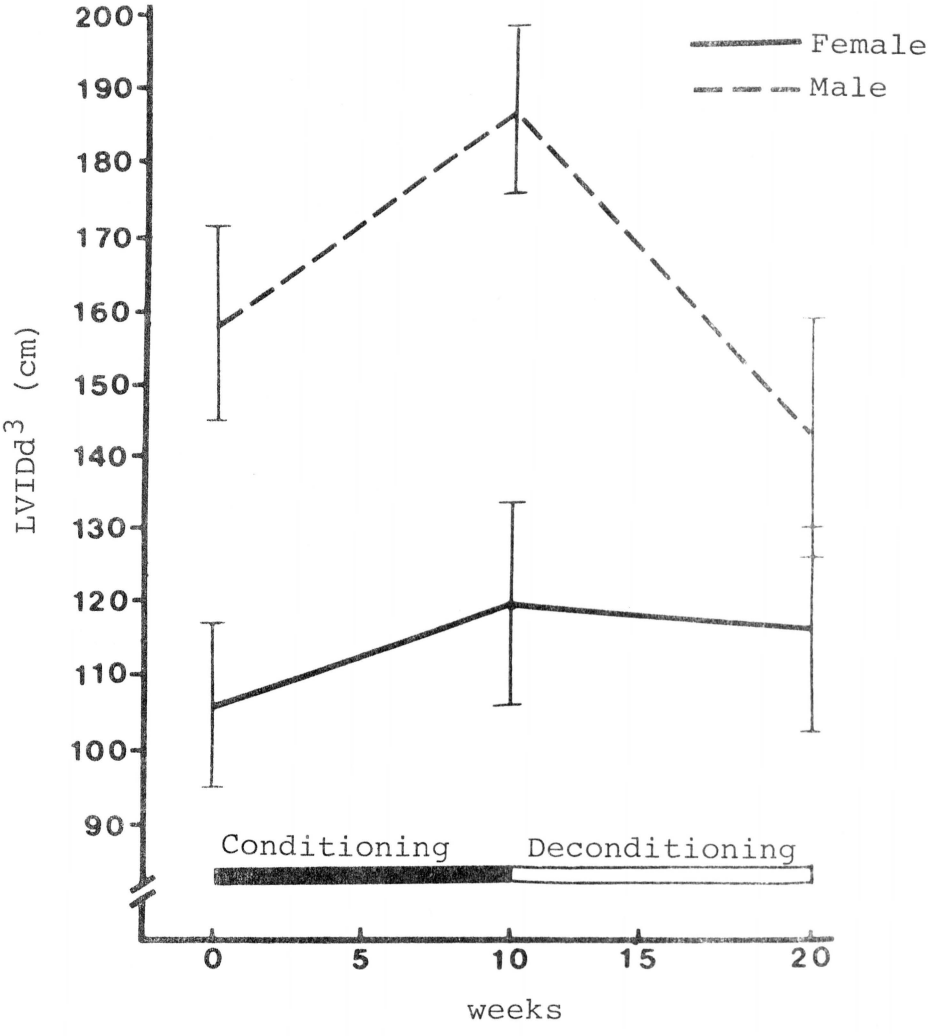


Figure 8. Left ventricular volume at diastole (mean ± SE) response during conditioning and deconditioning

significant difference noted between the LVIDd of females and males at the .05 level in Tables 22 and 23, and Figure 8.

Table 23

Matrix of Mean Differences for Left Ventricular Volume at Diastole

	Females			Males		
	T1	T2	T3	T1	T2	T3
Females T1	--	12.9	9.8	51.3*	80.7*	35.6*
Females T2		--	3.1	38.4*	67.8*	22.7
Females T3			--	41.5*	70.9*	25.8*
Males T1				--	29.5*	15.6
Males T2					--	45.1*
Males T3						--

*$C_{.05}(6,36) = \pm 23.65$

Left Ventricular Volume at Systole

The analysis of variance for changes on left ventricular volume at systole as a result of conditioning-deconditioning programs of female and male college students is presented in Table 24. There was a significant interaction noted between sexes and trials at the .05 level for the

left ventricular volume at systole, therefore, the reader should interpret the main effect with caution. The males showed significantly greater $LVIDs^3$ than the females except T3 as illustrated in Table 24 and Figure 9.

Table 24

Analysis of Variance on Left Ventricular Volume at Systole

SV	df	SS	MS	F	p
Between					
Group	1	6963.88	6963.88	8.29*	.010
S (group) error	18	15126.99	840.39		
Within					
Trials	2	143.56	71.78	.61	.547
Trials X Group	2	1075.02	537.51	4.60**	.017
TxS (group) error	36	4206.50	116.85		

*$F_{.05}(1,18) = 4.41$

**$F_{.05}(2,36) = 3.32$

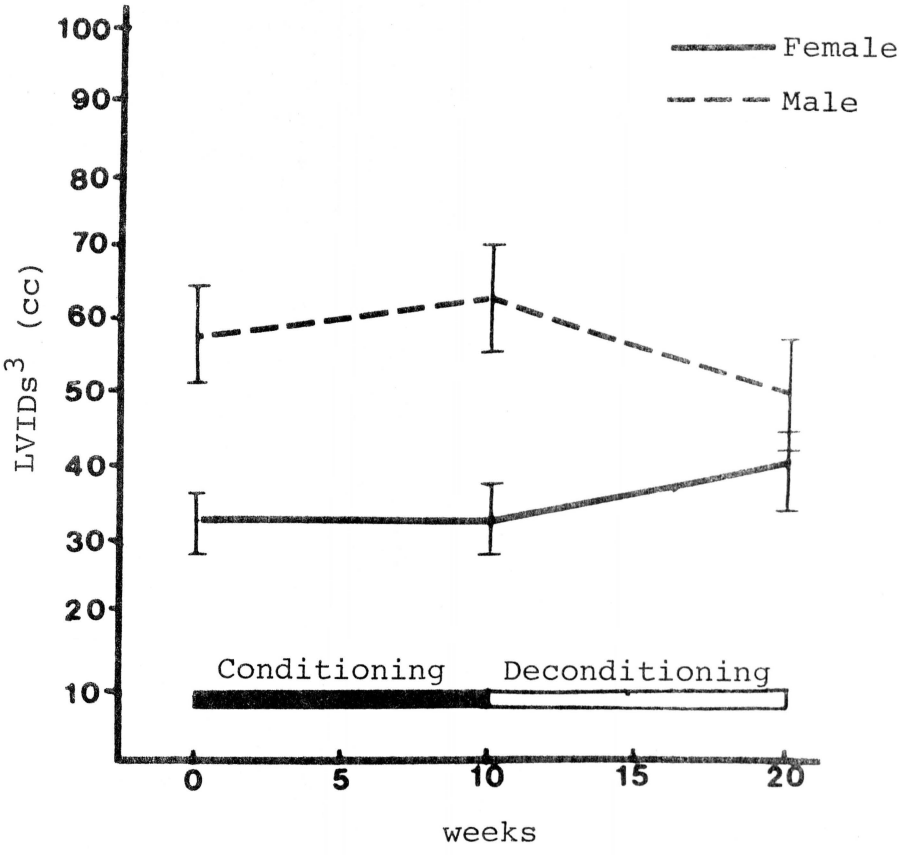


Figure 9. Left ventricular volume at systole (mean ± SE) response during conditioning and deconditioning

Table 25

Matrix of Mean Differences on Left Ventricular Volume at Systole

	Females			Males		
	T1	T2	T3	T1	T2	T3
Females T1	--	.86	7.45	25.01*	30.60*	17.34*
Females T2		--	6.59	24.14	29.74*	16.48*
Females T3			--	17.56*	23.15*	9.89
Males T1				--	5.59	7.67
Males T2					--	13.26
Males T3						--

*$\underline{C}_{.05}(6,36) = \pm 14.56$

Stroke Volume

Table 26 presents the analysis of variance of stroke volume changes in college female and male students as a result of conditioning and deconditioning programs. It may be seen that there was significant interaction between the sexes by trials when measuring the stroke volume. From the graph of mean SV values presented in Figure 10 and from the matrix on mean differences in Table 27, it may be noted that there was a significant increase in the

males' SV, from T1 to T2 and then it significantly decreased for the males but not for the females.

Table 26

Analysis of Variance of Stroke Volume

SF	df	SS	MS	F	p
Between					
Group	1	10805.78	10805.78	5.01*	.038
S (group) error	18	38796.70	2155.73		
Within					
Trials	2	5055.36	2527.68	14.57**	.000
Trials X Groups	2	1206.77	603.38	3.48**	.042
TxS (group) error	36	6243.68	173.43		

*$F_{.05}(1,18) = 4.41$

**$F_{.05}(2,36) = 3.32$

The changes in the males' SV that occurred following conditioning and deconditioning were responsible for the significant F ratio between trials illustrated in Table 26. The main effect of trials cannot, therefore, be interpreted. Using conservative degrees of freedom, therefore, no test of compound symmetry was performed. The F ratio between groups was, once again, significant, indicating that male subjects had a greater SV when compared to the female subjects. This

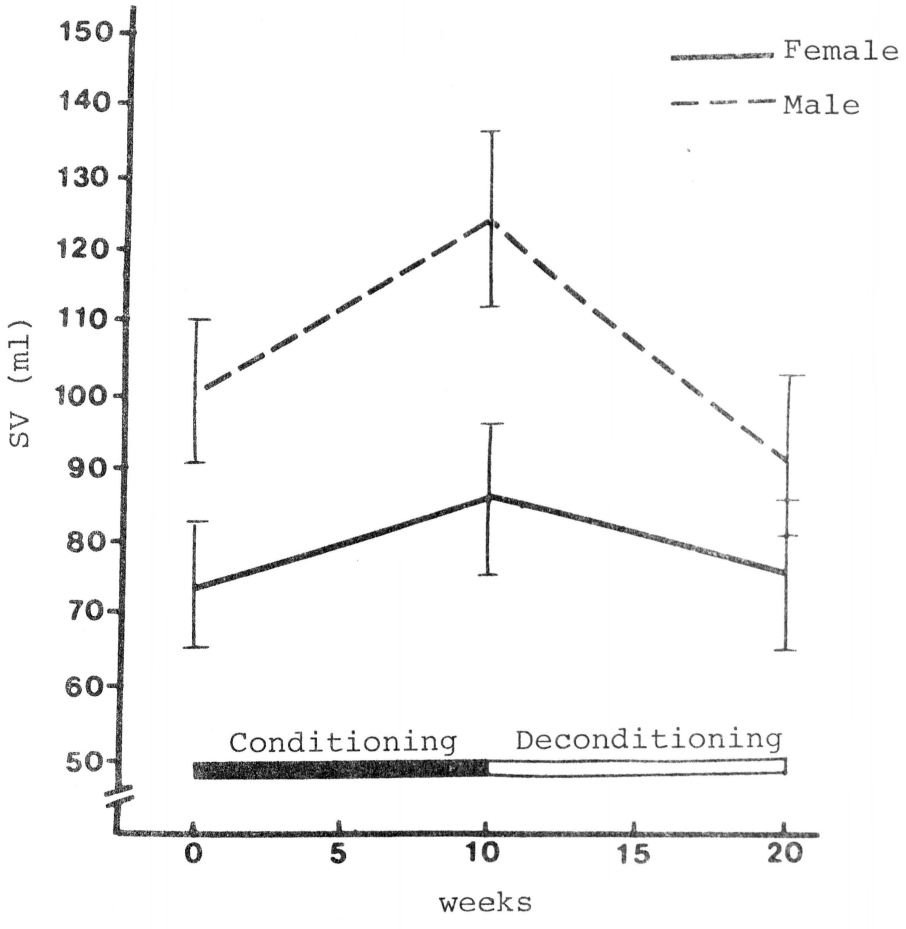


Figure 10. Stroke volume (mean ± SE) response during conditioning and deconditioning

is evidenced by the significant difference between the sexes during the first two trials (Table 27).

Table 27

Matrix of Mean Differences of Stroke Volume

	Females			Males		
	T1	T2	T3	T1	T2	T3
Females T1	--	12.1	2.4	26.3*	50.2*	18.50*
Females T2		--	9.7	14.2	38.1*	6.40
Females T3			--	23.9*	47.8*	16.10
Males T1				--	23.9*	7.76
Males T2					--	31.60*
Males T3						--

*$\underline{C}_{.05}(6,36) = \pm 17.74$

Ejection Fraction

Table 28 illustrates the influence of conditioning-deconditioning programs upon the ejection fraction of female and male students. No significant interaction was found between sexes and trials. Also, there were no significant differences between trials or between groups.

Table 28

Analysis of Variance on Ejection Fraction

SV	df	SS	MS	F	p
Between					
Group	1	193.32	193.32	1.47	.240
S (group) error	18	2359.33	131.07		
Within					
Trials	2	209.24	104.62	2.30	.114
Trials X Groups	2	113.05	56.52	1.25	.300
TxS (group) error	36	1634.11	45.39		

*$F_{.05}(1,18) = 4.41$

**$F_{.05}(2,36) = 3.32$

In Chapter V, a summary of the findings, a conclusion based upon the findings, a discussion of the findings of the study, and recommendations for further studies are presented.

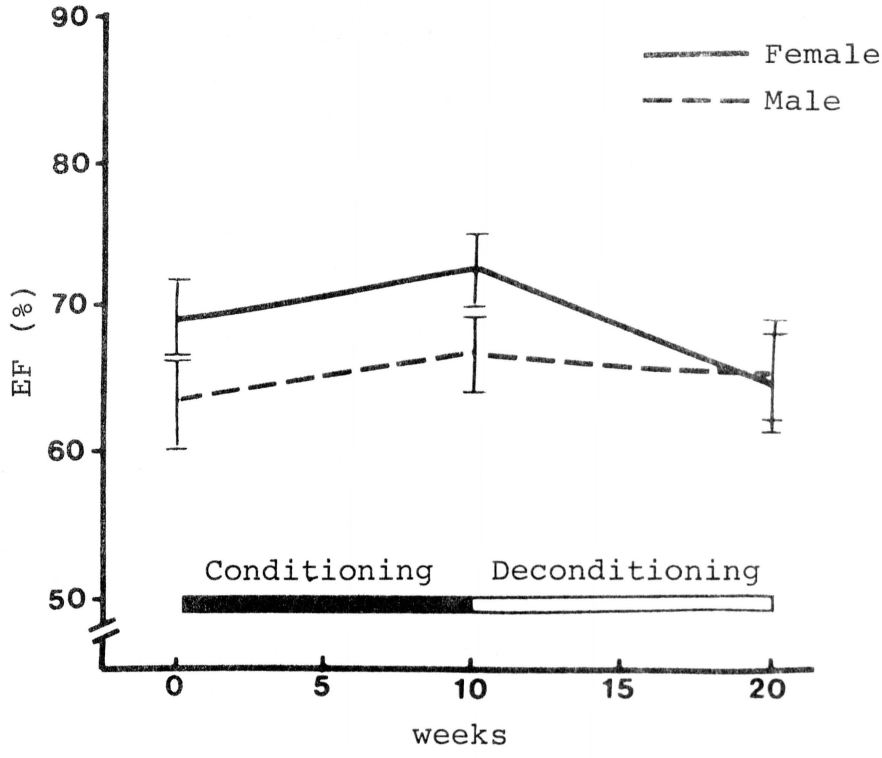


Figure 11. Ejection fraction (mean ± SE) response during conditioning and deconditioning

CHAPTER V

SUMMARY, CONCLUSIONS, DISCUSSION, AND RECOMMENDATIONS FOR FURTHER STUDIES

Summary

The literature reveals many studies that have considered, by means of echocardiography, the influence of prolonged physical conditioning upon the cardiac structure. The effects of prolonged deconditioning have not been determined by echo. Thus, structural changes from deconditioning are assumed rather than confirmed. The purpose of this investigation was to determine, by echo, the cardiac structural response to physical conditioning and deconditioning in college male and female students.

Ten male and ten female students were studied during a ten-week physical conditioning and a ten-week deconditioning program. The maximum oxygen uptake and selected blood serum enzymes were measured to validate the conditioning and deconditioning programs. Echocardiographic measurements were obtained from each subject prior to conditioning, following conditioning, and following deconditioning. A two-way analysis of variance with repeated

measures and Tukey's subsequent test were used to treat the data.

The following findings were obtained from this investigation:

1. In the comparison of the cardiovascular values of 20 female and male college students over the three trials, it was revealed that no significant interactions occurred between trials and sexes, but there were significant differences between the trials for the resting heart rate, the treadmill performance time, and the maximal oxygen uptake.
2. A comparison of the serum enzymes values of the subjects revealed that for LDH, no significant interaction was observed; there was a significant difference for the resting value between T1 and T2 and between T2 and T3. There were significant differences between the two sexes for the resting and postexercise CPK.
3. Comparison of the echo values for the 20 female and male students over the three trials revealed that there was significant interaction between sexes and trials for the Ao diameter, LVIDd, LVIDs, $LVIDd^3$, $LVIDs^3$, and SV. The analysis revealed that there were significant differences between the trials for the PWT, IVS thickness, LVIDd, $LVIDd^3$, and SV. It was further determined

that there were significant differences between sexes for the Ao diameter, LAD, PWT, IVS thickness, LVIDd, LVIDs, $LVIDd^3$, $LVIDs^3$, and SV.

Based upon the statistical analysis, the hypotheses of the study were answered as follows:

1. The first hypothesis, that there are no significant differences in the cardiac dimensions prior to conditioning, following conditioning, and following deconditioning programs, as measured by echo, was accepted. No significant changes occurred in the famales. For the males, significant increases occurred in LVIDd, $LVIDd^3$, and SV followong conditioning. After the completion of the deconditioning program there were significant decreases in LVIDd, $LVIDd^3$, $LVIDs^3$, and SV.
2. The second hypothesis that there are not significant differences between sexes in cardiac dimensions as measured by echo was rejected. Significant differences were found in all echo measures except in EF.
3. The third hypothesis that there are no significant interactions between trials and sexes in cardiac dimensions as measured by echo was rejected. Significant interactions were found in 6 of the 10 measures investigated.

Conclusion of the Study

It was concluded that some specific cardiac structures and dimensions increase following a short physical conditioning program and that following a deconditioning program some of the structures tend to decrease in the male only. The changes, in general, favored the male subjects. The females did not show the same adaptations as the male subjects.

Discussion

The problem of this study was to investigate the influence of 30 min of jogging at 70% of maximal heart rate for the first 2 weeks, then at 80%, every other day for 8 weeks, upon the heart structural changes of female and male subjects. These results were compared with data collected from the same subjects after ten weeks of deconditioning.

The findings of this study suggest that 10 weeks of physical conditioning and 10 weeks of deconditioning programs were sufficient for some specific cardiorespiratory adaptation to take place, as indicated by significant changes in the physical working capacity, blood enzymes, and certain echocardiographic parameters. Following the

conditioning period there was significant improvement of the oxygen transport system as the increase in the maximal oxygen uptake (20.7%), treadmill performance time (24.4%), decrease in resting heart rate (11.8%), and the resting LDH level (27.9%) illustrate. The maximal oxygen uptake measured in milliliter per kilogram of body weight per minute is considered to be the singular most indicative test for cardiovascular fitness (Astrand et al., 1977). The decrease in resting heart rate, which was found in this study to decrease following a physical conditioning program, supported the findings of previous studies (DeMaria et al., 1977); Gilbert et al., 1977; Wolfe et al., 1979; Zeldis et al., 1978). Increased sympathoinhibitory mechanism and centrogenic vagal cholinergic drive was assumed to be produced by physical conditioning which in turn caused a decrease in heart rate and therefore a greater filling of the heart (Astrand et al., 1977). The increased treadmill performance time following conditioning further supported the establishment of greater cardiovascular fitness. DeMaria et al. (1977), Wolfe et al. (1979) and the present investigator did not find a significant change in body weight following the conditioning-deconditioning programs. According to Astrand (1977), any weight that has been lost (fat tissue) might be replaced by muscle mass

hypertrophy due to training. Following 10 weeks of deconditioning, the parameters that were studied were reversed, indicating the loss of fitness.

An unexpected outcome was observed in the unchanged blood serum enzymes analysis at rest, and three hours following maximal treadmill performance, when the three trials were compared. This finding did not agree with previous studies (Fowler et al., 1962; Gardner et al., 1962; Hunter et al., 1971) which found an increase of CPK, LDH, and GOT in untrained subjects; after conditioning in well trained subjects, the efflux of these enzymes from muscle cells was found to be reduced. A major factor in the results of change in blood enzymes following maximal performance logically depends upon the timing of the blood drawing as it was claimed by Buyze and Egberts (1976). They found a significant increase in CPK, LDH, and GOT 3 hours following physical exercise. Therefore, the unchanged enzymes in this study cannot be explained.

Lactic dehydrogenase (LDH) was the only enzyme to decrease significantly after conditioning and then increase after deconditioning. This agreed with the findings of Gardner et al. (1964). The efflux from the muscle cell and its membrane permeability follows the anticipated path indicating the adaptation of cardiac and skeletal muscle.

The cardiac adaptation to physical conditioning measured by echo was found by previous investigators (DeMaria et al., 1977; Ehsani et al., 1978;Gilbert et al., 1977; Grayevskaya et al., 1979; Longhurst et al., 1980; Morganroth et al., 1975, Zeldis et al., 1978; Zoneraich et al., 1977). There has been no echo study, however, that compared the female and male cardiac adaptation to the same physical training program. Also, the influence of deconditioning upon this adaptation has not been evaluated by echo except in one study (Ehsani et al., 1978). That study was limited to 3 weeks of detraining 6 college cross-country male runners.

The diameters of the aorta and left atrium did not change following conditioning or deconditioning in this study. A comparison between marathon runners and sedentary men by Zoneraich et al. (1977) revealed no difference between their aortic and left atrial sizes. Morgaroth and his associates (1975) compared endurance athletes (swimmers and long distance runners) to athletes (wrestlers and shot putters) involved in more anaerobic activities, and then to non-athlete subjects. The subjects were all matched for age and weight. They found no significant difference in aortic and left atrial diameters. In this study the aortic and left atrial diameter of the male subjects were significantly greater than the females at the beginning and end

of the study. Longhurst et al. (1980) explained that this might be only a result of the differences in body size and weight.

Ten weeks of conditioning produced an increase in the posterior wall and interventricular septum following conditioning. The posterior wall and septal adaptation to conditioning was in agreement with the results of previous studies (DeMaria et al., 1977; Ehsani et al., 1978; Gilbert et al., 1977; Grayevskays et al., 1979). The increase in the heart wall dimension plus the increased left ventricular cavity were considered primary factors in the increase in the left ventricular mass of competitive long distance runners when compared with competitive and amateur weight lifters and with control subjects (Longhurst et al., 1980). Zeldis and associates (1978) found no significant difference between athletes and nonathlete female subjects in the posterior wall and the interventricular septal thickness. Ikaheimo et al. (1979) suggested that an increase in the volume overload in addition to the systolic blood elevation led to left ventricular hypertrophy in 22 competitive male endurance runners. However, it was noted by DeMaria et al. (1977) that a hypertrophy caused by hemodynamic overload was not associated with an actomyosin ATPase activity increase, which was found with myocardial hypertrophy following conditioning.

The nonsignificant response to deconditioning for all of the subjects in the posterior wall thickness and the interventrical septal thickness disagrees with the findings of Ehsani et al. (1978) and Leon et al. (1968). These studies suggested that adaptation of left ventricular size and weight in response to physical exercise appeared to be lost during deconditioning if the duration was less than half of the prior conditioning period. Longer periods of deconditioning may be needed to answer this definitely.

The increased left ventricular internal dimension and volume at diastole were major cardiac adaptations for the men only, to the 10 weeks of conditioning in this study. DeMaria and associates (1977) claimed that the reduction in heart rate, as a result of conditioning programs, the age of the subjects, and the physiological status of the subjects involved in the study were the factors that contributed to the enlargement of the left ventricular cavity. The last two explain the results of Wolfe et al. (1979) that even after 6 months of physical conditioning, the older (37 years) and more fit (42.4 ml/kg/min.) subjects did not develop significant left ventricular adaptation to training.

The left ventricular internal dimension and volume at systole were not significantly different between trials. This finding agreed with Ikaheimo et al. (1979). Some studies (DeMaria et al., 1977; Parker et al., 1978) found reduced left ventricular internal dimension and volume at diastole following 11 weeks of conditioning or in competitive long distance runners. Zeldis et al. (1978) found them to be increased when 10 female college age field hockey players were compared to 10 nonathletes.

The left ventricular adaptation at diastole and systole to conditioning and deconditioning produced a significant increase in the stroke volume for the males. The results for stroke volume after 10 weeks of conditioning was not in agreement to this data found by Wolfe et al. (1979). Their subjects showed no significant difference in the left ventricular dimensions and volumes at diastole and systole; yet, there was an increase in stroke volume following six months of conditioning. In explanation, Wolfe and colleagues noted that the increase was attributable to a lower resting heart rate following conditioning which, in turn, allowed an increased ventricular filling. Hirshleifer et al. (1975) were able to illustrate that as a result of atropine administration heart rates increased from 64 to 98 beats per minute while the mean of end diastolic dimension decreased 2 mm only. In their

study there was only a decrease of 8 beats per minute following conditioning. Therefore, the effect of the decreased resting heart rate upon the left ventricular dimension changes were believed to be minimal.

In the present study the ejection fraction was found to be normal between trials and between sexes. Ehsani et al. (1978) and Zeldis et al. (1978) found no significant changes in ejection fraction between athletes and nonathletes after conditioning. In addition, Zeldis et al. reported no difference between his athletic female subjects' ejection fractions and the athletic male values reported in previous studies. Roeske et al. (1976) and Parker et al. (1978) found greater left ventricular ejection fractions, whereas Rost et al. (1975) found the ejection fraction to be lower in athletes when compared to nonathletes. None of the investigators gave an explanation for the findings.

The 10 week deconditioning program produced a significant decrease in left ventricular cavity and stroke volume for the males in this study. These findings replicated the results of an investigation by Ehsani et al. (1978). The large decrease in stroke volume in the males following deconditioning was parallel to their left ventricular cavity reduction. Deconditioning resulted in a decrease of left ventricular dimension and volume at diastole, but did not influence the cardiac walls thickness following

deconditioning in both groups. Therefore, the deconditioning program in this study was sufficient to produce a reduction in the left heart ventricular dimensions, and performance of the heart, which in turn produced a loss in cardiovascular fitness in the male subjects. Other investigators (Drinkwater et al., 1972; Fardy, 1969; Fringer et al., 1974; Knuttgen et al., 1973) claimed a reduction in cardiovascular and respiratory efficiency due to deconditioning periods ranging from five weeks to eight months. Applegate et al. (1969) and Grayevskaya et al. (1979) noted that for a cardiac adaptation to conditioning to be preserved, a certain amount of training must be maintained.

In general, the findings of this study indicate that the conditioning and deconditioning programs produced a measurable increase and decrease, respectively, in the subjects' ability to perform maximal exercise (maxVo_2 and treadmill performance time) with a significant difference between the sexes occurring after training. More importantly for this study, there was a significant increase in a small number of specific heart structures for both sexes in response to the conditioning and for the males only, there was a deconditioning adaptation. The males had significantly larger heart volumes and dimensions than the female subjects. The male subjects were heavier and taller than the females, therefore, a larger heart

would be needed to support the greater body mass and surface area. The smaller female hearts were capable of transporting the required blood to the working organs in order to perform the same task as the males (Astrand et al., 1977; Longhurst et al., 1980).

Recommendations for Further Studies

After conducting the conditioning-deconditioning programs and analyzing the results of the present study, the investigator recommends the following studies be undertaken:

1. A study similar to the present one with subjects from different age groups to examine the influence of conditioning and deconditioning upon the cardiac structures of children, young, middle-age, and older subjects of both sexes.
2. A study similar to the present one utilizing a longer conditioning program with a longer duration of deconditioning to determine the effects of prolonged physical inactivity on the cardiac structures after training effects are noted.
3. A study similar to the present one utilizing echo recording to determine the effect of different durations of complete bed rest upon cardiac structures.

4. A study similar to the present one with coronary artery disease patients as subjects performing through the conditioning and deconditioning program.
5. A study similar to the present one utilizing correlation techniques for the cardiorespiratory performance measures and the echo parameter changes due to conditioning and deconditioning.

APPENDICES

Raw Values of Age, Weight and Height

	Age (Years)	Weight (kg)	Height (cm)
Female			
1	27	58.8	170.5
2	30	72.9	168.0
3	19	56.8	159.0
4	21	57.7	168.0
5	27	67.3	160.0
6	22	67.1	168.0
7	31	57.9	170.5
8	24	55.5	162.8
9	31	55.0	151.3
10	22	51.4	166.0
Male			
1	21	60.9	176.4
2	22	77.0	180.3
3	30	60.5	159.0
4	28	55.5	162.5
5	27	98.1	177.0
6	29	67.7	177.0
7	26	65.9	162.5
8	25	112.3	188.0
9	28	85.5	175.0
10	29	81.7	178.2

Raw Resting and Maximal Heart Rate Values (bpm)

	Resting Heart Rate			Maximal Heart Rate		
	T1	T2	T3	T1	T2	T3
Female						
1	68	55	64	185	188	198
2	54	51	61	195	172	200
3	83	62	63	180	200	175
4	66	65	68	192	184	190
5	93	82	75	200	192	190
6	81	88	80	188	188	188
7	66	59	65	190	190	194
8	80	78	82	196	190	185
9	63	49	46	182	186	180
10	94	77	88	200	188	190
Male						
1	85	68	88	190	196	190
2	54	50	60	192	202	200
3	81	60	55	188	182	189
4	69	72	60	182	182	188
5	64	54	90	172	178	189
6	78	83	75	198	178	186
7	54	63	85	150	170	185
8	94	75	80	200	178	190
9	87	53	87	190	190	199
10	85	78	75	180	182	185

Raw Max VO_2 (ml/kg/min) and Treadmill Performance Time (Min.) Values

	Max VO_2			Treadmill Time		
	T1	T2	T3	T1	T2	T3
Female						
1	28.86	43.40	36.11	7.5	10.0	8.8
2	40.08	46.60	43.60	8.5	10.8	10.9
3	37.32	42.87	30.23	8.6	11.0	8.1
4	46.46	60.40	41.92	10.0	10.8	8.4
5	33.90	35.80	33.97	7.7	9.7	8.0
6	44.72	57.24	43.28	11.0	13.4	10.0
7	40.14	44.38	32.62	8.5	10.2	10.0
8	42.55	44.23	35.81	8.4	11.9	9.1
9	39.58	46.62	41.56	9.4	12.6	11.2
10	26.52	38.70	38.80	5.5	10.5	8.5
Male						
1	39.27	47.64	45.20	9.3	13.3	10.8
2	49.79	49.06	44.79	10.5	11.0	10.5
3	49.90	55.35	46.99	9.7	11.8	8.9
4	50.60	58.11	45.74	9.3	10.7	9.0
5	36.07	42.82	39.48	7.7	10.1	8.1
6	42.72	42.86	46.46	9.6	8.2	11.7
7	36.01	55.77	39.23	7.9	10.4	8.3
8	30.02	45.60	34.89	7.0	10.8	7.4
9	38.31	52.21	43.62	9.7	10.5	11.5
10	43.22	51.50	43.13	10.2	11.3	8.0

Raw Resting and Post Exercise CPK Values (U/L)

	Resting GOT			Post Exercise GOT		
	T1	T2	T3	T1	T2	T3
Female						
1	57	44	62	60	67	56
2	92	44	64	69	152	82
3	78	38	40	49	49	52
4	26	37	19	40	39	40
5	29	19	35	20	29	50
6	39	78	48	64	71	74
7	40	48	52	56	78	59
8	58	55	66	61	79	62
9	45	86	48	49	190	51
10	20	22	35	25	35	26
Male						
1	79	85	74	87	79	63
2	136	62	64	128	51	76
3	66	60	55	66	109	71
4	106	75	72	74	92	75
5	65	46	77	80	112	102
6	53	76	34	57	62	82
7	91	68	85	75	67	58
8	76	72	95	90	95	100
9	54	64	73	87	112	77
10	89	59	65	90	88	90

Raw Resting and Post Exercise GOT Values in U/L

	Resting GOT			Post Exercise GOT		
	T1	T2	T3	T1	T2	T3
Female						
1	12	26	11	15	23	15
2	32	15	3	15	19	17
3	22	13	21	14	15	26
4	18	13	19	15	10	9
5	7	9	3	11	9	16
6	16	12	19	12	17	14
7	8	13	14	12	13	17
8	6	15	17	13	14	17
9	20	19	15	20	20	14
10	14	11	20	13	12	13
Male						
1	12	15	14	13	16	19
2	16	13	7	12	15	12
3	16	10	3	15	14	14
4	21	16	12	18	19	13
5	23	19	19	25	19	25
6	10	15	19	15	14	24
7	15	10	18	11	13	19
8	20	19	27	23	16	18
9	67	38	24	50	33	24
10	18	14	15	20	16	16

Raw Resting and Post Exercise LDH Value in U/L

	Resting LDH			Post Exercise LDH		
	T1	T2	T3	T1	T2	T3
Female						
1	191	147	209	226	187	248
2	198	138	192	209	168	201
3	193	107	130	93	146	141
4	185	162	156	183	135	162
5	140	85	158	145	119	163
6	163	131	205	197	186	205
7	151	134	154	202	130	170
8	157	134	150	197	171	173
9	215	191	182	166	200	187
10	148	120	278	177	144	175
Male						
1	174	108	148	193	139	157
2	209	114	130	133	178	138
3	192	128	159	112	165	167
4	164	125	139	75	162	136
5	186	141	197	153	153	196
6	170	105	200	96	158	175
7	157	109	188	157	170	131
8	220	140	244	263	182	236
9	279	175	215	357	220	260
10	178	147	146	221	163	172

Raw Values of Aortic and Left Atrial Diameter (cm)

	Ao Diameter			LAD		
	T1	T2	T3	T1	T2	T3
Female						
1	1.7	2.0	2.0	3.0	2.7	3.0
2	2.7	2.3	2.3	3.7	3.9	3.7
3	2.5	2.2	2.0	2.9	2.9	2.6
4	2.3	2.5	2.5	3.1	3.4	2.8
5	2.5	2.4	2.0	3.7	3.7	3.7
6	2.2	2.0	2.3	3.2	3.2	2.6
7	2.3	2.1	2.0	2.4	2.6	2.7
8	2.2	1.9	2.3	3.0	3.0	2.5
9	2.0	2.0	2.1	3.3	3.0	3.2
10	2.1	2.2	2.0	2.9	3.0	2.8
Male						
1	2.7	2.7	2.3	3.5	4.0	2.8
2	2.3	2.8	2.8	3.3	2.8	3.0
3	2.5	2.4	2.1	3.0	3.8	3.9
4	2.6	3.0	3.0	3.1	3.3	3.4
5	3.1	3.5	3.3	4.0	4.1	4.0
6	2.7	2.6	2.8	3.3	3.4	3.3
7	2.5	2.5	3.0	3.0	3.2	3.0
8	2.7	3.0	2.9	4.0	3.6	3.8
9	2.9	2.9	2.9	3.6	3.4	3.5
10	2.8	3.0	2.9	4.0	3.1	3.2

Raw Values of PWT and IVS Thickness (cm)

	PWT			IVS		
	T1	T2	T3	T1	T2	T3
Female						
1	.6	.7	.8	.6	.7	.8
2	.7	.7	.8	.6	.8	.9
3	.7	1.0	.7	.7	1.0	.7
4	.5	.6	.5	.5	.6	.5
5	.5	.6	.6	.5	.7	.6
6	.7	.7	.7	.7	.7	.6
7	.5	.6	.7	.5	.6	.7
8	.6	.7	.5	.7	.7	.5
9	.5	.6	.5	.5	.6	.5
10	.7	.7	.7	.6	.7	.7
Male						
1	.8	.8	.7	.8	.8	.7
2	.7	.8	.8	.8	.8	.8
3	.7	.9	.8	.8	.9	.8
4	.8	.8	.8	.8	.7	.7
5	.8	.8	.7	.8	.7	.8
6	.7	.7	.7	.8	.8	.7
7	.8	.8	.7	.7	.8	.7
8	.9	1.0	.8	.9	1.0	.8
9	.6	.8	.8	.7	.9	.7
10	.7	.7	.7	.7	.8	.7

Raw Values of LVIDd and LVIDs (cm)

	LVIDd			LVIDs		
	T1	T2	T3	T1	T2	T3
Female						
1	4.2	4.4	4.3	3.1	2.7	3.4
2	5.3	5.3	5.5	3.7	3.5	3.8
3	4.7	4.9	5.0	2.8	3.0	3.3
4	4.5	4.7	4.6	3.0	3.2	3.2
5	4.7	5.2	4.9	2.8	3.3	3.1
6	4.3	4.4	4.4	2.9	2.2	2.7
7	5.6	6.0	6.0	3.7	4.0	4.3
8	4.5	4.8	4.7	3.4	3.4	3.0
9	4.2	4.1	4.0	2.7	2.8	3.1
10	5.0	5.1	4.8	3.4	3.4	3.7
Male						
1	4.7	5.0	4.4	3.1	3.3	3.2
2	6.0	5.8	6.0	4.3	4.2	4.5
3	5.6	5.7	5.3	4.0	3.9	4.3
4	5.5	5.7	4.6	4.6	4.4	3.4
5	5.5	6.0	5.4	3.5	4.1	3.6
6	4.9	5.2	4.6	3.4	3.3	2.5
7	5.0	5.5	5.2	3.6	3.3	3.2
8	6.0	6.3	6.2	4.2	4.5	4.3
9	5.6	5.8	4.9	3.7	3.9	3.4
10	4.9	6.0	5.0	3.7	4.4	3.8

Raw Values of $LVIDd^3$ and $LVIDs^3$ (cc)

	$LVIDd^3$			$LVIDs^3$		
	T1	T2	T3	T1	T2	T3
Female						
1	74.2	85.2	79.5	29.8	19.7	39.3
2	148.9	148.9	166.4	50.7	42.9	54.9
3	103.8	117.6	125.0	22.0	27.0	35.9
4	91.1	103.8	97.3	27.0	32.8	32.8
5	103.8	140.6	117.6	22.0	35.9	29.8
6	79.5	85.2	85.2	24.4	10.6	19.7
7	175.6	216.0	216.0	50.6	64.0	79.5
8	91.1	97.3	103.8	39.3	39.3	27.0
9	74.2	68.9	64.0	19.7	21.9	29.8
10	125.0	132.7	110.6	39.3	39.3	50.6
Male						
1	103.8	125.0	85.2	29.8	35.9	32.8
2	216.0	195.1	216.0	79.5	74.1	91.1
3	175.6	185.2	148.9	64.0	59.3	74.0
4	166.4	185.2	97.3	97.3	85.2	39.3
5	166.4	216.0	157.5	42.9	68.9	38.9
6	117.6	140.6	97.3	39.3	35.9	15.6
7	125.0	166.4	140.6	46.7	35.9	32.8
8	216.0	250.1	238.3	74.1	91.1	79.5
9	175.6	195.1	117.6	50.6	59.3	39.3
10	117.6	216.0	125.0	50.7	85.2	54.9

Raw Values for SV (ml) and EF (%)

	SV			EF		
	T1	T2	T3	T1	T2	T3
Female						
1	44.3	65.5	40.2	59.7	76.9	50.6
2	98.2	106.0	111.5	66.0	71.2	67.0
3	81.8	90.6	89.1	78.8	77.0	71.3
4	64.1	71.0	64.5	70.4	68.4	66.3
5	81.8	104.7	87.8	78.8	74.5	74.7
6	55.1	74.6	65.5	69.3	87.5	76.9
7	125.0	152.0	136.5	71.2	70.4	63.2
8	51.8	58.0	76.8	56.9	59.6	74.0
9	54.4	47.0	34.2	73.3	68.2	53.4
10	85.7	93.7	60.0	68.8	70.6	54.2
Male						
1	74.0	89.1	52.4	71.3	71.3	61.3
2	136.5	121.1	124.9	63.2	62.1	57.8
3	111.6	125.9	74.9	63.6	68.0	50.3
4	69.1	100.0	58.0	41.5	54.0	59.6
5	123.5	147.1	118.6	74.2	68.1	75.3
6	78.3	104.7	81.7	66.5	74.5	84.0
7	78.3	130.5	107.8	62.6	78.4	76.7
8	141.9	159.0	158.8	65.7	63.6	66.6
9	125.0	135.8	78.3	71.2	69.6	66.6
10	66.9	130.8	70.1	56.9	60.6	56.1

INFORMED CONSENT FORM

In order to evaluate the functional performance of an individual it is necessary that the individual consent, voluntarily, to perform one or more exercise tests. Prior to being tested each individual will be orally questioned by an exercise physiologist or a graduate student in training, to determine whether any testing should be attempted. A standard series of questions relating to the health history will be asked. The basic exercise test consists of walking on a motor driven treadmill with the speed and gradient increasing every three minutes until the limits of fatigue, breathlessness, chest pain and/or other symptoms are of such severity that this test administrator or the subject feels that the test should be stopped, or when a predetermined heart rate or other value is reached. The echocardiography electrocardiogram and/or the blood pressure will be monitored prior to, during, and after the test. Expired air will be collected during the last minute or minutes of the test. A small blood sample will be drawn by venipuncture before and a few hours after the performance test.

Risks of testing include occasional changes in the rhythm of the heart beats and the possibility of unusual changes in blood pressure. There is a small chance of fainting and a very remote chance of a heart attack, particularly if the participant takes a hot shower after strenuous exercise testing. Professional supervision protects against injury by providing appropriate precautionary measures. In the unlikely event that these precautions are insufficient, emergency first aid and cardiopulmonary treatment is available. The university does not have available medical resources other than those except for registered students at the Student Health Service. No financial resources are available and the university cannot be responsible for any obligations incurred as a result of injury during the conduct of this investigation.

The physical conditioning program will consist of performance on a treadmill at 80% of your maximal oxygen uptake or jogging outside (at 80% of your maximal heart rate) for 30 minutes, three days a week, for eight to ten weeks.

The risks of the conditioning program would include strain to the muscles and/or sprains to the lower extremities joints, plus the risks for the functional performance test,

but at minimal chances. The deconditioning program consists of no exercise or hard work at all for the next eight to ten weeks.

The benefits of the testing include scientific assessment of the working capacity of each participant and an appraisal of any disorders or limitations that impair capacity for physical activity, which then compared with the echocardiography measurement, and the blood enzyme changes for the conditioning, deconditioning programs.

Both the right to withdraw from the test or tests at any time with impunity and the right to withhold confidential information from anyone not connected with the study in an administrative capacity without direct consent is assured. The welfare of each person will be protected.

Having read the information statement above and having had the opportunity to ask any questions, I willingly consent to be tested and to participate in the _____

_____

study conducted by _____

DATE_____ SIGNED_____

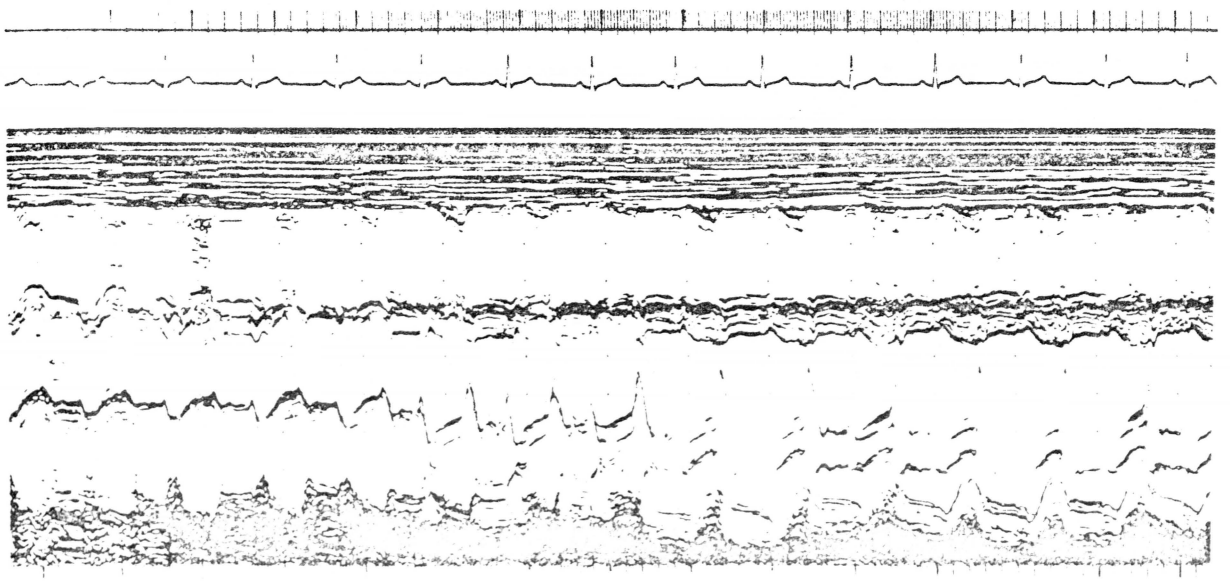
TIME_____ a.m. p.m.

WITNESS_____

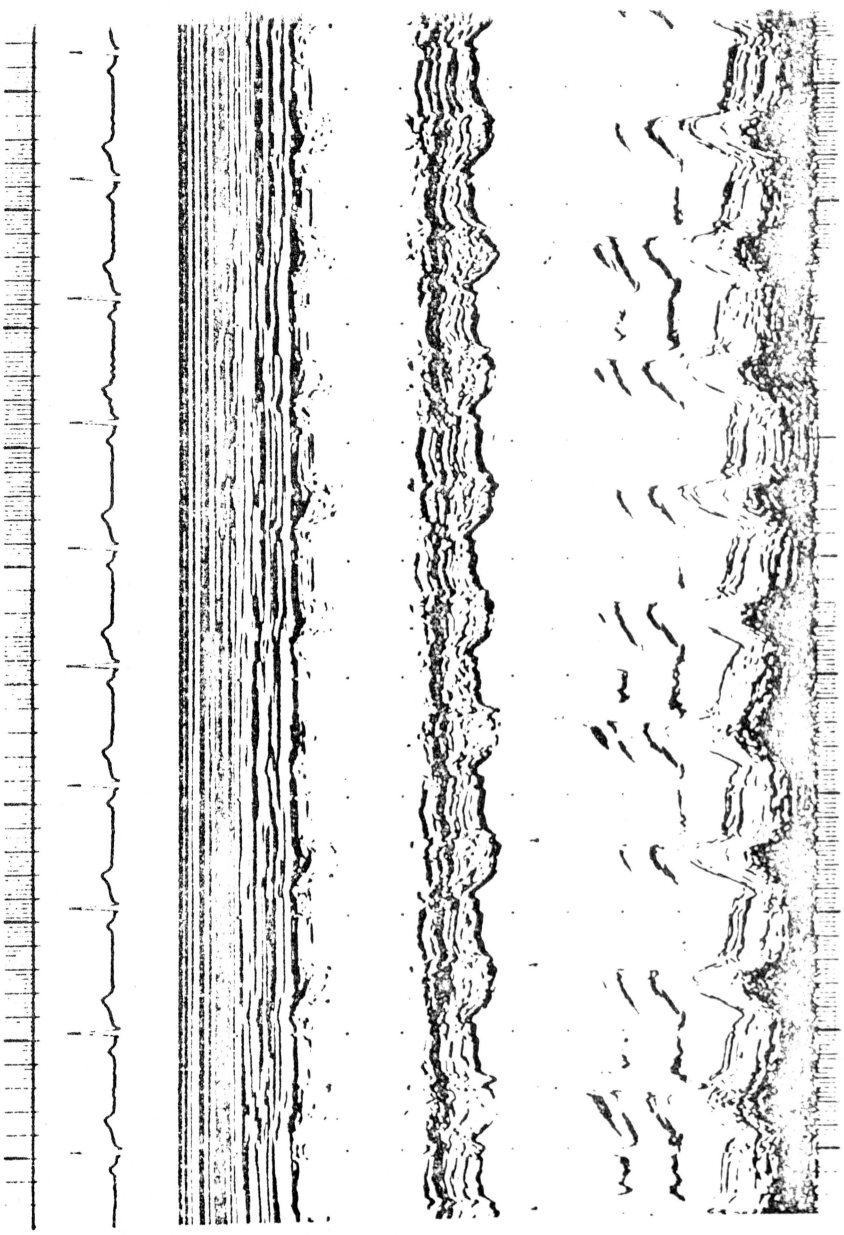
HEALTH SCREENING QUESTIONNAIRE

		Yes	No
1.	Do you wheeze or have to gasp to breathe?	___	___
2.	Are you bothered by coughing spells?	___	___
3.	Do you cough up a lot of phlegm (thick spit)?	___	___
4.	Have you ever coughed up blood?	___	___
5.	Do you get chest colds more than once a month?	___	___
6.	Are you sweating more than usual?	___	___
7.	Have you ever been told that you had high blood pressure?	___	___
8.	Have you been bothered by a thumping or racing heart?	___	___
9.	Do you ever get pains or tightness in your chest?	___	___
10.	Do you have trouble with dizziness or lightheadedness?	___	___
11.	Does every little effort leave you short of breath?	___	___
12.	Do you have trouble with swollen feet or ankles?	___	___
13.	Are you getting cramps in your legs at night or upon walking?	___	___
14.	Do you have hot flashes?	___	___
15.	Have you ever been told that you have a heart mumur?	___	___
16.	Are you a diabetic?	___	___
17.	Have you had surgery in the last six months?	___	___

		Yes	No
18.	Have you consulted a doctor within the last six months?	___	___
19.	Have you been ill within the last six months?	___	___
20.	Have you ever been told that you should not exercise stenuously?	___	___
21.	Have you ever been told you have had any heart complications?	___	___



A representative echo tracing of aortic, left atrial, and ventricular areas in a male subject.



A representative echo tracing of the ventricular area in a male subject.

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