INCIDENCE AND IMPLICATIONS OF ATYPICAL EXERCISE BLOOD PRESSURE RESPONSES IN ADULTS WITHOUT DIAGNOSED CORONARY HEART DISEASE

by

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(ABSTRACT)

Data were collected from the initial symptom-limited maximal exercise tests of 161 patients without diagnosed coronary heart disease (CHD). Subjects were grouped according to their systolic (SBP) and diastolic (DBP) blood pressure changes between the final two stages of exercise: S_{AT} = SBP $\downarrow$ > 4, no change > ±4, or $\uparrow \geq$ 30 mmHg; D_{AT} = DBP $\uparrow \geq$ 10 or above 110 mmHg; S_T = those SBP not categorized as S_{AT}; D_T = responses not defined as D_{AT}. Responders were categorized into groups as follows: S_TD_T; S_TD_{AT}; $S_{AT}D_T$; and $S_{AT}D_{AT}$. The atypical blood pressure response manifested in 41% of the subjects may have been influenced by a lack of age and sex restriction in the sample. The $S_{AT}D_T$, S_TD_{AT}, and $S_{AT}D_{AT}$ response groups represented 25%, 9%, and 8%, respectively. The large number of S_{AT} responders, a result of no SBP change, may be misleading. Increases in SBP are normally expected to taper off as maximum effort is approached. The groups did not differ physically, in health status, or in prescribed cardiovascular modifying medications. In predisposing CHD risk factors, the groups

did not differ in supine SBP, relative weight, or any plasma lipids. The significant ($p < .05$) group difference found in supine DBP was not confirmed in post-hoc analysis. The S_TD_{AT} responders tended to have higher supine DBP. At peak exercise, the groups did not differ in functional capacity, heart rate, endpoint symptoms, RPE, baseline ECG, ECG changes, arrhythmias, or intraventricular heart blocks. None of the groups were associated with excessively high or reduced blood pressures, ECG abnormalities, or arrhythmias at peak exercise. The results suggest that physician-referred, asymptomatic adults without diagnosed CHD entering medically supervised exercise programs may require minimal supervision during exercise testing, even in the presence of CHD risk factors or atypical exercise blood pressure responses.

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Chapter I

INTRODUCTION

Statement of the Problem

Coronary Heart Disease (CHD), the leading cause of death in the United States, is presently incurable. In most Western countries, atherosclerosis, one of the important precipitating factors in primary hypertension and CHD, begins during childhood, with 75% of males developing CHD to a significant degree by age 20 (Brooks & Fahey, 1974). Therefore, it is not surprising that a substantial modification of atherogenic habits in the American lifestyle has been recommended by the Intersociety Commission for Heart Disease Resources to reduce CHD morbidity/mortality in this country (Froelicher, Thompson, Longo, Triebwasser, & Lancaster, 1976; Stamler & Epstein, 1972). However, it appears practical to evaluate the screening techniques presently available for detection of subclinical or latent CHD to enhance the current trend toward lower incidence of CHD.

Since CHD is both widely prevalent and, in advanced stages, life-threatening, the purpose of a preventive health examination should be to identify medical conditions in their earliest stages with the hope that early intervention will prevent , or at least delay, disease evolution. Thus, one recent emphasis in "prevention-oriented" investigations

has been aimed at improving the specificity, predictive value, and sensitivity of screening techniques for detecting individuals at "high risk" for future CHD in asymptomatic populations. Knowledge of sensitivity and specificity is important in the development of screening programs for detection of individuals at increased risk for the development of CHD. Sensitivity refers to the ability of a given test to identify "true-positive" cases (positive test indication of CHD in a patient with CHD), while specificity refers to a test's ability to detect the "true-negatives" (no indication of CHD in a patient without CHD) (Epstein, 1967). In general, these two test characteristics show a reciprocal relationship, as one increases, the other decreases. Concurrently, predictive value, defined by a number of risk factors, indicates the probability that clinical disease will develop in an asymptomatic individual within a certain time (Epstein, 1967). Perhaps of greater importance in screening techniques is the "risk ratio," which incorporates sensitivity, specificity, and predictive value (Epstein, 1967). The risk ratio indicates the likelihood for clinical disease to develop in an individual with a certain level of risk factors compared to another who ranks lower in terms of these risk factors. Thus, the magnitude of this ratio reflects the extent to which preventive action is indicated in an individual.

Although the cause(s) of CHD are not known, a number of risk factors have long been recognized as valid and easily assessed indices for determining the liklihood of CHD or or its early development. Among these are resting hypertension, hyperlipidemia, smoking, obesity, heredity, and gender (Berglund, Ljungman, Hartford, Wilhelmsen, & Bjorntrop, 1982; Bruce, DeRouen, & Hossack, 1980; Jackson, Squires, Grimes, & Beard, 1983; Pader & Leiken, 1983). However, several researchers have determined that simple identification of these factors is especially inadequate when screening an asymptomatic population for latent CHD (Dahms, Giese, Nagle, & Corliss, 1978; Froelicher et al., 1976; Froelicher, Thomas, Pillow, & Lancaster, 1974; Gordon, Castelli, Hjortland, Kannel, & Dawber, 1977; Jackson, Squires, Grimes, & Beard, 1983). Therefore, realizing that it is undoubtedly advantageous, although more difficult, to detect those at high risk for CHD in the early asymptomatic stages, all methods of demonstrated value should be utilized to increase predictive value of screening techniques.

The graded exercise test (GXT) has frequently been used as a medical diagnostic test. More specifically, an abnormal electrocardiographic (ECG) response to exercise has been demonstrated to identify groups of men at high risk for CHD (Froelicher et al., 1974; 1976; Kattus, Jorgensen, Worden, & Alvaro, 1971). Unfortunately, an abnormal ECG

response does not precisely predict a future CHD event, nor does a normal exercise ECG response rule out this possibility.

Blood pressure responses to graded exercise have also been shown to have predictive validity with respect to future development of resting hypertension and CHD,even in the absence of other predisposing risk factors (Dahms, et al., 1978; Irving, Bruce, & DeRouen, 1977; Jackson et al., 1983; Sannerstedt, 1966; Wilson & Meyer, 1981). These researchers have found that some individuals exhibit a typical resting blood pressure, but an atypical blood pressure response to exercise. Several investigations (Dahms et al., 1978; Jackson et al., 1983; Wilson & Meyer, 1981) involving exercise blood pressure have shown an association between hyperresponses, especially in systolic pressure, and the development of resting hypertension within 2-5 years. In addition, Irving and associates (1977) have found that the absence of systolic blood pressure increases during exercise, probably due to left ventricular dysfunction, to be a more accurate predictor of CHD mortality than was S-T segment change in the stress electrocardiogram. Other researchers (Morris, Phillips, Jordan, & McHenry, 1978; Thompson & Kelemen, 1975) have reported that a decrease in exertional systolic blood pressure within exercise is a reliable sign of multiple

vessel coronary artery disease. In relation to diastolic pressure, Froelicher (1983) suggested that a rising diastolic blood pressure in treadmill exercise is indicative of CHD, while Irving et. al. (1977) found fourth and fifth phase Korotkoff sounds difficult to detect during exercise, thus diminishing the useful interpretation of exercise diastolic responses. Utilizing exercise blood pressure responses in combination with CHD risk factors to predict the future development of resting hypertension and CHD may prove invaluable in improving the clinical management of apparently healthy individuals by possibly preventing the occurrence of CHD.

Atypical blood pressure responses to exercise may not arouse similar levels of concern in all types of individuals or under all exercise conditions. Systolic hyporesponses may occur during exercise as a result of certain medications, left ventricular impairment, or critical coronary artery narrowing in the diseased individual, but may occur naturally in healthy individuals during maximal exhaustive exercise (Morris et al., 1978; Thompson & Kelemen, 1975). With respect to immediate test safety, these hyporesponses represent a major concern in the diseased individual, but relatively little concern in healthy individuals. Similarly, systolic hyperresponses to exercise in those with clinical heart disease have been

considered beneficial and associated with very low mortality risk (Irving et al., 1977), whereas hyperresponses indicate future development of hypertension and CHD in individuals without diagnosed disease (Dahms et al., 1978; Jackson et al., 1983; Wilson & Meyer, 1981). Therefore, when blood pressure changes to exercise are interpreted in conjunction with preliminary clinical findings plus accepted standards for test termination (ACSM, 1980), judgement of test safety may be improved.

Research Objectives

1. To examine the incidence of atypical systolic and diastolic blood pressure responses to exercise in a sample of patients without diagnosed coronary heart disease;

2. To examine the differences in predisposing CHD risk factors which are present in groups of typical versus atypical systolic and diastolic exercise blood pressure responders in a sample of patients without diagnosed coronary heart disease.

3. To examine the differences in appearance of clinical indicators of potentially hazardous acute myocardial dysfunction which are present in groups of typical versus atypical systolic and diastolic exercise blood pressure responders in a sample of patients without diagnosed coronary heart disease.

Significance of the Study

The initial purpose of this research was to determine the percentage of subjects without diagnosed CHD who might manifest an atypical response to graded exercise. The percentage of resting normo- and hypertensive subjects exhibiting an atypical blood pressure response during exercise would represent the magnitude of possible preclinical CHD existing in larger subgroups of apparently healthy individuals. A large percentage of atypical responses in otherwise apparently healthy individuals would exemplify the extent of the health problem and suggest the frequency with which such responders might be encountered in medically-based adult fitness programs.

The second purpose of this research was to view the screening parameters conducted prior to administration of the exercise test and examine their relationship to atypical blood pressure responses during the exercise test. A significant relationship between any of the screening variables and atypical blood pressure responses to exercise would prove valuable for adult fitness practitioners who attempt to identify those candidates for whom special testing and medical consultation might be needed. In a prevention/intervention setting, management of patients exhibiting any predisposing risk factor(s) found to significantly relate to atypical responses would include

additional emphasis on reduction of that particular risk factor(s). Test administrators would also benefit from the knowledge of which predisposing risk factors for CHD were highly associated with atypical blood pressure responses to exercise. With the ability to anticipate an atypical exercise responder prior to the testing, test administrators would improve the quality of test safety.

The final purpose of this research was to gain an improved understanding of the immediate implications of an atypical exercise blood pressure response in relation to myocardial function. This would result in a higher standard of test safety. An improved understanding of the relationship between atypical response to exercise and myocardial function would allow test administrators improved judgement when considering exercise continuation or termination.

Delimitations

The following delimitations were imposed:

1. The study was confined to the records of male and female adults without diagnosed coronary heart disease who participated in the Virginia Tech Intervention Exercise Program between February, 1978 and December, 1984.

2. The study was confined to participants classified as apparently healthy or with the sole cardiovascular medical diagnosis of hypertension.

3. The data for each subject were confined to the physician referral information and to the results from the initial graded exercise test (GXT) given at the participant program entry.

4. The study was confined to participants who had completed at least four stages of the exercise test on a motor-driven treadmill.

5. The study was confined to participants tested initially at the Virginia Tech Intervention Center laboratory on a Monday, Wednesday, or Friday morning between 7:00 and 8:00 a.m. using individualized protocols which increased every 2 minutes by 1-3 METs (predicted).

Limitations

The following factors were viewed as important constraints, limiting interpretation of the data:

1. The small number of subjects in the sample.

2. The wide variation in number of subjects in each group.

3. The possibility that some inconsistency occurred with respect to recording Phase IV or Phase V Korotkoff sounds as diastolic blood pressure, although the former was widely communicated to technicians as the "true" exercise diastolic value.

4. The inability to obtain complete data on all subjects.

5. The laboratory protocols differed between subjects according to the initial load and load increments.

Definitions and Symbols

Many of these definitions and symbols were previously devised by Ward (1985). There have been additions and minor changes made for the present investigation; these differences are denoted (*) in the list presented below:

DATE - the date of the initial GXT by month and year.

TEST SITE - site of the initial GXT; i.e., Virginia Tech or Montgomery County Hospital.

DEMOGRAPHICS -

AGE - age in years at the time of the initial GXT.

GENDER - gender of subject; i.e., male or female.

HT - height in inches.

HTCM - height in centimeters.

WTKG - weight in kilograms.

WTLBS - weight in pounds.

CLASS or SUBJECT CLASSIFICATION -

CARDIAC - any subject with diagnosed coronary artery disease, based upon the physician referral information.

NONCARDIAC - any subject with no diagnosed coronary artery disease, based upon the physician referral information.

CATEGORIES OF HEALTH/DISEASE - based upon the physician referral.

MI - myocardial infarction.

CABG - coronary artery bypass graft.

ANGINA - angina pectoris.

HYPERTEN - hypertension.

CLAUD - claudication.

ASCHD - asymptomatic coronary heart disease.

APPHLTY - apparently healthy.

BLOOD PRESSURE MEASUREMENTS -

DBP - diastolic blood pressure; the pressure, determined by auscultation, corresponding to fourth phase Korotkoff sound (point where the sound of the blood flow through the artery becomes muffled) during the relaxation phase of the left ventricle (ACSM, 1980; American Heart Association, 1979).

LDBP - DBP measured in the left arm.

RDBP - DBP measured in the right arm.

SBP - systolic blood pressure; the peak pressure, determined by auscultation, corresponding to first phase Korotkoff sound (the initial sound of blood flow through the artery) generated by the heart during the contraction of the left ventricle (ACSM, 1980).

LSBP - SBP measured in the left arm.

RSBP - SBP measured in the right arm.

RSU - SBP and DBP measured in the left and right arms with the subject's body at rest in a supine position.

RST - SBP and DBP measured in the left and right arms with the subject's body at rest in a standing position.

CRITERIA FOR ATYPICAL SBP RESPONSE IN EXERCISE - (ACSM, 1980; Froelicher, 1983; Ward, 1985).

DECSBP - decrease in SBP > 4 mmHg with increase in exercise intensity.

NOINCSBP - no change in SBP beyond ± 4 mmHg with increase in exercise intensity.

INCSBP - excessive increase in SBP ≥ 30 mmHg with increase in exercise intensity between the last two completed stages of exercise.

CRITERIA FOR ATYPICAL DBP RESPONSE IN EXERCISE - (ACSM, 1980; Froelicher, 1983; Ward, 1985).

HIDBP - excessively high DBP response ≥ 110 mmHg with increase in exercise intensity.

INCDBP - increase in DBP ≥ 10 mmHg between the last two completed stages of exercise with increase in exercise intensity.

EXERCISE BLOOD PRESSURE GROUPINGS - (based on above criteria)

TSTD or S_TD_T - typical systolic and typical diastolic blood pressure response to exercise.

TSATD or S_TD_{AT} - typical systolic blood pressure response to exercise, but an atypical diastolic pressure response to exercise.

ATSTD or $S_{AT}D_T$ - an atypical systolic blood pressure response to exercise, but a typical diastolic pressure response to exercise.

ATSATD or $S_{AT}D_{AT}$ - an atypical systolic and an atypical diastolic blood pressure response to exercise.

EXERCISE TESTING -

GXT - graded exercise test.

EXSTAGE or STAGE OF EXERCISE - the exercise bout performed at each load; usually lasted two minutes.

HYP - hyperventilation.

SUBMAX or SUBM* - submaximal exercise, i.e., load corresponding to 50-70% of peak exercise.

HRSUBMAX* - submaximal heart rate recorded during the EXSTAGE corresponding to 50-70% of maximal exercise.

MAX - maximal exercise.

HRMAX - highest achieved heart rate during the GXT.

IPE - immediate post-exercise; the period of time immediately following the cessation of the physical exercise performed by the subject; less than one minute post exercise.

REC - recovery; the period at one minute and beyond the termination of the physical exercise portion of the GXT.

MET - metabolic equivalent; the lowest MET level is the resting metabolic rate or the level of oxygen consumption needed to function at resting conditions. METs refer to the multiple of a given exercise level relative above the resting metabolic level; i.e., an activity equal to 5 METs requires five times the oxygen of rest (ACSM Prediction Tables, 1980).

FC - functional capacity; measurement of the aerobic exercise tolerance of an individual; determined in MET levels, predicted from testing guidelines provided by ACSM in 1980.

PEAK $\dot{V}O_2$ - the highest level of oxygen consumption achieved per minute during the GXT (Prediction formulas by ACSM, 1980).

RPE - rating of perceived exertion; a subjective response by the subject being testing as to his/her overall physical feelings during the GXT,

correlated with a rating scale of progressive clues of physical fatigue (Froelicher, 1983).

SIGNS AND SYMPTOMS OF EXERTIONAL INTOLERANCE -

CHDIS - chest discomfort sufficient to cause the subject to focus upon it.

DYSP - dyspnea or shortness of breath.

LFAT - leg fatigue.

GFAT - general fatigue.

OTHERSYM - any other sign or symptom of exertional intolerance.

ECG - electrocardiogram.

ECG MEASUREMENTS - (ACSM, 1980).

RAMP - amplitude of the R wave on an ECG.

JPT - J point; junction between the S wave and the S-T segment on an ECG.

ST - S-T segment; area of ECG tracing from the JPT and the beginning of the T wave on an ECG.

STDEV - S-T segment deviation.

ARRHYTHMIAS AND BLOCKS - (ACSM, 1980; Marriott, 1978).

SVA - supraventricular arrhythmias; electrical dysrhythmias occurring above the atrioventricular node.

SVACOND - SVA conditions:

Sinus bradycardia - regular sinus rhythm with heart rate less than 60 beats per minute; those subjects taking a beta-blocking drug were not included in this category.

Sinus tachycardia - regular sinus rhythm with heart rate greater than 100 beats per minute.

PAC - premature atrial contractions; condition in which the atria discharge before the arrival of the next sinus impulse.

Atrial flutter - condition in which the ventricles cannot respond as rapidly as the atrial stimulation, so there is a degree of block of atrial rate to ventricular response (atrial rate > 250 beats per minute).

Atrial fibrillation - condition in which atrial activity is represented by only fine fibrillatory waves due to totally disorganized atrial electrical activity.

AV Blocks - atrioventricular blocks; condition in which conduction of the electrical impulse is slowed abnormally through the atrioventricular node.

1st degree - a delay in passage of the electrical impulse through the AV node, so that the P-R

interval is prolonged, i.e., greater than 0.20 seconds.

2nd degree - some electrical impulses fail to pass through the ventricles, resulting in "dropped" beats.

3rd degree - complete blockage of electrical impulses from the atria to the ventricles, resulting in the atria and ventricles beating independently.

VA - ventricular arrhythmias; electrical dysrhythmias occurring below the atrioventricular node.

VACOND - VA conditions:

PVC - premature ventricular contraction; condition in which an ectopic focus in one of the ventricles initiates a contraction of the heart.

Ventricular flutter - condition in which the ventricular rate is much more rapid than the atrial rate, resulting in the inability to distinguish the atrial rhythm or rate on the ECG tracing.

IVB - intraventricular block; the delay of the conduction of the electrical impulse in either of the bundle branches resulting in the duration of the QRS being greater than or equal to 0.12

seconds.

IVBCOND - IVB conditions:

RBBB - right bundle branch block; condition in which the right bundle branch is blocked and cannot conduct the depolarization signal to the right ventricle.

LBBB - left bundle branch block; condition in which the septum and the left ventricle are both activated by the depolarizing right ventricle.

LAH - left anterior hemiblock; condition in which there is a blockage or delay of the electrical impulse in the left anterior fascicle of the left bundle branch.

LPH - left posterior hemiblock; condition in which there is a blockage or delay of the electrical impulse in the left posterior fascicle of the left bundle branch.

BLOOD CHEMISTRY -

CHOL - total serum cholesterol in the blood.

TRIG - triglyceride level in the blood.

HDL-C* - high-density lipoprotein in the blood.

TC/HDL-C* - the ratio of total serum cholesterol to high-density lipoprotein in the blood.

SKINFOLD ASSESSMENTS - (Males - Jackson and Pollock, 1978; females - Jackson, Pollock and Ward, 1980)

CHEST - chest skinfold site for males; diagonal fold one-half of the distance between the anterior-axillary line and the nipple.

ABD - abdomen skinfold site for males; vertical fold adjacent to and approximately two centimeters lateral to the umbilicus.

THIGH - thigh skinfold site for males and females; vertical fold on the anterior aspect of the thigh, midway between the hip and knee joints.

TRI - tricep skinfold site for females; vertical fold on the posterior midline of the upper arm (over the triceps muscle), halfway between the acromion and olecranon processes with the elbow extended and relaxed.

SUPRAIL - suprailiac skinfold site for females; diagonal fold on the crest of the ilium at the midaxillary line.

PFAT - percentage body fat.

DESWT - desirable weight; the desirable weight of each subject in pounds based on height, actual weight, gender and age (Goldbeck, 1984).

RELWT - relative weight; a ratio of desirable body weight to actual body weight (Goldbeck, 1984).

MED - medication(s): Action of each MED determined from physician referral and subject disease status. For listing of MED, see appendix; Table VII.

ANTIANG - antianginal medication.

ANTIHYP - antihypertensive medication.

ANTIARRY - antiarrhythmic medication.

DIG - digitalis medication.

ANTICOAG - anticoagulant medication.

ANTILIP - antilipidemic medication.

PYSCO - psychotropic medication.

OTHERMED - any other medication.

CMOD* - cardiovascular modifying medications.

SMOKE* - smoker or nonsmoker of cigarettes, cigars, or pipe.

Basic Assumptions

1. It is assumed that all GXTs were administered by trained technicians with physician supervision.

2. It is assumed that all data have been collected, measured and recorded accurately during the GXTs by trained technicians.

3. It is assumed that all diastolic blood pressures recorded were Korotkoff fourth phase diastolic pressure readings.

4. It is assumed that all blood pressure measurements were obtained from the left arm, unless otherwise stated.

5. It is assumed that all resting blood pressure measurements were made in the supine body position, unless otherwise stated.

6. It is assumed that all ECG tracings labelled "resting" refer to the supine body position, unless otherwise stated.

7. It is assumed that all signs and symptoms of exertional intolerance were recorded during the GXTs.

8. It is assumed that all peak $\dot{V}O_2$ values were measured using gas analysis or calculated based upon functional capacity determined by the speed and grade of the treadmill.

9. It is assumed that all subjects reported their smoking habits honestly and accurately.

10. It is assumed that no subjects had experience walking on a treadmill sufficient to exhibit a performance or physiological adaptation as a result of practice on the testing mode.

11. It is assumed that all testing equipment was standardized and calibrated at the time of each GXT.

12. It is assumed that all GXTs were symptom-limited.

Chapter II

REVIEW OF THE LITERATURE

Introduction

This review, divided into two major areas, progresses from a discussion of blood pressure and regulation at rest to that during exercise. The first section explains basic considerations involved in blood pressure, followed by environmental, physical, and nutritional influences. This section also includes mechanisms of blood pressure regulation in normo- and hypertensive adults.

The second section is devoted to blood pressure responses in dynamic exercise. Topics include: responses of healthy and hypertensive adults, influences of cardiovascular modifying medications and physical training, theories of regulation, significance of various systolic and diastolic responses, clinical standards associated with testing, and the use of exercise testing in detection of latent hypertension.

Blood Pressure and Its Regulation Under Non-Exercise Conditions

Basic Considerations

Blood pressure is the force exerted by the blood against the vessel wall (Guyton, 1977). Blood pressure is important because it is the force that makes blood flow through the circulatory system, which is necessary to

maintain homeostasis, i.e., life support of the entire body. The maximum pressure generated by the heart is during contraction of the left ventricle when blood is forced into the aorta and pulmonary artery. This is referred to as systolic pressure and provides an estimate of the work of the heart, as well as the strain against arterial walls. Systolic pressure is measured as the pressure required to occlude the brachial artery and corresponds to the first Korotkoff sound. The minimum blood pressure occurs late in ventricular relaxation and provides an indication of peripheral resistance imposed by the arterial system. This is referred to as diastolic pressure and corresponds to the Phase IV (muffling of sound) or Phase V (disappearance of sound) Korotkoff sounds. Although opinions of various researchers differ, the American Heart Association (AHA, 1979) and the World Health Association (Peart, 1977) have accepted the point of muffling as the definitive diastolic measurement, as it more closely correlates with direct measurements. Conversely, Smith and Kampine (1984) have stated that disappearance of sound is regarded as the best index of diastolic pressure in adults, while muffling is the best index in children.

Resting blood pressure is acknowledged to be a measurement which fluctuates physiologically in response to factors such as age, gender, stress, physical conditioning,

nutrition, environment, etc. Although blood pressure is actually a continuous variable representing a progression of cardiovascular health risk, arbitrary and fixed cutting points have been used to distinguish normotension from hypertension. The average resting systolic and diastolic blood pressure for young adults has been determined to be approximately 120 and 80 mmHg, respectively (Guyton, 1979; McArdle, Katch, & Katch, 1981). However, resting systolic values as high as 140 mmHg and diastolic values less than 90 have widely been considered normal (Boyer & Kasch, 1970; Dahms, Giese, Nagle, & Corliss, 1978; Hull, Wolthuis, Fischer, Triebwasser, Curtis, & McAfoose, 1981; Julius, 1977a; Kannel, Gordon, & Schwartz, 1971; Sparrow, Garvey, Rosner, & Thomas, 1982; Wilson & Meyer, 1981). Hypertension, or high arterial blood pressure, has been arbitrarily defined by many authorities as a systolic pressure of 160 mmHg or greater and/or a diastolic pressure exceeding 95 mmHg (Boyer & Kasch, 1970; Chewning, 1982; Kannel et al., 1971; Kannel, Sorlie, & Gordon; 1980; Simon, Levenson, & Safar, 1982; Sparrow et al., 1982). Blood pressures intermediately between 140/90 mmHg and 160/95 mmHg have been designated as "borderline" hypertension (Chewning, 1982; Kannel et al., 1971; Kannel et al., 1980; Julius, 1977b). In the past the term "labile" hypertension has been confused with the definition of borderline hypertension

(Horan, Kennedy, & Padgett, 1981; Julius, 1977a; Julius & Schork, 1971). However, the World Health Organization has established distinguishing criteria for labile hypertension as arterial blood pressure sometimes below and sometimes above 140/90 mmHg (Horan et al., 1981). Several researchers have suggested the need to use lower criterion measures for women (Irving, Bruce, & DeRouen, 1977; Jackson, Squires, Grimes, & Beard, 1983; Sannerstedt, 1981; Wolthuis, Froelicher, Fischer, & Triebwasser, 1977). Furthermore, other researchers have found that such blood pressure definitions based on systolic and diastolic cutting points that do not change (increase) with age are clinically unreasonable (Julius, 1977a; Julius & Schork, 1971; Kannel et al., 1971).

Environmental Influences

Numerous environmental factors are known to have an affect on blood pressure at rest. Resting blood pressure may rise simply due to the anxiety associated with the measurement (Stegemann, 1981). Resting values are influenced by altitude and temperature, as well. The effects of gravity during static standing and tilt table test are particularly distressing (Brooks & Fahey, 1984). In response to an inverted position on the tilt table, heart rate increases and systolic blood pressure falls until the subject faints.

A recent investigation involving 4,634 men and women established a seasonal effect on systolic and diastolic pressures (Khaw, Barrett-Conner & Suarez, 1984). There was a significant difference between the highest values in winter and spring and the lowest values in summer which were not associated with ambient temperature. These results were similar to those of Brennan, Greenberg, Miall, and Thompson (cited by Khaw et al., 1984) who also reported the lowest pressures in the summer months. Khaw et al. (1984) also noted research by Hata and several other investigators in which a seasonal variation was observed in 20 male hypertensive patients, although no variation was exhibited by the 16 normotensive subjects.

In a study limited to 10 subjects, Reilly, Robinson, and Minors (1984) recently examined blood pressure at various times of day. Blood pressures were observed at 3:00 a.m. and p.m. and 9:00 a.m. and p.m. Although not significant, the minimum resting systolic pressure average was observed to be 122 mmHg at 3:00 a.m., while the maximum systolic pressure averaged 129 mmHg at 9:00 p.m. Resting diastolic changes of 85 mmHg at 3:00 a.m. to 76 mmHg at 3:00 p.m. were significantly different. In spite of the fact that exercise blood pressure measurements were obtained, only resting and recovery measurements were disclosed. The recovery responses revealed no significant time-of-day

effect. Although evidence in this investigation proved inconclusive, studies by Millar-Craig, Bishop, and Raftery and Smith and Malyi (cited by Reilly et al., 1984) have shown a circadian variation in systolic and diastolic pressures.

Physical and Nutritional Influences

Other factors known to influence resting blood pressure are age, gender, body weight, obesity, and physical fitness. Longitudinal research has revealed a tendency for average blood pressures to increase with age. In a newborn baby, average systolic and diastolic pressures average 90/55 mmHg (Guyton, 1979). This pressure usually reaches 120/80 mmHg by young adulthood and increases to 150/90 mmHg in old age (Guyton, 1979). Julius and Schork (1971) stated that if patients with borderline hypertension were to stay at the same level, their blood pressure over a period of years would actually be less than the remainder of the population. Furthermore, Thomas (cited by Julius & Schork, 1971) observed the blood pressures of 8-21 year old subjects and found that higher initial readings, even within the normotensive range, were predictive of future steeper blood pressure increases. Conversely, Khaw et al. (1984) observed a significant decrease in blood pressure over a period of two years which could not be attributed to antihypertensive medications. This unique response could not be explained,

but may be related to reduced mortality as a result of hypertension-related diseases over the past 20 years.

There is evidence that women tend to have lower average resting blood pressures than men. Irving et al. (1977) found that healthy women had significantly lower average systolic pressures than healthy men (119 mmHg and 123 mmHg, respectively). They also found that in men, there was no appreciable change in systolic pressure with age, whereas women showed an increase with age. However, Julius and Schork (1971) reported that prevalence of borderline hypertension among young and middle-aged females is lower than in males.

In addition to age and gender, resting blood pressure is influenced by body weight. This relationship is enhanced by a positive relationship between body fat and blood pressure. In a random sampling of untreated 49-year-old men, blood pressure increased linearly with body weight across the entire range of pressue (Berglund, Ljungman, Hartford, Wilhelmsen, & Bjorntorp, 1982). However, body fat and fat cell size, but not fat cell number, increased as blood pressure increased only in the normotensive subjects. The increase in fat cell size was associated with metabolic imbalances, i.e., impaired glucose tolerance, hyperinsulinemia, and sodium intake. The relationship between blood pressure and body weight/obesity was further

supported by the results of an investigation by Sparrow et al. (1982), in which subscapular skinfold thickness and body mass index were positively related to blood pressure.

The relationship between resting blood pressure and physical fitness, defined as physical work capacity on a treadmill, was examined in 4,820 men and 1,219 women (Blair, Goodyear, Gibbons, & Cooper, 1984). Healthy normotensive adults were followed-up for an average of four years for the development of hypertension. Two hundred and forty subjects developed hypertension within one to 12 years. A negative relationship between initial physical fitness level and development of hypertension was revealed. Subsequently, a second fitness test was given to 1,303 of the subjects, including 38 subjects who had developed hypertension during the follow-up period. Results revealed the incidence of hypertension was 18 per 1,000 in the high fitness group, compared to 32 per 1,000 in the low fitness group. Therefore, Blair et al. (1984) concluded that physical fitness is significantly associated with hypertension risk. They further concluded that patients in the normal blood pressure range of 120 to 129 mmHg systolic and 81 to 84 mmHg diastolic had almost a threefold increase in hypertension risk over those with lower baseline pressures.

Control of Blood Pressure in Normotension

In the fifth edition of the Textbook of Medical

Physiology, Guyton (1976) described mechanisms by which blood pressure is normally maintained. Underlying the mechanisms of blood pressure is the concept that arterial pressure is a product of cardiac output and total peripheral resistance. Blood pressure is maintained by combined reciprocal interactions between various short-term and long-term regulatory systems influencing cardiac output and peripheral resistance. The short-term, quickly-responding controls mediated through the nervous system include baroreceptor reflexes, atrial and pulmonary artery reflexes, central nervous system ischemic response, chemoreceptor reflexes, and skeletal muscle activity. Baroreceptor reflexes consist of afferent fibers which detect stretch/pressure changes, especially increases in pressure, in the aortic arch and internal carotid arteries. Signals are transmitted to the vasomotor center of the medulla where impulses inhibit the vasoconstrictor center and excite the vagal center. This results in vasodilation, decreased cardiac rate, and decreased strength of contraction. Similar to the baroreceptors, both the atria and the pulmonary arteries have stretch receptors called low pressure receptors. The pulmonary artery receptors operate almost identically the same as the baroreceptors, while the atrial receptors operate somewhat differently. The atrial receptors react to increases in pressure by causing reflex

vasodilation leading to increased capillary pressure and increased filtering of circulatory fluid into the tissues. These atrial stimuli also increase pressure in the kidneys, thus decreasing antidiuretic hormone output, which leads to rapid loss of fluid into the urine.

In contrast, the immediate action of the central nervous system (CNS) ischemic response occurs when arterial pressure falls very low, resulting in high concentrations of carbon dioxide and low concentrations of oxygen in the brain. This response is one of the most powerful of all activators and results in vasoconstriction of the peripheral vessels and increased heart rate. Chemoreceptors, located in the aortic arch and carotid arteries, are also sensitive to reduced oxygen, along with carbon dioxide and hydrogen ion excess. The chemoreceptor reflex reacts to elevated pressure much the same way as the CNS ischemic response, except it is not as powerful. In addition, whenever the sympathetic vasoconstrictor system is stimulated, the vasomotor center transmits impulses simultaneously through the skeletal muscles. This increases muscle tone and the constriction compresses vessel reservoirs, helping to translocate blood away from the reservoirs toward the heart.

In addition to the rapidly acting nervous system mechanisms for control of arterial pressure, there are hormonal mechanisms that also provide either rapid or

moderately rapid control. These mechanisms include norepinephrine-epinephrine, renin-angiotensin, and vasopressin vasoconstrictor mechanisms. The norepinephrine-epinephrine hormones are released into the blood from the adrenal medulla following sympathetic nervous systen stimulation. These hormones cause essentially the same effects as direct sympathetic stimulation, i.e., excitation of the heart and constriction of most blood vessels, but they last longer and can reach parts of the circulation that have no sympathetic nervous supply. The hormone, angiotensin II, is the most potent vasoconstrictor known, and although it is slower to act than nervous reflexes or the norepinephrine-epinephrine system, it has a longer duration of action. When arterial pressure falls too low, the kidneys secrete renin, the renin causes formation of angiotensin II, and this in turn causes peripheral vasoconstriction. In addition, vasopressin, secreted from hypothalamus, also acts as a vasoconstrictor when arterial pressure becomes too low, but has less power than angiotensin II.

Beyond the nervous and hormonal mechanisms for rapid to moderately rapid control of arterial pressure, two physical mechanisms are known to function with intermediate rapidity. These include mechanisms for capillary fluid shift and vascular stress-relaxation. When arterial pressure rises,

capillary pressure also rises, and if the pressure rises too high, fluid escapes through the capillary membrane into the interstitial spaces. This capillary fluid shift causes reductions in blood volume and a return of pressure to normal. The mechanism of vascular stress-relaxation simply refers to vessel adaptation to a new size by relaxing during high pressures and tightening during lower pressures.

The long-term mechanisms that control baseline pressure consist almost entirely of systems to adjust body fluid volumes, and the kidneys are the most important of all parts of the body in this long-term regulation. When arterial pressure rises too high, the kidneys excrete increased quanities of water and sodium, which decreases fluid volume and normalizes pressure. Conversely, angiotensin directly effects the kidneys to cause decreased excretion of water and sodium. Angiotensin further stimulates the secretion of aldosterone by the adrenal cortex, and this hormone in turn acts on the kidneys to cause decreased excretion of water and sodium. Vasopressin, also known as antidiuretic hormone, also acts on the kidneys to decrease water excretion. Rate of intake of sodium and potassium also affects long-term regulation of arterial pressure.

Control of Blood Pressure in Essential Hypertension

The exact mechanisms involved in essential hypertension are uncertain. However, it is known that blood volume and

cardiac output are normal in essential hypertension, while total peripheral resistance is greatly increased (Frohlich, 1977; Julius, 1977a). Because of this high total peripheral resistance, the majority of hypertension investigators sought for a vasoconstrictor agent underlying the basis of hypertension. For many years it was suggested that the renin-angiotensin system was responsible for hypertension. However, in studies cited by Guyton (1980), it has recently been determined that essential hypertension is associated with normal or low levels of circulatory renin, rather than high renin levels.

More recently, investigators cited by Guyton (1980) have found evidence relating abnormal renal function to essential hypertension. The most important evidence is that the kidneys can excrete water and electrolytes at normal rates only when arterial pressure is at a hypertensive level. An ever increasing pressure is needed to maintain normal glomerular filtration rate to compensate for reduced renal blood flow and increased renal vascular resistance. This results in ever increasing levels of renal damage and dysfunction over a period of years.

Blood Pressure and Its Regulation Under Exercise Conditions

Responses of Healthy Adults

Normally, there is a linear rise in the systolic blood

pressure with gradual increases in dynamic exercise (Froelicher, 1983; Sannerstedt, 1981). However, the normal diastolic pressure response to dynamic exercise is no change or a slight decrease (less than 10 mmHg) followed by a small decrease (less than 4 mmHG) during the recovery phase (Bruce, Gey, Cooper, Fischer, & Pererson, 1974; Froelicher, 1983; Sannerstedt, 1981). The increases in systolic pressure are expected to taper off as maximum effort is approached, while diastolic pressure remains constant or falls slightly (Bruce et al., 1974; Wolthuis, 1977). Sannerstedt (1981) reports that his own invasive studies using the bicycle ergometer correspond well with noninvasive research by Irving et al. (1977) using maximal treadmill exercise. These investigators found that approximately 230 mmHg represents the highest level for systolic blood pressure normally seen in response to heavy, near-maximal physical exercise in healthy men, whereas 100 mmHg represented the level above which diastolic pressure will not normally exceed, even under conditions of heavy, dynamic exercise. The figures for maximal systolic pressure were observed to be lower, 200-209 mmHg, for women. Froelicher (1983) reported that systolic pressure can rise above 280 mmHg with no clinical implications or complications, while diastolic Korotkoff sounds can sometimes be heard all the way to zero in healthy young men.

The research reported by Sannerstedt (1981) revealed that maximal values for systolic pressure tended to be somewhat higher with increasing age, which was also evidenced in the noninvasive research by Wolthuis et al. (1977). The research by Wolthuis et al. (1977) involved three progressive age decades (25-34, 35-44, and 45-54). Maximal systolic pressure increased slightly across the three age groups (164-212, 164-214, and 162-216, respectively), as did maximum diastolic pressure (60-90, 64-94, 68-96, respectively). It should be noted that these investigators utilized Phase V Korotkoff sound as the diastolic endpoint.

Bruce et al. (1974) reported average resting blood pressure of healthy men to be 123 ± 13 mmHg systolic and 77 ± 10 mmHg diastolic. Considering that he found increases of 62 ± 19 mmHg systolic at maximal exercise, average maximal systolic pressure approximated 185 mmHg. This corresponds closely to average values of 200 mmHg observed by Sannerstedt (1981) and 187 mmHg obvserved by Irving et al. (1977). Bruce et al. (1974) found healthy men to experience a slight reduction in mean diastolic pressure at maximal exercise compared with their pressures at rest.

Immediately following exercise termination, blood pressure, especially systolic, is expected to decrease, usually reaching its initial resting level in one to three

minutes (Sannerstedt, 1981). As expected, Wolthuis et al. (1977) witnessed a decrease at recovery minute two and a further decline at minute five. This approximated average pre-exercise values, with systolic pressure slightly above resting and diastolic pressure slightly below resting values. Froelicher (1983) reported that following test termination, approximately 10 percent of people tested will show a decline in systolic pressure as a result of peripheral pooling.

Responses of Hypertensive Adults

In general, investigators have found varied blood pressure responses to dynamic exercise among hypertensive adults. Hypertensive adults who are young or only mildly hypertensive tend to exhibit exercise blood presure responses similar to those of normotensive adults, whereas hypertensive adults who are older or have considerable hypertension tend to exhibit a greater rise in blood pressure than normotensive adults (Amery, Julius, Whitlock, & Conway, 1967; Aoki, Kato, Mochizuki, Kawaguchi, & Yamamoto, 1982; Sannerstedt, 1966).

Aoki et al. (1982) studied only recovery blood pressure responses following the master's two-step exercise test in normo- and hypertensive subjects. They defined diastolic pressure as either Phase IV or Phase V Korotkoff sounds. Resting systolic and diastolic pressures were significantly

higher in the hypertensive subjects than in the normotensive subjects. Compared to the responses of normotensive subjects following the two-step exercise, a great rise in systolic and diastolic pressure was exhibited in the "pre-hypertensive" (resting pressures less than 140/90 mmHg) and "borderline" hypertensive (resting systolic pressure 140-159 mmHg and/or diastolic pressure 90-99 mmHg) stages of essential hypertension. Conversely, only a small rise was observed in the established stage (systolic pressure ≥ 160 mmHg and/or diastolic pressure ≥ 100 mmHg). The hypertensive subjects also exhibited an increase in diastolic pressure following the two-step exercise, compared to a decline in the normotensive subjects. The return of systolic pressure to resting level was delayed in the "borderline" and "established" hypertension groups as compared to normotensive subjects. The return of diastolic pressure to resting level was also delayed in the hypertensive subjects. Prior to this investigation, Sannerstedt (1981) openly criticized the practice of using recovery blood pressures to evaluate exercise responses, stating that following exercise termination, blood pressures immediately begin to fall, usually returning to resting levels within one to three minutes. Therefore, a true conception of exercise responses must be acquired during exercise, as other procedures may yield erroneous values.

In a study using bicycle ergometer exercise, Sannerstedt (1966) observed a normal rise in systolic blood pressure with exercise in mildly hypertensive subjects, but patients with higher stages of hypertension exhibited significantly steeper systolic pressures than normotensive controls. Exercise systolic blood pressures in hypertensive subjects ranged from approximately 250 mmHg in the mildly hypertensive subjects, to a maximum of 331 mmHg in subjects with advanced hypertension. As in normotensive controls, hypertensive patients of all stages exhibited a slight to moderate rise in diastolic pressure during dynamic exercise. Therefore, patients with high resting pressures sometimes exhibited very high diastolic values of 150 to 170 mmHg.

Similar results were observed in a previous study involving bicycle ergometer exercise (Amery et al., 1967), in which subjects were divided into three age groups (19-34, 35-49, and 50-69 years, respectively). In the youngest group, changes in systolic pressure were not greater than that seen in the normotensive subjects of the same age. However, the systolic pressures in the two older groups revealed a greater rise than in the normotensive subjects of the same age. The changes in diastolic pressure with exercise in hypertensive subjects were small and did not differ from the normotensive subjects. Similarly, Wolthuis et al. (1977) mentioned a 1975 study by Froelicher,

Thompson, Yanowitz, and Lancaster involving treadmill exercise. Systolic blood pressure in hypertensive subjects was observed to rise above the illustrated limits in this investigation of aircrewmen.

Medication Influences

Koch-Weser (1973) detailed many of the medications known to influence blood pressure used in antihypertensive therapy. Patients with essential hypertension vary in the extent to which cardiac output, peripheral resistance, blood volume, and the functional state of their renin-angiotensin-aldosterone and sympathetic nervous systems contribute to their hypertension. Therefore, with the knowledge of the specific mechanisms of action in given antihypertensive medications, combined with characterization of the hypertensive mechanisms operating in individuals, prediction of the most effective therapeutic medication(s) is theoretically possible.

In addition to medication therapy, treatment of hypertension often includes regular dynamic exercise. Thus, a brief review of the pharmacodynamics of important antihypertensive medications is followed by a discussion of their effects on blood pressure response in exercise.

Three major groups of antihypertensive medications (i.e., diuretics, vasodialtors, and beta-adrenergic inhibitors) are reviewed. Diuretic medications, such as

hydrochlorothiazide, function by decreasing sodium, potassium, and fluid volume, and eventually peripheral vascular resistance. Although the mechanisms for these actions are not yet fully understood, it may involve direct action on vascular smooth muscle or changes in electrolyte concentration or fluid content in the arteriolar wall that affect its thickness, tone, or pressor responsiveness. It is known that plasma renin activity increases and extracellular fluid and blood volume are initially reduced. Maximum doses of diuretic agents have resulted in pressure reductions of only as much as 15 mmHg. However, these agents are still useful in treatment of severely hypertensive patients who require additional medications, since most other antihypertensive medications result in gradual sodium retention and expansion of blood volume and extracellular fluid.

Vasodilating medications, such as hydralazine, diazoxide, and nitroprusside, relax arteriolar smooth muscle, resulting in decreased total peripheral resistance. Although hydralazine is less effective than diazoxide or nitroprusside, it has the potential to reduce total peripheral resistance by as much as 50%. These medications have limited long-term effectiveness in reducing arterial pressure because: (1) there is a reflex enhancement in sympathetic activity resulting in increased heart rate and

cardiac output; (2) vasodilators result in increased plasma renin activity in hypertensive patients, probably due to an increased sympathetic discharge to the kidney; and (3) there is retention of sodium and water resulting in increased extracellular fluid and blood volume. Concomitant administration of propranolol can prevent the first two undesirable responses, and diuretics can prevent the third. This combination of medications can control all stages of hypertension. Simon et al. (1982) recommended the use of nitroprusside in the management of systolic pressure in the elderly hypertensive patient.

In general, beta-adrenergic inhibitors, such as propranolol, quanethidine, reserpine, and methyldopa, interfere with sympathetic transmission to the heart. Propranolol reduces pressure by (1) decreasing secretion of renin by the kidney, (2) decreasing myocardial contractility, heart rate, and cardiac output by reducing sympathetic stimulation to the heart, and perhaps by (3) cholinergic neurons in the central nervous system. Simon et al. (1982) recommended the use of propranolol in younger systolic hypertensive patients. Guanethidine reduces pressure by interfering with neurotransmission at adrenergic nerve endings, resulting in decreased arteriolar and venous tone and depressed myocardial function. The effect of this beta-blocking medication is more pronounced during increased

sympathetic discharge, tending to cause orthostatic and exercise hypotension. Reserpine reduces peripheral vascular resistance through diminished sympathetic stimulation of the heart and vessels, due to both a depletion of stored norepinephrine in the sympathetic nerve endings, and an action on the hypothalamus and vasomotor center. In sufficient dosages, this medication can decrease heart rate, myocardial contractility, and cardiac output. The effects of usual dosages of reserpine rarely exceed 10 mmHg, and orthostatic and exercise hypotension are uncommon. Methyldopa is known to decrease peripheral arteriolar resistance, while having little or no effect on heart rate and cardiac output. The mechanism if this action is uncertain but may result from a central nervous system mechanism that decreases sympathetic vasomotor conductance. There is variability in the hypotensive responses of methyldopa, ranging from no effect to 25 mmHg. Although the postural variation of pressure reduction is much less in methyldopa than in guanethidine, side effects of methyldopa on the central nervous system limit its therapeutic value. When used for long periods without a diuretic agent, all of these beta-adrenergic inhibitors cause sodium and fluid retention and expansion of plasma and extracellular fluid volume, thereby losing their hypotensive effect.

Blood pressure modificaton in response to dynamic

exercise may vary considerably from one type of antihypertensive medication to another, and also between various groups of patients depending on such factors as age, and stage of hypertension. Sannerstedt (1981) identified four different types of medically modified responses to dynamic exercise: (1) Resting blood pressure is lowered to the same extent as exercise pressure. In this case, the rise in exercise blood pressure is equal to that prior to treatment, resulting in a higher percentage of pressue increase during treatment. This is the most common response and is usually observed during monotherapy with medications such as thiazides or methyldopa. (2) The hypotensive effect becomes more prominent during exercise than at rest. The increase in exercise blood pressure is less than before treatment, resulting in a percent rise in pressure equal to or less than that observed before treatment. This response is associated with established hypertension which usually requires combination therapy, e.g., a vasodilator plus a thiazide or beta-blocker, or a triple regimine using agents from all three groups. (3) Blood pressure increases in exercise are absent, and instead, decreasing pressure response may be exhibited. This response is associated with treatment of potent beta-blocking medications such as guanethidine, sometimes leading to attacks of syncope during dynamic exercise. This response may also appear in elderly

patients receiving excessive diuretics or in patients with advanced myocardial necrosis. (4) The hypotensive effect of treatment becomes evident only during exercise. In this case, blood pressure is unchanged at rest, but exhibits lower values during exercise than without treatment. This is an occasional response observed during monotherapy with beta-blockers, especially in young patients with near-normal resting blood pressures who, in the absence of treatment, react hypertensively to exercise. In patients with asymptomatic, uncomplicated hypertension, Allen, Craven, Rosenbloom, and Sutton (1984) found that beta-blockers impair exercise performance, especially aerobic and endurance exercise. Beta-blockers also make it more difficult for these patients to improve their performance with training. However, in patients with CHD and angina, exercise capacity often increases substantially during the initial conditioning period, followed by an impaired capacity for endurance training with beta-blockers. Allen et al. (1984) suggested that metabolic limitation may be reduced by administration of more cardioselective medications, e.g., metoprolol impairs lipolysis and glycogen breakdown less than does propranolol, resulting in less fatigue during submaximal endurance exercise.

Physical Training Influences

Numerous studies have been conducted to test the

advantages and disadvantages of physical training on normotensive individuals, and hypertensive and CHD patients, yet current conflicting data fall short of proving that an increase in habitual physical activity is actually beneficial for all types of blood pressures (Fox & Naughton, 1972; Tipton, 1984). Fox & Naughton (1972) contend that the benefits may be as much or more in the area of an improved quality of life as in the extension of life. In "Exercise, Training, and Hypertension," Tipton reviewed numerous studies and concluded that there is little or no effect on blood pressure as a result of endurance training in normotensive individuals. However, in hypertensive and older individuals, there appears to be a more beneficial effect on resting blood pressure.

Similarly, Sannerstedt (1981) has stated that fitness improvement through physical training has proven to be effective in the treatment of CHD patients, and suggested that physical training may also prove worthwhile in treating hypertensive patients. He claimed that training lowers mean arterial blood pressure, heart rate, and cardiac output. The results of a previous investigation by Boyer and Kasch (1970) were consistent with his claim only with respect to blood pressure.

Boyer and Kasch (1970) conducted an exercise study on 23 hypertensive and 22 normotensive middle-aged men. Two

days per week the men participated in a controlled exercise program involving a 15-20 minute warm-up, followed by 30-35 minutes of heart rate-controlled walk-jog sequences. The program spanned six months, with an exercise heart rate intensity of 60% of maximum for the first three months, and 70% the last three months. A 11.8 mmHg drop in mean diastolic pressure and a 13.5 mmHg drop in mean systolic pressure occurred in the hypertensive group. The normotensive group exhibited a mean diastolic decrease of 6 mmHg, but no significant change in mean systolic pressure. It was concluded that hypertensive patients may be trained without undue risk and that interval exercise is of supplemental value in the therapy of hypertensive middle-aged men.

Likewise, Brooks & Fahey (1984) state that endurance training tends to reduce resting and submaximal exercise systolic, diastolic, and mean blood pressures. Although the mechanism for the reduction is uncertain, Sannerstedt (1981) noted that the response pattern with training closely mimics the hemodynamic alterations achieved through sustained administration of beta-blocking agents. He suggested that these training-induced changes probably indicate lowered adrenergic tone.

Shephard (1984) stated that during vigorous physical activity the risk of a cardiac emergency, both in patients

with known CHD and in apparently healthy middle-aged individuals, is 5-10 times higher than during quiescence. Nevertheless, he advocated a gentle progression of physical activity for both supposedly healthy and "postcoronary" patients, claiming that a cardiac catastrophe is a very rare event and physical activity probably improves long-term outlooks for both types of individuals. However, Tipton (1984) emphasized the importance of considering the particular exercise blood pressure response of each individual patient in setting an exercise prescription. He advised lower training intensities for the hypertensive patients compared to normotensive adults.

Theories of Control

When exercise is initiated, possibly sooner, the cerebral cortex inhibits parasympathetic activity and increases sympathetic impulses, resulting in a higher heart rate and increased contractile force (Astrand, 1977). Right away, impulses from the central nervous system, transmitted by sympathetic cholinergic vasodilator fibers, dilate the arterioles in the muscles and increase blood flow. Conversely, sympathetic adrenergic vasoconstrictor fibers act on the skin and abdominal vessels, reducing blood flow to these tissues. The redistribution of blood may result in no decrease, or only a slight decrease, in the systolic blood pressure during initial exercise, despite the marked

dilatation of arterioles in the muscles. The veins are constricted passively and through constrictor fiber activity. This constriction of veins, combined with the pumping action of the working muscles and forced respiratory movements, facilitates return of the blood to the heart and increased cardiac output.

In active muscles, the effect of increased metabolism is a local change in pH and interstitial fluid composition, causing sympathetic vasodilation of even more arterioles and capillaries. This local control is the most important factor in achievement of adequate blood supply to active muscles, because mechanisms controlling muscle vasodilation are more responsive to muscular exercise than to hypoxia. This response is due to the high extracellular concentration of potassium ions and other metabolites during exercise, which accounts for a major part of the vascular dilation accompanying muscle activity. Oxygen deficient blood further strengthens these vasodilating effects. In the inactive muscles, the sympathetic vasodilator fibers are inactive, and hormonal activity contributes to vasoconstriction in these muscles.

As exercise proceeds, the blood vessels of the skin dilate, to facilitate transport of produced heat to the body surface. Indirectly, sympathetic impulses are the cause of this vasodilation, with the hypothalamus guiding the

impulses. However, adverse conditions for heat dissipation are expected to induce greater competition for blood flow between the active muscles and skin, potentially resulting in systemic acidosis, complicated by further increased demands on the heart (Mole & Coulson, 1985). Charles Wade (1984) suggested that vasopressin, involved in the maintenance of blood pressure during hemorrhage and dehydration, may also contribute to the maintenance and/or elevation of pressure during exercise, because individuals often undergo voluntary dehydration and reduction of plasma volume during prolonged exercise.

Although the specific mechanisms by which exercise blood pressure is regulated are currently uncertain, a number of hypotheses have been presented. In a presentation delivered at an annual meeting of the American College of Sports Medicine, Rowell (1980) discussed the possibility of involvement of multiple, redundant regulatory systems in exercise blood pressure mediation. After analyzing each hypothesized mechanism separately, he suggested that a "central command," acting through cortical and spinal cord motor systems, could serve to set basic patterns of effector activity, which in turn are modulated by baroreceptors, muscle mechanoreceptors, and muscle chemoreceptors. He further suggested that the interaction of multiple systems in exercise blood pressure response may mask the important

role of any individual system. These multiple systems may also mask the view held by Amery, Fagard, Lijnen, and Reybrouck (1981) that angiotensin II contributes to a rise in systolic pressure during heavy exercise, by preventing further vasodilation.

More recently, investigators have found muscle fiber type to be related to blood pressure regulation (Karlsson, Dlin, Tesch, & Kaijser, 1983). Slow-twitch fibers are surrounded by more capillaries and favor aerobic metabolic processes, whereas fast-twitch fibers favor anaerobic processes. Blood pressure responses were found to be inversely related to the percentage of slow-twitch fibers in portions of the leg, whereas local blood flow in the leg was shown to be positively related to the percentage of slow-twitch fibers. Karlsson et al. (1983) also found a higher level of sensitivity for sympathetic nervous activity in subjects rich in slow-twitch fibers. In addition, they observed a higher percentage of fast-twitch fibers in patients with essential hypertension. Karlsson et al. (1983) further cited studies by (1) Mitchell, Reardon, McCloskley, and Wildenthal and (2) Frisk-Holmberg, Essen, Fredricksson, and Strom in which higher blood pressure responses were observed in felines and men with muscles rich in fast-twitch fibers than those rich in slow-twitch muscle fibers. Although these results involved static exercise,

they were believed to be applicable to dynamic exercise. These studies lend support to an earlier concept revealed by Shepherd, Blomqvist, Lind, Mitchell, and Saltin (cited by Karlsson et al., 1983) that ergoreceptors could be involved in regulating the central circulation. Ergoreceptors function by means of afferents from the active muscle to the vasomotor center, followed by activation of the central circulation from vasomotor center sympathetic impulses. Thus, the impact of the periphery, expressed as muscle fiber composition, should be considered in addition to the concept of "central command" when discussing the regulation of peripheral and central circulation.

Significance of Various Systolic Blood Pressure Responses

Investigators have witnessed several blood pressure responses to dynamic exercise which are inconsistent with the typical responses exhibited by apparently healthy adults, i.e., a linear rise in systolic pressure with increases in exercise intensity and very little, if any, rise in diastolic pressure. These atypical blood pressure responses have been associated with various types of cardiovascular distress and varying degrees of cardiovascular disease.

Excessive rise in systolic blood pressure. The atypical responses associated with systolic blood pressure include an excessive increase in pressure, failure of

pressure to increase with gradual increases in exercise intensity, and a decrease in pressure with increases in exercise intensity. The least distressing atypical systolic response appears to be an excessive increase in pressure. Hyperresponses to exercise have been reported to occur in hypertensive subjects (Amery et al., 1967; Irving et al., 1977; Sannerstedt, 1966). Amery et al. (1967) found that, in general, older hypertensive patients demonstrated a greater rise in systolic pressure with exercise than normotensive subjects. This was attributed to sclerosis of the large blood vessels. Amery et al. (1967) further cited a study by Malmcrona, Cramer, and Varnauskas in which excessive increases in systolic pressure during exercise were noted in patients with coronary artery disease. Aoki et al. (1982) speculated that a greater sensitivity in the intrinsic factors of arterial smooth muscle to contraction in essential hypertenson may be a responsible factor in hyperresponses. Excessive increases in systolic blood pressure during exercise have resulted in considerable concern among test administrators; however, Irving et al. (1977) concluded that men with clinical heart disease whose increase in systolic pressure with exercise exceeded the average for normal men were at very low mortality risk. Consistently, Froelicher, Thompson, Yanowitz, and Lancaster (cited by Wolthuis et al., 1977) reported in 1975 that

although systolic pressure in hypertensive patients has been observed to rise above illustrated limits, such elevations were not associated with any complications or ominous implications. Although this atypical response seems to pose no immediate danger to apparently healthy individuals, an excessive increase in pressure has been implicated as an indicator for detecting individuals predisposed to hypertension development within 2-5 years (Dahms et al., 1978; Jackson et al., 1983; Wilson & Meyer, 1981).

Hypotensive systolic blood pressure responses. Other atypical systolic responses to exercise are cause for considerably greater concern than systolic hyperresponses. Aoki et al. (1982) found a great rise in systolic pressure in response to the Master's two-step exercise in the "pre-hypertensive" and "borderline" stages of essential hypertension, and conversely, a small rise in systolic pressure in the "established" stage. The causes for conflicting results were uncertain, but were believed to be induced by multiple factors, such as the nature of vascular smooth muscle, connective tissue in the vascular wall, and vasoactive humoral factors, among other factors.

Representing an even greater concern, Irving et al. (1977) found that the lowest average maximal systolic pressures were in patients with CHD and were often associated with two or three vessel disease or reduced

ejection fractions, or both. However, a maximal systolic pressure of less than 140 mmHg was not of predictive value in subjects without CHD, and these subjects were characterized by significantly lower resting systolic pressures. A treadmill study by Thompson and Kelemen (1975) involving 17 patients with serious obstructive coronary artery disease revealed two patterns of atypical systolic pressure response. Three patients exhibited a progressive fall in pressure to below resting levels from the first moment of exercise, and the remaining patients exhibited an initial increase in blood pressure with a subsequent fall to below resting levels in systolic pressure. These responses coincided with the appearance of S-T segment depression and angina. Furthermore, these hyporesponses could not be attributed to any pharmocologic effects. Morris, Phillips, Jordan, and McHenry (1978) reported that it is not necessary for systolic blood pressue to drop below resting levels in order to suspect severe compromises to the coronary circulation. These investigators determined that an exercise-induced decrease of 10 mmHg or more below peak systolic pressure was a useful and highly specific manifestation of severe multiple vessel coronary artery disease. This atypical systolic response observed by Thompson and Kelemen (1975) and Morris et al. (1978) was attributed to acute left ventricular failure secondary to

extensive myocardial ischemia. Morris et al. (1978) reported studies with opposing results. Research by Saltin and Sternberg and Smith, Guyton, and Manning (cited by Morris et al., 1978) suggested that hypotension commonly occurs during prolonged, exhaustive strenuous exercise in normal subjects. However, graded exercise testing is not an example of this type of endurance exercise. Morris et al. (1978) reported the conflicting results observed by Baker, Levites, and Anderson, as well. In an abstract, these investigators reported that a decreasing systolic blood pressure may be observed during graded exercise in the absence of significant coronary artery disease, especially in women.

In view of these conflicting results, Morris et al. (1978) provided a set of conditions which, in the presence of an exercise-induced decrease in systolic pressure of 10 mmHg or more below peak values, indicate multiple vessel coronary artery disease. These criteria were: (1) following the initial measurement, a decrease in pressure sustained for one or more measurements obtained at 15 to 20 second intervals; (2) no existing cardiomyopathy, heart valve disease, hypovolemia, orthostatic hypotension, or congestive heart failure at rest; (3) no cardiac arrhythmias during blood pressure measurement; (4) absence of patient-ingested pharmacologic agents which significantly

reduce cardiac output or boost the vasodilation accompanying exercise, or both; and (5) exercise duration of at least one minute.

Significance of Various Diastolic Blood Pressure Responses

The atypical exercise responses associated with diastolic blood pressure include an increase of 20 mmHg or more and/or a rise above 110 to 120 mmHg (ACSM, 1980). In a study by Sheps, Ernst, Briese, and Myerburg (1979), an increase in diastolic pressure of more than 15 mmHg in at least two exercise measurements, compared to resting values, was considered an atypical response. These investigators used Phase V Korotkoff sound in determination of diastolic indexes, believing it was more reliable amidst the background noise of the treadmill. Of the 281 patients, 72 patients exhibited an atypical diastolic blood pressure response to submaximal treadmill exercise, which could not be related to atypical systolic responses or S-T segment changes. In a subgroup of 41 patients who underwent coronary angiography, 50% of patients who exhibited a normal diastolic response actually had normal coronary arteries, whereas only 17% of those who exhibited an atypical response had normal arteries. Conversely, only 11% of patients who exhibited a normal diastolic response had triple vessel or left main coronary artery disease, compared with 44% of those who exhibited an atypical response.

The method of increase in diastolic blood pressure is presently unknown. However, it was suggested that there may be a muscle afferent feedback mechanism which, when muscles contract, causes transmission of afferent signals from receptors in active muscles to the brain, stimulating sympathetic centers. It is known that patients with congestive heart failure have greater vascular sodium content, which tends to limit vasodilation (Sheps et al., 1979). Mason, Zelis, Longhurst, and Lee (cited by Sheps et al., 1979) believe this to be a protective mechanism to maintain blood pressure during exercise, because patients with congestive heart failure have below normal increases in cardiac output, systemic pressure, and local blood flow, combined with higher perpheral resistance.

In addition to the abnormal increases in diastolic blood pressure during exercise, Aoki et al. (1982) have observed atypical diastolic increases following exercise termination. Diastolic pressure normally falls after exercise in healthy normotensive men (Aoki et al., 1982; Bruce et al., 1974; Wolthuis et al., 1977). However, Aoki et al. (1982) noted a diastolic increase in hypertensive subjects following treadmill exercise. These investigators speculated that this response may be associated with a defect or hypofunction of arterial vasodialtion following dynamic exercise, as a result of myogenic mechanisms in the

vascular smooth muscle, and metabolic and nervous blood pressure regulation mechanisms. They speculated that a defective relaxing activity of the arterial smooth muscle may be a responsible factor, as well.

Blood Pressure Standards for Clinical Exercise Testing

In the second edition of Guidelines for Graded Exercise Testing and Exercise Prescription, ACSM (1980) has suggested several criteria for test termination which are considered to be absolute in exercise tests conducted without physicians. However, in the presence of a physician, they were intended to offer guidance in decisions to terminate exercise tests. Among other signs and symptoms, ACSM suggested possible endpoints related to blood pressure responses to exercise. ACSM (1980) has determined that a failure of systolic pressue to rise with increasing intensities, except in the early adjustment stages, or a decline of systolic blood pressure by 10 mmHg or more should usually prompt test administrators to terminate exercise. An increase in systolic blood pressure in the range of 250 mmHg or above may be considered an indication to stop exercise, as well. In relation to diastolic blood pressure responses, an increase of more than 20 mmHg or a rise above 110 to 120 mmHg may be an indication to terminate exercise testing.

In 1972, the American Heart Association stated in

Exercise Testing and Training of Apparently Healthy Individuals: A Handbook for Physicians criteria concerned with systolic pressure responses. A localized or general pallor, indicative of cutaneous vasoconstriction and often associated with falling systolic pressure, was indicated as a sign which may dictate test termination. In addition, systolic blood pressure which fails to rise or rises temporarily and then falls progressively as exercise increases was indicated to dictate termination of exercise testing irrespective of other symptoms (AHA, 1972). In 1979, the AHA further stated in The Exercise Standards Book (revised edition) that a test will usually be prematurely discontinued if an excessive rise in systolic blood pressure to 230-250 mmHg is manifested.

A distinction was made between absolute and relative termination criteria for exercise testing (Froelicher, 1983). An absolute indication for definite test termination included a decline in systolic pressure with increases in exercise. A relative indication for termination involved hypertensive responses of greater than 280 mmHg systolic or greater than 115 mmHg diastolic. The relative endpoint indicators require well-informed clinical judgement.

Prospects for Detecting Latent Hypertension Through Exercise Testing

Numerous epidemiological studies have been performed

which express a strong relationship between risk factor variables such as elevated resting blood pressure, and future development of hypertension and/or CHD. However, only three studies have concentrated on the ability to predict subsequent resting hypertension from exercise blood pressure responses. In the research conducted by Wilson & Meyer (1981), normal resting blood pressure was defined as systolic less than 140 mmHg and diastolic less than 90 mmHg; normal exercise blood pressure was defined as systolic less than 225 mmHg and diastolic less than 90 mmHg. Of the 3,395 men and 425 women, only 341 exhibited normal resting blood pressure with a high exercise blood pressure at the initial visit. Of these, 21% developed resting hypertension within 32 months, compared with 9% in the normal resting/normal exercise blood pressure control group. In an earlier retrospective study using the same resting and exercise blood pressure criteria, Dahms et al. (1978) found that 19 out of the 26 subjects exhibiting normal resting and hypertensive exercise blood pressure responses developed resting hypertension within five years.

A similar study by Jackson et al. (1983) utilized the same resting hypertensive criteria as the previous investigation, but altered the exercise criteria to match that recommended by Sannerstedt (1981) for men. Thus, a hypertensive response to exercise was characterized by

systolic pressure ≥ 230 mmHg and/or diastolic pressure ≥ 110 mmHg. Furthermore, Jackson et al. (1983) defined diastolic pressure as Phase V Korotkoff sound. Of the 4,856 patients, 53 men were found to be normotensive at rest but hypertensive during exercise. However, only 23 of these men returned for a follow-up visit 2-4 years later. Of these men 51% were found to be hypertensive, compared with only 15% of normotensive rest/exercise controls. The resting normotensive men exhibiting exercise hypertension were three times more likely to develop resting hypertension within 2-4 years, than men exhibiting resting and exercise normotension. These studies suggest that measurement of blood pressure during exercise testing provides a means of earlier detection of otherwise normal adults who are at higher risk of developing subsequent resting hypertension.

Summary

Blood pressure, important in maintaining homeostasis, normally averages 120/80 mmHg in young adults at rest. Resting blood pressure may be increased during periods of anxiety, winter and spring seasons of the year, and late evening, and due to increases in age, body weight, and body fat. Resting blood pressure may be lower in women, physically fit or younger individuals, and in inverted positions. Resting blood pressure is regulated by the reciprocal interaction between various short-term,

intermediate, and long-term mechanisms, such as the sympathetic nervous system, baroreceptor reflexes, CNS ischemic response, and various hormones.

Although it is known that blood volume and cardiac output are normal in resting hypertension, whereas total peripheral resistance is greatly enhanced, the malfunctioning mechanisms involved are yet to be fully explained with certainty. A possible explanation for resting hypertension lies in abnormal renal function. Mechanisms for regulation of exercise blood pressure are currently uncertain as well, but possible mechanisms, such as a "central command," muscle fiber type, and muscle ergoreceptors, have been suggested.

With respect to blood pressure responses to dynamic exerise, apparently healthy adults exhibit a linear rise in systolic pressure, with no change in diastolic pressure. Hypertensive patients exhibit varied blood pressure responses to exercise, ranging from near normal responses in younger or mildly hypertensive patients to hyper- and hyporesponses in the established stages of hypertension.

The recommended blood pressure criteria for exercise test termination are associated with myocardial dysfunction. They include: failure of systolic pressure to rise with increasing exercise intensities, a systolic decrease of 10 mmHg or more, a systolic increase into the range of 250 mmHg

or higher, a diastolic rise of more than 20 mmHg, or a diastolic rise above 100 to 120 mmHg. The most distressing exercise blood pressure responses involve a failure of systolic pressure to rise or a decrease in systolic with increases in exercise intensity. These responses are indicative of left ventricular dysfunction and severe multiple vessel disease. Diastolic hyperresponses have been associated with triple vessel disease, as well.

Antihypertensive medications, such as diuretics, vasodialtors, and beta-blockers, cause either a decrease in total peripheral resistance or a decrease in coronary sympathetic stimulation. These effects may vary with exercise, depending on the type of medication, age of patient, and stage of hypertension. Physical training has been shown to mimic the effects of these medications in older and hypertensive patients and therefore, has been a recommended therapy for these patients. Blood pressure hyperresponses to exercise in normotensive subjects have been shown to predict the future development of resting hypertension within 2-5 years.

Chapter III

JOURNAL MANUSCRIPT

INCIDENCE AND IMPLICATIONS OF ATYPICAL EXERCISE BLOOD PRESSURE RESPONSES IN ADULTS WITHOUT DIAGNOSED CORONARY HEART DISEASE

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INCIDENCE AND IMPLICATIONS OF ATYPICAL EXERCISE BLOOD PRESSURE RESPONSES IN ADULTS WITHOUT DIAGNOSED CORONARY HEART DISEASE

Angela B. Williams

ABSTRACT

Data were collected from the initial symptom-limited maximal exercise tests of 161 patients without diagnosed coronary heart disease (CHD). Subjects were grouped according to their systolic (SBP) and diastolic (DBP) blood pressure changes between the final two stages of exercise: S_{AT} = SBP $\downarrow$ > 4, no change > ±4, or $\uparrow \geq$ 30 mmHg; D_{AT} = DBP $\uparrow \geq$ 10 or above 110 mmHg; S_T = those SBP not categorized as S_{AT}; D_T = responses not defined as D_{AT}. Responders were categorized into groups as follows: S_TD_T; S_TD_{AT}; $S_{AT}D_T$; and $S_{AT}D_{AT}$. The atypical blood pressure response manifested in 41% of the subjects may have been influenced by a lack of age and sex restriction in the sample. The $S_{AT}D_T$, S_TD_{AT}, and $S_{AT}D_{AT}$ response groups represented 25%, 9%, and 8%, respectively. The large number of S_{AT} responders, a result of no SBP change, may be misleading. Increases in SBP are normally expected to taper off as maximum effort is approached. The groups did not differ physically, in health status, or in prescribed cardiovascular modifying

medications. In predisposing CHD risk factors, the groups did not differ in supine SBP, relative weight, or any plasma lipids. The significant ($p < .05$) group difference found in supine DBP was not confirmed in post-hoc analysis. The S_TD_{AT} responders tended to have higher supine DBP. At peak exercise, the groups did not differ in functional capacity, heart rate, endpoint symptoms, RPE, baseline ECG, ECG changes, arrhythmias, or intraventricular heart blocks. None of the groups were associated with excessively high or reduced blood pressures, ECG abnormalities, or arrhythmias at peak exercise. The results suggest that physician-referred, asymptomatic adults without diagnosed CHD entering medically supervised exercise programs may require minimal supervision during exercise testing, even in the presence of CHD risk factors or atypical exercise blood pressure responses.

Introduction

A number of risk factors have long been recognized as valuable indices for identifying the presence of coronary heart disease (CHD). Among these, resting hypertension, is considered to be particularly important, i.e., a blood pressure measured during a resting metabolic state which is equal to or greater than 140 mmHg systolic and/or 90 mmHg diastolic.[1-3] More recently, however, researchers have demonstrated that examination of blood pressure responses to exercise improves predictability in assessing present and future resting blood pressure status.[2,3] These investigators have found that some individuals exhibit a typical resting blood pressure with an atypical blood pressure response to exercise, and this may forecast development of resting hypertension. Early detection of a predisposition toward development of resting hypertension in apparently healthy individuals may lead to more effective clinical management leading to prevention of hypertension.

A primary objective of this research was to examine the incidence of atypical exercise blood pressure responses to exercise in a group of men and women without diagnosed CHD referred for a medically supervised exercise program. A second objective was to determine if those manifesting atypical blood pressure responses to exercise also exhibited relatively more predisposing CHD risk, or evidenced within

their exercise tests any clinical signs and symptoms of potentially hazardous myocardial dysfunction.

Methods

Subjects. Data were obtained from the clinical records of all clients without diagnosed CHD which were available in the files at the Virginia Tech Intervention Center as of December, 1984. Only those considered to be apparently healthy or hypertensive, and completing a minimum of four exercise stages during their initial treadmill GXT were included. The sample consisted of 16.2% smokers and ranged in age from 21-69 years. Other demographic information of the sample are presented in Table I.

Demographic variables and exercise test protocol. Symptom-limited treadmill exercise tests (GXTs) were administered using individualized protocols which increased 1-3 METs (predicted) every 2 minutes, with endpoints based upon guidelines set by the American College of Sports Medicine.[4] The 12-lead ECG was recorded prior to exercise and at 2 minutes intervals throughout the test and for at least 6 minutes post-exercise. Heart rate and blood pressure were measured at rest, toward the end of each stage, and in the post-exercise period. Blood pressure was taken by auscultation using mercury sphygmomanometers. The Phase IV Korotkoff sound was accepted as the indicator of diastolic pressure.[5] Rating of perceived exertion (RPE) was

also determined at the end of each stage of exercise. Signs and symptoms of exertional intolerance were assessed throughout the tests. Current health history, height, weight, skinfold thicknesses, and smoking (self-report) were assessed immediately prior to each test. Fasting blood samples for lipid assessment were drawn by a registered nurse within 1 week of the GXT. A physician supervised each GXT. Subjects gave informed consent prior to testing.

Measurement of blood pressure and ECG variables. Resting blood pressure measurements were made prior to exercise with the subjects in supine and standing positions. Exercise blood pressures were measured during the last minute of each stage of exercise. Recovery blood pressures were measured at 2 and 4 minutes post-exercise.

Electrocardiographic measurements (± 0.05 mv) were made for R wave amplitude, J point level, and S-T segment deviation at .04 and .08 seconds following the J point at resting supine, resting standing, hyperventilation, the exercise stage corresponding to 50-70% of peak exercise, and at peak exercise.[6] Measurements were made from lead V_5, and an average of three complexes was recorded whenever possible. For purposes of assessing amplitude components of the ECG, the horizontal level of the P-R interval was taken as the baseline.

Arrhythmias and intraventricular heart blocks were

recorded only if they represented a permanent condition or if considered to be of "more than isolated" frequency.

Criteria for exercise blood pressure. An atypical systolic blood pressure response to exercise was defined either as a decrease > 4 mmHg, no change beyond ± 4 mmHg, or a rise ≥ 30 mmHg between the last two blood pressure responses during the GXT. Those subjects not meeting the above criteria were considered to exhibit a typical systolic response to exercise. An atypical diastolic response was defined as either a level ≥ 110 in the final stage of exercise or an increase ≥ 10 mmHg between the last two blood pressure responses during the GXT. Those subjects not meeting the above criteria were considered to exhibit a typical diastolic response to exercise.

Criteria for blood pressure grouping. Subjects were placed into one of four groups based upon the blood pressure criteria previously described for typical and atypical response to exercise. The groups were those who exhibited both a typical systolic and diastolic response (S_TD_T); those who exhibited a typical systolic and an atypical diastolic response (S_TD_{AT}); those who exhibited an atypical systolic and a typical diastolic response ($S_{AT}D_T$); and those who exhibited both an atypical systolic and an atypical diastolic response ($S_{AT}D_{AT}$).

Data analyses. The subject sample was described using means and standard deviations. Incidence of atypical blood pressure responses was determined using frequency counts and percentages. Group comparisons were made using the chi-square distribution on categorical variables such as gender, health status, signs and symptoms of exertional intolerance, etc. A one-way analysis of variance (ANOVA) procedure was used to make group comparisons on continuous variables such as age, functional capacity, weight, blood pressure, etc. A post-hoc procedure (Tukey Test) was performed for cases wherein a significant F ratio was first established by ANOVA ($p < .05$).

Results

Incidence of atypical responses. Of the entire sample, 59% (n=95) exhibited both typical systolic and diastolic blood pressure changes between the last two stages of exercise, while the remainder responded with either atypical systolic, atypical diastolic, or both atypical systolic and diastolic changes (See left panel in Figure 1). Of the total sample, the $S_{AT}D_{T}$, $S_{T}D_{AT}$, and $S_{AT}D_{AT}$ response groups represented 24.8%, 8.7%, and 7.5%, respectively (See right panel in Figure 1). Of the three atypical response groups, the majority (60.6%) exhibited exclusively an atypical systolic exercise response (See left panel in Figure 2). The $S_{T}D_{AT}$ and $S_{AT}D_{AT}$ response groups represented 21.1% and

18.2%, respectively (See left panel in Figure 2). Of the atypical response patterns, the majority (63.6%) exhibited no change in systolic pressure (See right panel in Figure 2). An excessive increase in diastolic pressure and a high (≥ 110 mmHg) diastolic response represented 19.7%, each, of the atypical response patterns; whereas a decrease in systolic pressure and an excessive increase in systolic pressure represented only 10.6% and 4.5%, respectively (See right panel in Figure 2).

Predisposing factors. Tables I and II present demographic variables and cardiovascular risk factor data for the four groups. Significant group differences were found for only one of the variables presented therein, i.e., diastolic blood pressure. In the post-hoc analysis of diastolic blood pressure following ANOVA ($p < .05$), however, none of groups were found to differ. The greatest difference occurred between the S_TD_T and the S_TD_{AT} responders (difference between means was 6.8 mmHg; see Table II).

There were no statistically significant ($p < .05$) differences among groups relative to prescribed cardiovascular modifying medications. The majority of subjects (59%) were not taking any medications, 23.6% were taking medications that may affect blood pressure, and 16.1% were prescribed more than one medication.

Peak exercise. Only one statistically significant difference was observed between groups for peak exercise parameters (Table III). A significant difference ($p < .0001$) was observed among groups for diastolic blood pressure. The two groups which included atypical diastolic responders had significantly higher peak diastolic blood pressures ($\bar{X}$ = 100 mmHg and 103.3 mmHg, respectively), than the other two groups.

Myocardial dysfunction. Table IV presents the data for ECG changes at peak exercise, wherein the resting supine ECG complex represented the baseline pattern. There were no significant differences among the groups for baseline ECG measurements or for ECG changes.

Chi-square analysis revealed no significant differences ($p < .05$) between the groups for any of the arrhythmias or intraventricular heart blocks (Table IV).

Recovery blood pressures. A significant ($p < .05$) differenc was revealed among groups for diastolic blood pressure at 2 minutes following exercise. Significant ($p < .10$) group differences were also found for systolic pressure at 2 minutes and diastolic pressure at 4 minutes following exercise. Although group differences were not found in post-hoc analysis, inspection of the data in Table V shows that the S_TD_{AT} and $S_{AT}D_{AT}$ responders tended to have higher average diastolic pressure levels (86.4 mmHg and 87.5 mmHg)

than the other groups. The typical responders tended to have lower diastolic pressures (78.4 mmHg) than the other groups at 4 minutes post-exercise. Inspection of Table V also reveals that the S_TD_{AT} and $S_{AT}D_{AT}$ responding groups tended to have higher systolic pressures 2 minutes post-exercise (172.5 mmHg and 176.6 mmHg, respectively) than the other groups.

Discussion

The results indicate that a considerable proportion of physician-referred entrants into medically supervised adult exercise programs are apt to exhibit atypical blood pressure changes during exercise testing. No change in systolic pressure was, by far, the major (63.6%) atypical response found. The results may be confounded by considering this an atypical response during the final two stages of exercise. Systolic pressure is normally expected to taper off as maximum effort is approached.[7,8,9] Furthermore, adjustments were not made for subject variation in MET level increases between the stages.

The groups in this study were similar in physical characteristics and usage of cardiovascular modifying medications. The lack of medication influence was further supported by the small percentage (23.6%) of subjects receiving those which affect the cardiovascular system.

Several predisposing risk factors have been associated

with the development of coronary heart disease.[4,5] The groups were also similar in these CHD risk factors. Additional group differences may have been concealed as a result of the small number of subjects in the groups, especially the S_TD_{AT} (N=14) and $S_{AT}D_{AT}$ (N=12) groups.

The groups were similar in peak exercise responses and indicators of myocardial strain. The groups did not exhibit systolic hypotensive blood pressure responses indicative of left ventricular dysfunction[10], nor did they experience excessive diastolic responses relating to faulty peripheral vascular regulation of blood pressure.[1] The S-T segment depression from baseline to peak exercise was consistently less than that considered critical plus R amplitude decreased in all groups, indicating an absence of myocardial ischemia.

These results lend conflicting support to the standards for exercise testing setforth by ACSM[4] with respect to suggested medical supervision. The ACSM[4] recommendation that exercise tests of asymptomatic adults without CHD risk factors be administered by qualified exercise test personnel was supported. However, not supported was the recommendation that in the absence of known CHD, testing adults with CHD risk factors requires a physician within the area. The results indicate that apparently healthy entrants into supervised exercise programs may require minimal

supervision during exercise testing and training, even in the presence of CHD risk factors or atypical blood pressure responses. However, before more lenient criteria for medical supervision or test termination are warranted, additional investigations should enlarge the size of the groups, view blood pressure changes throughout exercise, and account for varying MET level increases between exercise stages.

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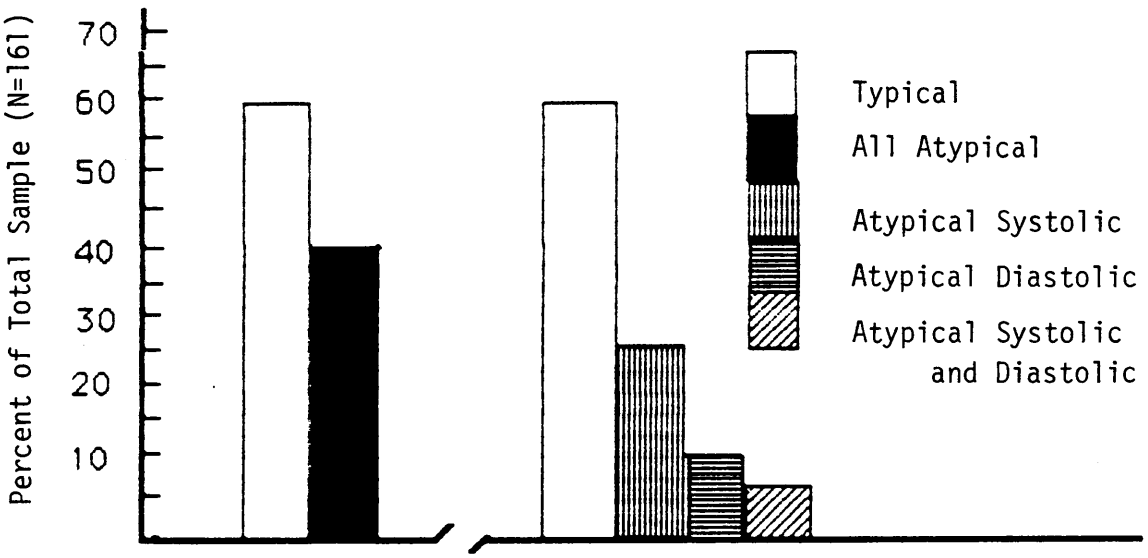

Figure 1. Incidence of typical and atypical exercise blood pressure responses in a group of adults without diagnosed CHD. The incidence of All Atypical (left panel) is represented by Atypical Systolic, Atypical Diastolic, and Atypical Systolic and Diastolic (right panel).

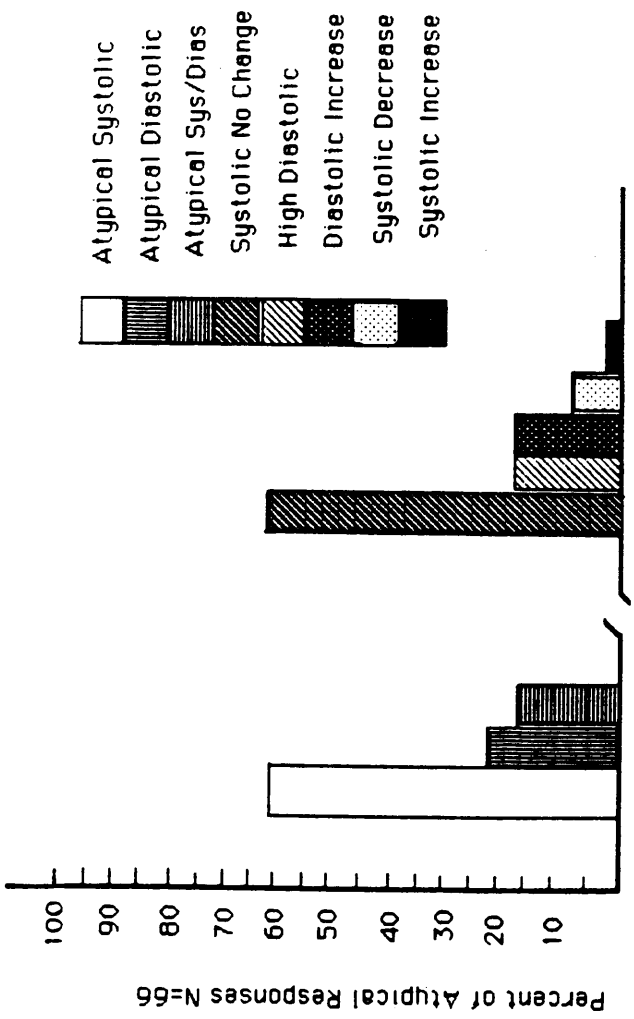

Figure 2. Incidence of atypical exercise blood pressure responses in a group of adults without diagnosed CHD

TABLE I

DEMOGRAPHICS AND HEALTH STATUS
FOR EACH EXERCISE BLOOD PRESSURE RESPONSE GROUP

	Physical Characteristics										Clinical Status	
	Age (yrs)		Gender (male)	Gender (female)	Height (cm)		Weight (kg)		PFat (%)		HYPER	APPHLTY
BP Groups	$\bar{X}$	SD	%	%	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	%	%
S_TD_T N=95	42.5	8.3	74.7	25.3	176.5	9.3	83.4	16.1	23.4	6.5	29.5	70.5
S_TD_{AT} N=14	45.1	11.0	78.6	21.4	177.2	6.8	81.9	18.3	21.4	4.7	35.7	64.3
$S_{AT}D_T$ N=40	43.0	8.7	77.5	22.5	175.2	6.9	80.9	14.8	22.2	7.2	37.5	62.5
$S_{AT}D_{AT}$ N=12	44.0	8.7	75.0	25.0	172.3	7.6	74.8	11.8	20.0	7.3	33.3	66.7
Total Sample N=161	42.9	8.6	75.8	24.2	175.9	8.5	82.0	15.8	22.7	6.6	32.3	67.7

Pfat = percentage body fat; HYPER = hypertension; APPHLTY = apparently healthy; BP = blood pressure; $\bar{X}$ = mean; SD = standard deviation; S_T = typical systolic; D_T = typical diastolic; D_{AT} = atypical diastolic; S_{AT} = atypical systolic.

TABLE II

PREDISPOSING VARIABLES
FOR EACH EXERCISE BLOOD PRESSURE RESPONSE GROUP

	SBP (mmHg)		DBP[a] (mmHg)		RELWT		CHOL (mg/dl)		TRIG (mg/dl)		HDL-C (mg/dl)		TC/HDL-C RATIO		SMOKE %
BG Groups	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	%
S_TD_T N=95	128.7	13.9	82.8	9.4	112.8	18.2	222.8	47.5	148.6	93.2	42.8	8.8	5.0	1.4	12.6
S_TD_{AT} N=14	136.3	16.2	89.6	12.7	109.3	18.1	208.9	30.4	155.9	80.8	48.4	19.5	5.2	3.1	28.6
$S_{AT}D_T$ N=40	133.4	15.1	86.3	7.9	111.5	18.1	213.3	42.8	151.2	59.6	47.3	15.2	4.6	1.3	25.0
$S_{AT}D_{AT}$ N=12	135.3	23.0	85.7	13.1	105.5	13.6	204.3	27.8	185.5	86.6	44.4	14.4	4.7	1.3	0.0

SBP = systolic blood pressure; DBP = diastolic blood pressure; RELWT = relative weight; CHOL = cholesterol; TRIG = triglyceride; HDL-C = high-density lipoprotein cholesterol; TC/HDL-C = total serum cholesterol/high-density lipoprotein; SMOKE = smokers; $\bar{X}$ = mean; SD = standard deviation; S_T = typical systolic; D_T = typical diastolic; D_{AT} = atypical diastolic; S_{AT} = atypical systolic.

a - Significant ($p < .05$) variation revealed in ANOVA analysis, but not in post-hoc analysis.

TABLE III

PEAK EXERCISE RESPONSES
FOR EACH EXERCISE BLOOD PRESSURE RESPONSE GROUP

	Performance and Hemodynamic Variables								Endpoint Symptomatology					
	FC (METs)[1]		HR (beats/min)		SBP (mmHg)		DBP[a] (mmHg)		CHDIS	DYS	LFAT	GFAT	RPE	
BP Group	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	%	%	%	%	$\bar{X}$	SD
S_TD_T N=95	9.8	2.4	167.5	16.9	194.6	25.5	84.3	12.5	3.3	26.2	52.5	27.9	17.6	2.2
S_TD_{AT} N=14	9.1	1.0	165.6	16.0	200.1	36.4	100.0	17.2	0.0	25.0	41.7	25.0	18.0	1.0
$S_{AT}D_T$ N=40	10.6	2.0	173.7	13.9	192.0	20.2	88.2	8.6	0.0	18.5	51.9	37.0	17.1	2.0
$S_{AT}D_{AT}$ N=12	9.7	1.8	166.8	15.8	195.8	36.0	103.3	17.5	0.0	10.0	30.0	10.0	16.2	1.6

[1] MET level is predicted from speed and grade of treadmill; ACSM, 1980.

FC = functional capacity; HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure; CHDIS = chest discomfort; DYS = dyspnea; LFAT = leg fatigue; GFAT = general fatigue; RPE = rating of perceived exertion based on Borg's 6-20 point scale; BP = blood pressure; $\bar{X}$ = mean; SD = standard deviation; S_T = typical systolic; D_T = typical diastolic; D_{AT} = atypical diastolic; S_{AT} = atypical systolic.

a - S_TD_{AT} and $S_{AT}D_{AT}$ groups higher average than other two groups; $p < .0001$.

TABLE IV

ECG RESPONSES DURING EXERCISE
FOR EACH EXERCISE BLOOD PRESSURE RESPONSE GROUP

	ECG Changes[1]								Arrhythmias and Blocks		
	R Wave (mv)		J Point (mv)		ST @ .04 (mv)		ST @ .08 (mv)		SVA	VA	IVB
BP Groups	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	%	%	%
S_TD_T N=95	-.14	.31	-.13	.09	-.06	.07	.05	.13	15.8	10.5	2.1
S_TD_{AT} N=14	-.08	.24	-.15	.08	-.11	.07	.00	.09	21.4	0.0	0.0
$S_{AT}D_T$ N=40	-.18	.18	-.15	.08	-.07	.07	.02	.16	22.5	12.5	0.0
$S_{AT}D_{AT}$ N=12	-.05	.21	-.15	.09	-.07	.06	.02	.08	8.3	25.0	0.0

[1] Changes from supine rest to peak exercise.

R Wave = amplitude of R wave; J Point = amplitude of J point; ST @ .04 = S-T segment deviation at .04 seconds from J point; ST @ .08 = S-T segment deviation at .08 seconds from J point; SVA = supraventricular arrhythmias; VA = ventricular arrhythmias; IVB = intraventricular blocks; BP = blood pressure; $\bar{X}$ = mean; SD = standard deviation; S_T = typical systolic; D_T = typical diastolic; D_{AT} = atypical diastolic; S_{AT} = atypical systolic.

TABLE V

RECOVERY SYSTOLIC AND DIASTOLIC BLOOD PRESSURES
FOR EACH EXERCISE BLOOD PRESSURE RESPONSE GROUP

	Recovery Blood Pressures							
	SBP[a] (2 minutes) (mmHg)		DBP[b] (2 minutes) (mmHg)		SBP (4 minutes) (mmHg)		DBP[c] (4 minutes) (mmHg)	
BP Groups	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD
S_TD_T N=95	161.8	21.4	79.1	10.5	140.3	17.0	78.4	10.3
S_TD_{AT} N=14	172.5	30.6	86.4	14.0	148.0	29.1	83.2	14.6
$S_{AT}D_T$ N=40	164.2	16.4	82.5	9.5	145.8	13.8	82.1	8.5
$S_{AT}D_{AT}$ N=12	176.6	33.4	87.5	15.3	150.7	22.3	85.1	16.7

SBP = systolic blood pressure; DBP = diastolic blood pressure;

BP = blood pressure; $\bar{X}$ = mean; SD = standard deviation;

S_T = typical systolic; D_T = typical diastolic; D_{AT} = atypical diastolic; S_{AT} = atypical systolic.

a - Significant ($p < .10$).

b - Significant ($p < .05$) variance revealed in ANOVA analysis, but not confirmed post-hoc analysis.

c - Significant ($p < .10$).

Chapter IV

CONCLUSIONS, IMPLICATIONS, AND RECOMMENDATIONS FOR FUTURE RESEARCH

Summary

In recent years, studies have shown that some individuals exhibit a typical resting blood pressure, but an atypical blood pressure response to exercise. Atypical blood pressure responses to exercise have been of considerable interest to investigators because of the possible link of such patterns to future development of hypertension, a risk factor for coronary heart disease (CHD). Early detection of an atypical response to exercise may lead to earlier institution of preventive treatment.

This investigation was conducted to determine the incidence of atypical exercise blood pressure responses to exercise in a group of adults without diagnosed CHD. The study was further designed to determine if any such atypical blood pressure responders possessed a preponderance of CHD risk factors, or if they were acutely associated with evidence of myocardial strain in their exercise tests.

Data were collected from the initial graded exercise tests of 161 men and women without diagnosed CHD available in the file records at the Virginia Tech Intervention Center as of December, 1984. Subjects were grouped according to their exercise blood pressure response between the last two

blood pressure responses based upon typical and atypical values cited in the literature. Criteria for atypical systolic response to exercise were: a decrease in systolic pressure > 4 mmHg with increases in exercise intensity; no change in systolic pressure beyond ± 4 mmHg with increases in exercise intensity; or an excessive increase in systolic pressure ≥ 30 mmHg with increases in exercise intensity. Criteria for atypical diastolic response to exercise were an increase in diastolic pressure ≥ 10 mmHg with increases in exercise intensity or an excessively high diastolic response ≥ 110 mmHg with increases in exercise intensity. Exercise blood pressure groupings based upon the criteria described previously were: a typical systolic and a typical diastolic response to exercise (S_TD_T); a typical systolic response to exercise, but an atypical diastolic response (S_TD_{AT}); an atypical systolic response to exercise, but a typical diastolic response ($S_{AT}D_T$); and both an atypical systolic and diastolic blood pressure response to exercise ($S_{AT}D_{AT}$). Blood pressure response groups were investigated for differences in physical characteristics, health status, predisposing CHD risk factors, prescribed medications, peak exercise responses, indicators of myocardial dysfunction, and recovery blood pressure responses.

Results of the study revealed atypical blood pressure responses to exercise in 41% of the sample. The majority of

atypical responses were related to systolic, rather than diastolic pressure. No change in systolic pressure between the final two stages of exercise occurred with the greatest frequency (63.6%). This finding may distort the incidence of actual atypical responses since systolic pressure is normally expected to taper off as maximum effort is approached (Wolthuis et al., 1977). The four groups were similar with respect to physical characteristics, health status, and prescribed cardiovascular modifying medications. No single group exhibited a preponderance of CHD risk factors. The groups were also similar in peak exercise data and indicators of myocardial strain. None of the groups were associated with hypotensive or hypertensive blood pressure responses, ECG abnormalities, or arrhythmias at peak exercise. The results indicate that apparently healthy entrants into medically supervised exercise programs may require minimal supervision during exercise testing and training, even in the presence of CHD risk factors or atypical exercise blood pressure responses.

Recommendations for Future Research

This investigation should be repeated using a larger sample size in order to reveal results which may not have become apparent with the present sample size.

Future studies should also view blood pressure responses throughout the exercise test, not exclusively at

near-maximal levels of effort. Wolthuis and associates (1977) found that the normal increases in blood pressure during graded exersise level off as maximal effort is approached. Therefore, determining groups based upon blood pressure responses throughout exercise may reveal conflicting incidence of atypical responses from that in the present investigation. Such a study may also reveal group differences not shown in the present study. Furthermore, investigations should account for the differences in MET-level increments between stages throughout the exercise tests.

Investigations should be conducted combining the data from the present investigation with those of a study previously conducted by Ward (1985) on CHD patients at the Virginia Tech Cardiac Therapy Center. One such investigation should compare differences in indicators of myocardial stress between a group exhibiting a typical systolic response and a group exhibiting a decrease in systolic blood pressure. Such a study may reveal variations between healthy responses and responses considered to be most critical.

Recommendations for Practitioners in Adult Fitness

Practitioners at the Va Tech Cardiac Therapy and Intervention Center should place extreme importance upon precise measurement-taking and thorough record-keeping. A

thorough pre-test screening should incorporate a review of previous exercise tests, the health history of clients and his/her family members, physical measurements (i.e., weight, height, body fat, etc.), prescribed medications, resting blood pressure, and information concerning the clients history of smoking (i.e., duration, quantity, etc.). In conjunction with the GXT, blood lipid levels and hematocrit should be analyzed, and diet, stress level, and stress tolerance should be assessed. The importance of accurately measuring and recording each of these variables cannot be over-emphasized. This information may be valuable not only to immediately provide the client with the best possible maintenance or rehabilitation program, but also to provide long-term insight into the prevention of CHD.

The American College of Sports Medicine should reassess their standards for test safety with respect to asymptomatic adults. They may find that the major objective of exercise testing asymptomatic, apparently healthy adults should be to assess physical capabilities in order to administer an appropriate exercise prescription. They may also find that graded exercise tests can safely be administered by qualified test personnel in the absence of a physician in adults with and without CHD risk factors.

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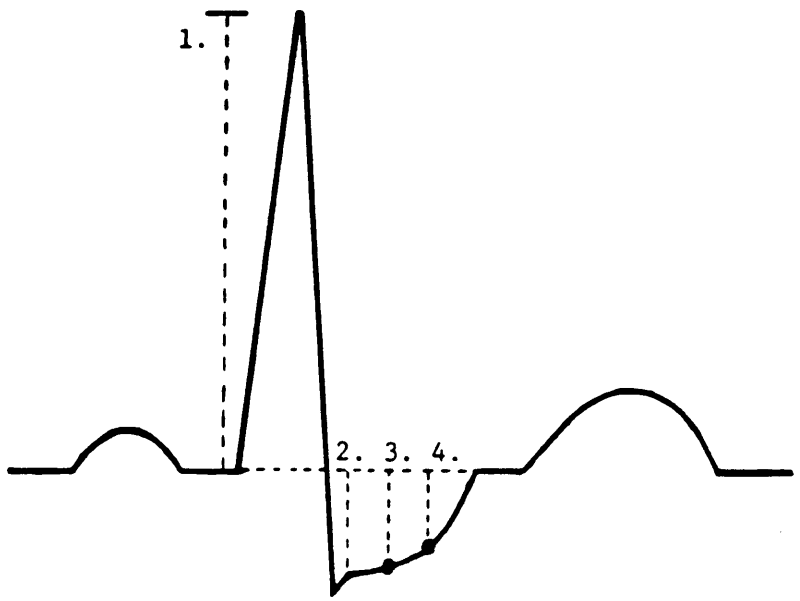
FIGURES



1. R-wave amplitude
2. J-point amplitude
3. S-T segment deviation @ 0.04 seconds
4. S-T segment deviation @ 0.08 seconds

Figure 3. Method of ECG Measurement (from Ward, 1985).

TABLES

TABLE VI

MEDICATIONS PRESCRIBED[1]
FOR EACH EXERCISE BLOOD PRESSURE RESPONSE GROUP

	Types of Medications			
	Antihyp	Dig	Other Meds	CMOD*
BP Group	%	%	%	%
S_TD_T N=95	16.8	1.1	22.1	19.0
S_TD_{AT} N=14	42.9	0.0	50.0	42.9
$S_{AT}D_T$ N=40	25.0	0.0	20.0	27.5
$S_{AT}D_{AT}$ N=12	25.0	0.0	33.3	25.0

[1] Number of subjects in each group should be considered in interpretation.

* Includes all medications from other categories which modify cardiovascular function.

Antihyp = antihypertensive; Dig = digitalis; Other Meds = other medications; CMOD = cardiovascular modifiers; S_T = typical systolic; D_T = typical diastolic; D_{AT} = atypical diastolic; S_{AT} = atypical systolic.

TABLE VII

MEDICATIONS PRESRCIBED*
FOR SUBJECTS WITHOUT KNOWN CHD

ANTIHYPERTENSIVE**

aldactazide	hygroton
aldomet	inderal
apresazide	inderide
apresoline	ismelin
catapres	KCL
combipres	lopressor
diazide	metahydrin
esimil	minipres
HYDROCHLOROTHIAZIDE	tenormin
hydrodiuril	zaroxolyn

DIGITALIS

digoxin

OTHER MEDICATIONS

actifed	demulen	ogen
aldactone**	diuril**	orinase
allergy	drixoral	ortho-novum
ALLOPURINOL	eskalith**	PHENOBARBITOL
antabuse	ESTROGEN	sinemet
antihistamine	FERROUS SULFATE	synthroid
asendin	fiorinal	tagamet
ASPIRIN	insulin	teldrin
atarax	lasix**	TETRACYCLINE
ativan	librium	THYROID HORMONE
atromid	MEPROBAMATE	thyrolar
chlortrimeton	modicon	tolinase
clinoril	mysoline	tranxene
dimetapp	nicobid	valium

* Generic names are given in upper case; brand names are given in lower case.

** Prescribed medications that modify cardiovascular function.

TABLE VIII

CHI-SQUARE ANALYSIS ACROSS GROUPS (S_TD_T, S_TD_{AT}, $S_{AT}D_T$, $S_{AT}D_{AT}$) FOR PREDISPOSING VARIABLES

Variable		p
Gender:	Male Female	0.98
Health Status:		
	Hypertension	0.82
	Apparently Healthy	0.82
Medications:		
	Antihypertensive	0.15
	Digitalis	0.87
	Other Medications	0.11
	Cardiovascular Modifiers*	0.22
Smoking		0.07[a]

* Includes all medications from other categories which modify cardiovascular function.

a - Significant ($p < .10$).

TABLE IX

CHI-SQUARE ANALYSIS TABLE OF GROUP BY SMOKING

Table of Group by Smoking

Group Frequency Percent Row PCT Col PCT	Smoking Absence	 Presence	Total
S_TD_T	83 51.55 87.37 61.48	12 7.45 12.63 46.15	95 59.01
S_TD_{AT}	10 6.21 71.43 7.41	4 2.48 28.57 15.38	14 8.70
$S_{AT}D_T$	30 18.63 75.00 22.22	10 6.21 25.00 38.46	40 24.84
$S_{AT}D_{AT}$	12 7.45 100.00 8.89	0 0.00 0.00 0.00	12 7.45
Total	135 83.85	26 16.15	161 100.00

PROB = 0.0691

TABLE X

PERCENTAGE OF GROUP S_TD_T FOR PREDISPOSING VARIABLES

Variable	N=95	%
Gender:		
Male	71	74.7
Female	24	25.3
Health Status:		
Hypertensive	28	29.5
Apparently Healthy	67	70.5
Medications:		
Antihypertensive	16	16.8
Digitalis	1	1.1
Other Medications	21	22.1
Cardiovascular Modifiers*	18	19.0
Smoking	12	12.6

* Includes all medications from other categories which modify cardiovascular function.

TABLE XI

PERCENTAGE OF GROUP S_TD_{AT} FOR PREDISPOSING VARIABLES

Variable	N=14	%
Gender:		
Male	11	78.6
Female	3	21.4
Health Status:		
Hypertensive	5	35.7
Apparently Healthy	9	64.3
Medications:		
Antihypertensive	6	42.9
Digitalis	0	00.0
Other Medications	7	50.0
Cardiovascular Modifiers*	6	42.9
Smoking	4	28.6

* Includes all medications from other categories which modify cardiovascular function.

TABLE XII

PERCENTAGE OF GROUP $S_{AT}D_T$ FOR PREDISPOSING VARIABLES

Variable	N=40	%
Gender:		
Male	31	77.5
Female	9	22.5
Health Status:		
Hypertension	15	37.5
Apparently Healthy	25	62.5
Medications:		
Antihypertensive	10	25.0
Digitalis	0	00.0
Other Medications	8	20.0
Cardiovascular Modifiers*	11	27.5
Smoking	10	25.0

* Includes all medications from other categories which modify cardiovascular function.

TABLE XIII

PERCENTAGE OF GROUP $S_{AT}D_{AT}$ FOR PREDISPOSING VARIABLES

Variable	N=12	%
Gender:		
Male	9	75.0
Female	3	25.0
Health Status:		
Hypertensive	4	33.3
Apparently Healthy	8	66.7
Medications:		
Antihypertensive	3	25.0
Digitalis	0	00.0
Other Medications	4	33.3
Cardiovascular Modifiers*	3	25.0
Smoking	0	00.0

* Includes all medications from other categories which modify cardiovascular function.

TABLE XIV

ANOVA ACROSS GROUPS (S_TD_T, S_TD_{AT}, $S_{AT}D_T$, $S_{AT}D_{AT}$) FOR PREDISPOSING VARIABLES

Variable	F	p
Age	0.46	0.71
Body Fat	1.23	0.30
Relative Weight	0.68	0.57
Cholesterol	1.09	0.36
Triglycerides	0.68	0.57
High-Density Lipoprotein	0.69	0.56
TC/HDL-C Ratio	0.45	0.72
Resting Blood Pressures:		
Supine SBP (left arm)	1.93	0.13
Supine DBP (left arm)	2.78	0.04[a]
Supine SBP (right arm)	0.48	0.70*
Supine DBP (right arm)	0.26	0.85*
Standing SBP (left arm)	0.82	0.49*
Standing DBP (left arm)	0.44	0.73*

TC/HDL-C Ratio = total serum cholesterol/high-density lipoprotein cholesterol ratio; SBP = systolic blood pressure; DBP = diastolic blood pressure.

a - Significant ($p < .05$) variance revealed in ANOVA analysis, but not confirmed post-hoc analysis.

* Percentage of group responses small.

TABLE XV

MEANS OF GROUP S_TD_T FOR PREDISPOSING VARIABLES

Variable	N=95	$\bar{X}$	SD
Age	95	42.5	8.3
Body Fat	89	23.4	6.5
Relative Weight	95	112.8	18.2
Cholesterol	92	222.8	47.5
Triglycerdies	93	148.6	93.2
High-Density Lipoprotein	30	42.8	8.8
TC/HDL-C Ratio	30	5.0	1.4
Resting Blood Pressures:			
Supine SBP (left arm)	94	128.7	13.9
Supine DBP (left arm)	94	82.8	9.4
Supine SBP (right arm)	7	133.1	14.1
Supine DBP (right arm)	7	85.4	12.8
Standing SBP (left arm)	68	126.2	14.3
Standing DBP (left arm)	68	83.8	10.9

TC/HDL-C Ratio = total serum cholesterol/high-density lipoprotein ratio; SBP = systolic blood pressure; DBP = diastolic blood pressure.

TABLE XVI

MEANS OF GROUP S_TD_{AT} FOR PREDISPOSING VARIABLES

Variable	N=14	$\bar{X}$	SD
Age	14	45.1	11.0
Body Fat	14	21.4	4.7
Relative Weight	14	109.3	18.1
Cholesterol	13	208.9	30.4
Triglycerides	13	155.9	80.8
High-Density Lipoprotein	7	48.4	19.5
TC/HDL-C Ratio	7	5.2	3.1
Resting Blood Pressures:			
Supine SBP (left arm)	14	136.3	16.2
Supine DBP (left arm)	14	89.6	12.7
Supine SBP (right arm)	1	124.0	0.0
Supine DBP (right arm)	1	84.0	0.0
Standing SBP (left arm)	11	129.6	22.8
Standing DBP (left arm)	11	85.3	23.5

TC/HDL-C Ratio = total serum cholesterol/high-density lipoprotein ratio; SBP = systolic blood pressure; DBP = diastolic blood pressure.

TABLE XVII

MEANS OF GROUP $S_{AT}D_T$ FOR PREDISPOSING VARIABLES

Variable	N=40	$\bar{X}$	SD
Age	39	43.0	8.7
Body Fat	39	22.2	7.2
Relative Weight	38	111.5	18.1
Cholesterol	39	213.3	42.8
Triglycerides	39	151.2	59.6
High-Density Lipoprotein	23	47.3	15.2
TC/HDL-C Ratio	23	4.6	1.3
Resting Blood Pressures:			
Supine SBP (left arm)	39	133.4	15.1
Supine DBP (left arm)	39	86.3	7.9
Supine SBP (right arm)	3	124.0	12.2
Supine DBP (right arm)	3	80.0	2.0
Standing SBP (left arm)	36	128.8	13.9
Standing DBP (left arm)	36	86.5	7.6

TC/HDL-C Ratio = total serum cholesterol/high-density lipoprotein ratio; SBP = systolic blood pressure; DBP = diastolic blood pressure.

TABLE XVIII

MEANS OF GROUP $S_{AT}D_{AT}$ FOR PREDISPOSING VARIABLES

Variable	N=12	$\bar{X}$	SD
Age	12	44.0	8.7
Body Fat	11	20.0	7.3
Relative Weight	12	105.5	13.6
Cholesterol	12	204.3	27.8
Triglycerides	12	185.5	86.6
High-Density Lipoprotein	7	44.4	14.4
TC/HDL-C Ratio	7	4.7	1.3
Resting Blood Pressures:			
Supine SBP (left arm)	12	135.3	23.0
Supine DBP (left arm)	12	85.7	13.1
Supine SBP (right arm)	3	144.0	40.4
Supine DBP (right arm)	3	88.7	16.3
Standing SBP (left arm)	12	133.6	23.9
Standing DBP (left arm)	12	85.7	12.9

TC/HDL-C Ratio = total serum cholesterol/high-density lipoprotein ratio; SBP = systolic blood pressure; DBP = diastolic blood pressure.

TABLE XIX

CHI-SQUARE ANALYSIS ACROSS GROUPS (S_TD_T, S_TD_{AT}, $S_{AT}D_T$, $S_{AT}D_{AT}$) FOR INDICATORS OF MYOCARDIAL DYSFUNCTION

Variable	p
Signs and Symptoms:	
Chest discomfort	0.65
Dyspnea	0.65
Leg Fatigue	0.56
General Fatigue	0.43
Other symptoms	0.03[a]
Arrhythmias and Blocks:	
Supraventricular	0.63
Ventricular	0.24
Intraventricular Blocks	0.70

a - Significant ($p < .05$).

TABLE XX

CHI-SQUARE ANALYSIS TABLE OF GROUP BY OTHER SYMPTOMS

Table of Group by Other Symptoms

Group Frequency Percent Row PCT Col PCT	Other Symptoms			
	Missing	Absence	Presence	Total
S_TD_T	34 . . .	42 38.18 68.85 56.00	19 17.27 31.15 54.29	61 55.45
S_TD_{AT}	2 . . .	6 5.45 50.00 8.00	6 5.45 50.00 17.14	12 10.91
$S_{AT}D_T$	13 . . .	23 20.91 85.19 30.67	4 3.64 14.81 11.43	27 24.55
$S_{AT}D_{AT}$	2 . . .	4 3.64 40.00 5.33	6 5.45 60.00 17.14	10 9.09
Total	. .	75 68.18	35 31.82	110 100.00

PROB = 0.0280

TABLE XXI

PERCENTAGE OF GROUP S_TD_T FOR INDICATORS OF MYOCARDIAL DYSFUNCTION

Variable	N=95	%
Signs and Symptoms:		
Chest discomfort	2	3.3
Dyspnea	16	26.2
Leg Fatigue	32	52.5
General Fatigue	17	27.9
Other symptoms	19	31.2
Arrhythmias and Blocks:		
Supraventricular	15	15.8
Ventricular	10	10.5
Intraventricular Blocks	2	2.1

TABLE XXII

PERCENTAGE OF GROUP S_TD_{AT} FOR INDICATORS OF MYOCARDIAL DYSFUNCTION

Variable	N=14	%
Signs and Symptoms:		
Chest discomfort	0	0.0
Dyspnea	3	25.0
Leg Fatigue	5	41.7
General Fatigue	3	25.0
Other symptoms	6	50.0
Arrhythmias and Blocks:		
Supraventricular	3	21.4
Ventricular	0	0.0
Intraventricular Blocks	0	0.0

TABLE XXIII

PERCENTAGE OF GROUP $S_{AT}D_T$ FOR INDICATORS OF MYOCARDIAL DYSFUNCTION

Variable	N=40	%
Signs and Symptoms:		
Chest discomfort	0	0.0
Dyspnea	5	18.5
Leg Fatigue	14	51.9
General Fatigue	10	37.0
Other symptoms	4	14.8
Arrhythmias and Blocks:		
Supraventricular	9	22.5
Ventricular	5	12.5
Intraventricular Blocks	0	0.0

TABLE XXIV

PERCENTAGE OF GROUP $S_{AT}D_{AT}$ FOR INDICATORS OF MYOCARDIAL DYSFUNCTION

Variable	N=12	%
Signs and Symptoms:		
Chest discomfort	0	0.0
Dyspnea	1	10.0
Leg Fatigue	3	30.0
General Fatigue	1	10.0
Other symptoms	6	60.0
Arrhythmias and Blocks:		
Supraventricular	1	8.3
Ventricular	3	25.0
Intraventricular Blocks	0	0.0

TABLE XXV

ANOVA ACROSS GROUPS (S_TD_T, S_TD_{AT}, $S_{AT}D_T$, $S_{AT}D_{AT}$)
INDICATORS OF MYOCARDIAL DYSFUNCTION

Variable	F	p
Testing Parameters:		
Functional Capacity	2.02	0.11
$\dot{V}O_2$ Peak	1.11	0.35
Peak Heart Rate	1.70	0.17
RPE (6-20 point)	1.04	0.38
ECG Measures:		
R wave amplitude		
Supine	0.59	0.62
Hyperventilation	0.53	0.66
Standing	0.06	0.98
Submaximal exercise	0.33	0.80
Peak exercise	0.18	0.91
J point amplitude		
Supine	0.65	0.59
Hyperventilation	0.77	0.52
Standing	0.66	0.58
Submaximal exercise	0.28	0.84
Peak exercise	0.82	0.49
ST DEV @ .04 seconds*		
Supine	0.18	0.91
Hyperventilation	0.05	0.98
Standing	0.32	0.81
Submaximal exercise	0.43	0.74
Peak exercise	1.32	0.27
ST DEV @ .08 seconds**		
Supine	0.72	0.54
Hyperventilation	0.30	0.83
Standing	0.11	0.95
Submaximal exercise	0.68	0.57
Peak exercise	0.93	0.43
Recovery Blood Pressures:		
SBP (2 minutes)	2.23	0.09[a]
DBP (2 minutes)	3.71	0.01[b]
SBP (4 minutes)	2.06	0.11
DBP (4 minutes)	2.32	0.08[c]

SBP = systolic blood pressure; DBP = diastolic blood pressure.

* S-T segment deviation 0.04 seconds from the J point.

** S-T segment deviation 0.08 seconds from the J point.

a - Significant ($p < .10$).

b - Significant ($p < .05$) variance revealed in ANOVA analysis, but not confirmed post-hoc analysis.

c - Significant ($p < .10$).

TABLE XXVI

MEANS OF GROUP S_TD_T FOR INDICATORS OF MYOCARDIAL DYSFUNCTION

Variable	N=95	$\bar{X}$	SD
Testing Parameters:			
Functional Capacity	95	9.8	2.4
$\dot{V}O_2$ Peak	95	32.4	7.7
Peak Heart Rate	94	167.5	16.9
RPE (6-20 point)	33	17.6	2.2
ECG Measures (mv):			
R wave amplitude			
Supine	89	1.45	0.46
Hyperventilation	89	1.32	0.46
Standing	51	1.24	0.44
Submaximal exercise	87	1.41	0.48
Peak exercise	84	1.28	0.45
J point amplitude			
Supine	89	-0.00	0.05
Hyperventilation	88	-0.01	0.05
Standing	51	-0.00	0.06
Submaximal exercise	85	-0.10	0.06
Peak exercise	80	-0.13	0.07
ST DEV @ .04 seconds*			
Supine	89	0.01	0.04
Hyperventilation	86	0.00	0.05
Standing	51	0.01	0.05
Submaximal exercise	85	-0.05	0.07
Peak exercise	78	-0.06	0.08
ST DEV @ .08 seconds**			
Supine	89	0.03	0.05
Hyperventilation	86	0.02	0.07
Standing	51	0.04	0.06
Submaximal exercise	85	0.03	0.10
Peak exercise	78	0.07	0.15
Recovery Blood Pressures:			
SBP (2 minutes)	94	161.8	21.4
DBP (2 minutes)	93	79.1	10.5
SBP (4 minutes)	93	140.3	17.0
DBP (4 minutes)	93	78.4	10.3

SBP = systolic blood pressure; DBP = diastolic blood pressure.

* S-T segment deviation 0.04 seconds from the J point.

** S-T segment deviation 0.08 seconds from the J point.

TABLE XXVII

MEANS OF GROUP S_TD_{AT} FOR INDICATORS OF MYOCARDIAL DYSFUNCTION

Variable	N=14	$\bar{X}$	SD
Testing Parameters:			
Functional Capacity	14	9.1	1.0
$\dot{V}O_2$ Peak	14	31.7	5.2
Peak Heart Rate	14	165.6	16.0
RPE (6-20 point)	5	18.0	1.0
ECG Measures (mv):			
R wave amplitude			
Supine	13	1.34	0.33
Hyperventilation	13	1.26	0.23
Standing	10	1.29	0.35
Submaximal exercise	13	1.30	0.30
Peak exercise	13	1.26	0.35
J point amplitude			
Supine	13	-0.01	0.04
Hyperventilation	13	-0.02	0.03
Standing	10	0.02	0.04
Submaximal exercise	13	-0.09	0.05
Peak exercise	13	-0.16	0.07
ST DEV @ .04 seconds*			
Supine	13	0.01	0.03
Hyperventilation	13	0.00	0.04
Standing	10	0.02	0.03
Submaximal exercise	13	-0.06	0.06
Peak exercise	13	-0.10	0.08
ST DEV @ .08 seconds**			
Supine	13	0.01	0.04
Hyperventilation	13	0.01	0.05
Standing	10	0.04	0.04
Submaximal exercise	13	0.00	0.09
Peak exercise	13	0.00	0.11
Recovery Blood Pressures:			
SBP (2 minutes)	14	172.5	30.6
DBP (2 minutes)	14	86.4	14.0
SBP (4 minutes)	14	148.0	29.1
DBP (4 minutes)	14	83.2	14.6

SBP = systolic blood pressure; DBP = diastolic blood pressure.

* S-T segment deviation 0.04 seconds from the J point.

** S-T segment deviation 0.08 seconds from the J point.

TABLE XXVIII

MEANS OF GROUP $S_{AT}D_T$ FOR INDICATORS OF MYOCARDIAL DYSFUNCTION

Variable	N=40	$\bar{X}$	SD
Testing Parameters:			
Functional Capacity	40	10.6	2.0
$\dot{V}O_2$ Peak	39	34.9	8.5
Peak Heart Rate	39	173.7	13.9
RPE (6-20 point)	23	17.1	2.0
ECG Measures (mv):			
R wave amplitude			
Supine	39	1.51	0.57
Hyperventilation	39	1.39	0.57
Standing	29	1.26	0.60
Submaximal exercise	35	1.42	0.53
Peak exercise	34	1.33	0.50
J point amplitude			
Supine	39	0.01	0.06
Hyperventilation	39	0.00	0.05
Standing	29	0.02	0.05
Submaximal exercise	35	-0.10	0.05
Peak exercise	34	-0.14	0.07
ST DEV @ .04 seconds*			
Supine	39	0.00	0.04
Hyperventilation	39	-0.00	0.03
Standing	29	0.01	0.04
Submaximal exercise	35	-0.07	0.06
Peak exercise	34	-0.07	0.07
ST DEV @ .08 seconds**			
Supine	39	0.02	0.05
Hyperventilation	39	0.01	0.04
Standing	29	0.03	0.05
Submaximal exercise	35	0.01	0.09
Peak exercise	32	0.04	0.17
Recovery Blood Pressures:			
SBP (2 minutes)	40	164.2	16.4
DBP (2 minutes)	40	82.5	9.5
SBP (4 minutes)	37	145.8	13.8
DBP (4 minutes)	37	82.1	8.5

SBP = systolic blood pressure; DBP = diastolic blood pressure.

* S-T segment deviation 0.04 seconds from the J point.

** S-T segment deviation 0.08 seconds from the J point.

TABLE XXIX

MEANS OF GROUP $S_{AT}D_{AT}$ FOR INDICATORS OF MYOCARDIAL DYSFUNCTION

Variable	N=12	$\bar{X}$	SD
Testing Parameters:			
Functional Capacity	12	9.7	1.8
$\dot{V}O_2$ Peak	12	33.4	8.4
Peak Heart Rate	12	166.8	15.8
RPE (6-20 point)	5	16.2	1.6
ECG Measures (mv):			
R wave amplitude			
Supine	12	1.36	0.45
Hyperventilation	11	1.21	0.36
Standing	10	1.29	0.42
Submaximal exercise	12	1.32	0.39
Peak exercise	11	1.23	0.39
J point amplitude			
Supine	12	0.01	0.06
Hyperventilation	11	-0.01	0.05
Standing	10	0.01	0.06
Submaximal exercise	12	-0.11	0.08
Peak exercise	11	-0.14	0.08
ST DEV @ .04 seconds*			
Supine	12	0.00	0.05
Hyperventilation	11	-0.00	0.04
Standing	10	0.00	0.06
Submaximal exercise	12	-0.06	0.10
Peak exercise	11	-0.07	0.06
St DEV @ .08 seconds**			
Supine	12	0.02	0.05
Hyperventilation	11	0.03	0.05
Standing	10	0.03	0.07
Submaximal exercise	12	0.01	0.11
Peak exercise	11	0.04	0.12
Recovery Blood Pressures:			
SBP (2 minutes)	12	176.6	33.4
DBP (2 minutes)	12	87.5	15.3
SBP (4 minutes)	12	150.7	22.3
DBP (4 minutes)	12	85.1	16.7

SBP = systolic blood pressure; DBP = diastolic blood pressure.

* S-T segment deviation 0.04 seconds from the J point.

** S-T segment deviation 0.08 seconds from the J point.

Appendix A

DETAILED METHODOLOGY

APPENDIX A

Selection of Subjects

Subjects were selected from the initial GXT's of all intervention participants' records in the Virginia Tech Intervention Exercise Program files. Only apparently healthy and hypertensive participants, classified as noncardiac and achieving a minimum of four completed exercise stages, were selected. Of the 234 male and female subjects initially reviewed, twenty-six were classified as cardiac. An additional 47 subjects were eliminated due to achievement of fewer than four exercise stages, or missing values for SBP, DBP, or both. All 161 subjects were tested at the Virginia Tech Intervention Center by their laboratory staff and a center-affiliated physician. Refer to Table I for the characteristic profile of the final subjects (N = 161).

Sampling Procedures

The Virginia Tech Intervention Center files were reviewed for all subjects who met the requirements set by the experimenter. Prior to performing the exercise test, each subject was referred by a physician to assure that he/she was a suitable candidate for a GXT. In addition, each subject signed an informed consent form prior to testing. Each candidate who met the experimenter-set requirements for the study was included in the sample.

General Methods

1. Measurements and Techniques

Subject Classification -- Based upon the physician referral, any subject with no diagnosed coronary artery disease was classified as noncardiac. Any subject with diagnosed coronary artery disease, based upon the physician referral, was classified as cardiac and dropped from the sample.

Categories of Disease -- Each subject's file records were reviewed to determine which of the following conditions existed as documented in the physicin referral form:

1. Myocardial infarction
2. Coronary artery bypass graft
3. Angina pectoris
4. Hypertension
5. Claudication
6. Asymptomatic coronary heart disease
7. Apparently healthy

Resting Blood Pressure -- Prior to exercise, resting blood pressure measurements were taken with the subject in supine and standing positions. Pressure was measured by auscultation using a mercury manometer, standard blood pressure cuff, and stethoscope. Blood pressure measurements were

observed in the left and right arms. Fourth phase Korotkoff sounds were recorded as diastolic blood pressure.

Exercise Blood Pressure -- Exercise blood pressure was measured by auscultation during the last minute of each stage of exercise during the GXT. Exercise blood pressure was measured using the left arm. Fourth phase Korotkoff sounds were recorded as diastolic blood pressure. Any questionable measurements were immediately repeated.

IPE Blood Pressure -- IPE blood pressure values were not considered by the researcher due to the variability in time lapsed and body position between subjects.

Recovery Blood Pressure -- Recovery blood pressures were recorded every two minutes, beginning two minutes post-exercise and ending six minutes post-exercise. Measurements were made by auscultation from the left arm of the subject. Diastolic blood pressure was recorded using the fourth phase Korotkoff sound.

ECG Measurements -- RAMP, JPT and STDEV were measured during resting supine, resting standing, hyperventilation, the submaximal EXSTAGE corresponding to 50-70% of the MET level of peak

exercise, and the peak EXSTAGE. Whenever possible, a value was averaged from three complexes. All measurements were made on lead V_5, recorded in millimeters and later converted to millivolts. All ECG values were estimated within one-half millimeter, with the P-R interval level of the tracing equalling zero millimeters. Measurements below the P-R interval were indicated with a negative sign (-), and all other numbers indicated above the P-R interval. The RAMP was measured beginning at the P-R interval level upward to the peak of the "R" wave. The JPT was measured from the P-R interval level upward or downward to the junction between the "S" wave and the S-T segment. The STDEV was measured at 0.04 and 0.08 seconds from the JPT, beginning at the P-R interval level and measuring upward or downward (Figure 3).

Arrhythmias and Blocks -- Any SVA, VA, or IVB were based upon the testing physician's evaluation and the experimenter's interpretation of the GXT. Presence of conditions were recorded only if considered to be either "more than isolated" or a permanent condition. These conditions are defined in Chapter I.

Functional Capacity -- The FC was estimated in

METs, based upon the speed of the treadmill and the percent grade at which the walking surface was set at the last completed exercise stage, and determined by the prediction formulas provided by the American College of Sports Medicine (ACSM, 1980). Test termination criteria were subject fatigue or physcian termination, based upon the guidelines for graded exercise testing setforth by the American College of Sports Medicine (ACSM, 1980). These include signs and symptoms of exertional intolerance, electrocardiographic changes, atypical blood pressure responses, inadequate heart rate responses, inadequate respirtory responses, or malfunctioning equipment.

Rating of Perceived Exertion -- Subjective RPE was assessed using Borg's 10- (range = 1 to 10) and 15- (range = 6 to 20) point scales. Subjects were asked to point to the number on a visual chart which best corresponded to their overall feelings of exertion at the end of each EXSTAGE. The numbers on the chart were provided with corresponding written cues which described increasing levels of physical exertion.

Signs and Symptoms of Exertional Intolerance -- Subjects were questioned immediately following test

termination to determine the most predominant factor(s) resulting in exertional intolerance. These signs and symptoms consisted of chest discomfort sufficient to cause the subject to focus upon it, dyspnea or shortness of breath, leg fatigue, general fatigue, or any other factors resulting in test termination.

Peak $\dot{V}O_2$ --Maximal oxygen consumption was determined by gas analysis using an open circuit gas analysis system with fast response O_2 and CO_2 electronic analyzers and either a Hewlett-Packard Pneumotach or Parkinson-Cowan dry gas meter system. If gas values by this method were unavailable, then the maximal oxygen consumption was estimated from FC using prediction formulas established by the American College of Sports Medicine (1980).

Serum Cholesterol, Triglyceride, and High-Density Lipoprotein Levels, and the Ratio Between Total Serum Cholesterol and High-Density Lipoprotein --Blood samples were drawn from each subject following a twelve-hour fast. Samples were drawn either the day of the GXT or within one week of the test. The majority of the blood samples were analyzed for total serum cholesterol, triglyceride, and high-density lipoprotein levels by the staff of

Roche Biomedical Laboratories, Inc.

Skinfold Assessment -- Anthropometric estimation by skinfold thickness was measured on each subject in conjunction with the GXT. Each skinfold site measurement was repeated three times with the average of the three values recorded. Harpenden calipers were used to measure three sites on each subject. These three sites differed between the genders. The male skinfold sites were the chest, abdomen, and thigh, while the female sites were the triceps, suprailiac, and thigh. All skinfold sites are defined in Chapter I.

Height -- In conjunction with the GXT, an anthropometer was used to measure each subject's height while wearing athletic or other low-heeled shoes. Measurements were made in centimeters to the nearest one-tenth centimeter and the measurement was converted to inches, to the nearest one-tenth inch.

Weight -- A medical beam balance was used at the time of the GXT to determine each subject's weight while wearing shoes. Each subject's weight was measured in kilograms to the nearest one-tenth kilogram and converted to pounds, to the nearest one-tenth pound.

Smoking -- Each subject was questioned concerning whether or not they smoked and if so, they were asked to disclose the quanity, frequency, and type of smoking. All types of smokers were recorded as smokers by the experimenter.

Medications -- Each subject's medications were recorded by the GXT administrators and the referring physician. Each medication was categorized into the following groups based upon the primary purpose of each subject being prescribed that medication as determined by the experimenter's interpretation of the physician referral and the disease status associated with each subject:

1. Antianginal
2. Antihypertensive
3. Antiarrhythmic
4. Digitalis
5. Anticoagulant
6. Antilipidemic
7. Psychotropic
8. Other medications
9. Cardiovascular modifying medications

Categories other than antihypertensive, digitalis, "other medications," and cardiovascular modifying

medications were later dropped from statistical analysis due to an absence of any prescribed medications in those categories. Subjects' prescribed medications were categorized and can be found in Table VII.

Percentage Body Fat -- Each subject's PFAT was estimated using the sum of the three skinfolds, gender, and age in accordance with the tables developed by Jackson and Pollock, 1978 and Jackson, Pollock and Ward, 1980.

Desirable Body Weight -- DESWT was estimated according to height, gender, and body weight in pounds using tables established by the Health and Nutrition Examination Survey for 1971-74 (Goldbeck, 1984). Since these tables utilized height measured without shoes, one inch was subtracted from each subject's height for determination of DESWT only.

2. Definition and Selection of Appropriate Criterion Scores for all Measures and Definitions for Computer Data Coding

IDENTIFICATION NUMBER -- Each subject was given a four-digit identification number in which the first three digits refer to the subject number and the last digit refers to the computer line of data; i.e., the number 0011 represents the first line of

data for the first subject. There were seven computer lines of data for each subject requiring the lines of data for the first subject to be 0011-0017, etc.

DATE -- The date of the initial GXT for each subject was recorded by the number of the month and the last two digits of the year; e.g., mmyy.

TESTSITE -- The site of the initial GXT for each subject was recorded as 1 if performed at Montgomery County Hospital or 0 if performed at the Virginia Tech Intervention Center . Following elimination of all ineligible subjects, the entire sample utilized in this research was tested at the Virginia Tech Intervention Center.

AGE -- The actual age in years at the time of the initial GXT was recorded for each subject.

GENDER -- The gender for each subject was recorded as male = 1 or female = 0.

CLASSIFICATION -- Each subject was categorized as either cardiac = 1 or noncardiac = 0. Those subjects categorized as cardiac were dropped from the sample.

CATEGORIES OF HEALTH/DISEASE -- The health/disease status of each subject was assessed based upon the physician referral, with a presence of each

condition recorded as = 1 or an absence of each condition recorded as = 0. The health/disease categories are defined in Chapter I.

RESTING BLOOD PRESSURE -- Prior to the GXT, resting blood pressure was measured twice in each position with a two-minute rest between measurements. The lower of the two values was recorded. Measurements were taken with the subject in the supine body position and then in the standing position. Values were recorded in millimeters of mercury. The only resting data point with an adequate number of responses was the supine measurement taken from the left arm.

EXERCISE BLOOD PRESSURE -- During the GXT, blood pressure measurements were taken only once at the end of each EXSTAGE. Only questionable readings were repeated immediately. One value was recorded for each EXSTAGE and measured in millimeters of mercury.

IPE BLOOD PRESSURE -- Immediate post-exercise blood pressure measurements were dropped from statistical analysis due to variability in the time-lapse and variability in body position between subjects..

RECOVERY BLOOD PRESSURE -- Blood pressure measurements were made at two-minute intervals

post-exercise. Only the two- and four-minute measurements were used in this research. Values were recorded in millimeters of mercury.

ECG MEASUREMENTS -- Electrocardiographic recordings were evaluated as previously described within one-half millimeter. All tracings were evaluated using using three complexes whenever possible, with an average of the three values recorded. The ECG changes were evaluated by computer using the following commands:

DIFF1 = RMAX - RSUP

DIFF2 = JMAX - JSUP

DIFF3 = ST04MAX - ST04SUP

DIFF4 = ST08MAX - ST08SUP

DIFF1A = RMAX - RSUBMAX

DIFF2A = JMAX - JSUBMAX

DIFF3A = ST04MAX - ST04SUBM

DIFF4A = ST08MAX - ST08SUBM

DIFF1B = RHYP - RSUP

DIFF2B = JHYP - JSUP

DIFF3B = ST04HYP - ST04SUP

DIFF4B = ST08HYP - ST08SUP

DIFF1C = RSUBMAX - RHYP

DIFF2C = JSUBMAX - JHYP

DIFF3C = ST04SUBM - ST04HYP

DIFF4C = ST08SUBM - ST08HYP,

where, DIFF1 always represented the difference between two RAMP measurements; DIFF2 always represented the difference between two JPT measurements; DIFF3 always represented the difference between two STDEV measurements at 0.04 seconds following the JPT; and DIFF4 always represented the difference between two STDEV measurements at 0.08 seconds following the JPT. The notation DIFF1 - DIFF4 with no corresponding letter notation represented the ECG difference between the peak EXSTAGE and the SUP position; DIFF1 - DIFF4 with a corresponding letter "A" represented the ECG difference between the MAX and the SUBMAX EXSTAGE; DIFF1 - DIFF4 with a corresponding letter "B" represented the ECG difference between the HYP and the SUP positions; DIFF1 - DIFF4 with a corresponding letter "C" represented the ECG difference between SUBMAX EXSTAGE and HYP. Although no ECG changes were significant, the greatest change was observed between the supine resting and peak exercise measurements. All other ECG changes were dropped from analysis.

SUBMAXIMAL HEART RATE -- Heart rate was obtained

from the GXT worksheet, and the corresponding ECG recording was examined by the experimenter for verification in the EXSTAGE corresponding to 50-70% of the MET level at peak exercise. Heart rate was recorded in beats per minute.

PEAK HEART RATE -- Heart rate was obtained from the GXT worksheet, and the corresponding ECG recording was examined by the experimenter for verification in the last completed EXSTAGE. Values were recorded in beats per minute.

SUPRAVENTRICULAR ARRHYTHMIAS -- SVA were recorded as presence = 1 if any of the defined SVA were determined by the testing physician or the experimenter to occur at a "more than isolated" frequency; absence = 0 if none or only isolated SVA were observed.

VENTRICULAR ARRHYTHMIAS -- VA were recorded as presence = 1 if any of the previously defined VA were determined by the testing physician or the experimenter to occur at a "more than isolated" frequency; absence = 0 if none or only isolated VA were observed.

INTRAVENTRICULAR BLOCKS -- IVB were recorded as presence = 1 if any of the previously described IVB were noted on the physician referral, by the

attending physician, or interpreted by the experimenter; absence = 0 if none observed.

FUNCTIONAL CAPACITY -- The FC was determined by the MET level corresponding to the last completed two-minute stage of exercise. The MET levels were estimated by prediction formulas supplied by ACSM. These formulas made use of the speed of the treadmill and the percent grade of the walking surface which was changed manually at the beginning of each EXSTAGE. The FC was recorded to the nearest one-tenth MET.

RATING OF PERCEIVED EXERTION -- At the end of each EXSTAGE, subjects were asked to point to a reference number on a visual chart to which the written expression of exertion corresponded closest to their overall physical feelings of exertion. Ratings by the subjects were verbally confirmed by one of the test administrators. Only the RPE corresponding to the last completed stage of exercise was recorded for use in this research.

SIGNS AND SYMPTOMS OF EXERTIONAL INTOLERANCE -- Upon completion of the GXT, each subject was questioned concerning the cause(s) for termination of exercise. Each previously described sign and symptom was recorded as presence = 1 if experienced

by the subject, or absence = 0 if not experienced by the subject.

PEAK $\dot{V}O_2$ -- Peak oxygen consumption was either measured by gas analysis or estimated from FC using calculation formulas developed by ACSM. Maximal oxygen consumption was recorded in millimeters of oxygen per kilogram of body weight per minute and recorded to the nearest one-tenth.

CHOLESTEROL LEVEL -- Blood samples were analyzed by Roche Biomedical Laboratories, Inc. and reported in milligrams per deci-liter. Kannel (1983) has stated that optimal CHOL values for prevention of CHD are between 140 and 180 mg/dl, while those in the range of 180 to 200 mg/dl are also favorable, with values higher than 200 mg/dl appearing to be suboptimal in regard to cardiovascular health.

TRIGLYCERIDE LEVEL -- Blood samples were analyzed by Roche Biomedical Laboratories, Inc. and reported in milligrams per deci-liter. Guyton (1976) has stated that average TRIG concentrations are considered to be 160 mg/dl, with increasing values associated with increasing risk of CHD.

HIGH-DENSITY LIPOPROTEIN LEVEL -- Blood samples were analyzed by Roche Biomedical Laboratories, Inc. and recorded in milligrams per deci-liter.

Persons having HDL-C levels below 35 mg/dl have been associated with a CHD incidence rate of more than eight times that of persons having levels of 65 mg/dl or above (Gordon et al., 1977).

TOTAL SERUM CHOLESTEROL/HIGH-DENSITY LIPOPROTEIN RATIO -- In order to determine TC/HDL-C, the experimenter divided the CHOL level by the HDL-C level from blood samples analyzed as previously described. A TC/HDL-C ratio of 5.0 is associated with average risk of CHD; optimal ratios, around 3.5, correspond to half the standard risk (Kannel, 1983).

SKINFOLD ASSESSMENT -- Each skinfold site was measured three times and the average of the three trials was recorded to the nearest one-tenth.

HEIGHT -- In conjunction with the GXT, height was measured in centimeters, then converted and recorded in inches. Height was later converted back to centimeters by the researcher. All subjects were measured wearing athletic shoes. Values were recorded to the nearest one-tenth.

WEIGHT -- Body weight was measured in kilograms and converted to pounds. Subjects were weighed wearing athletic shoes and clothing. Values were recorded in both kilograms (WTKG) and pounds (WTLB) to the

nearest one-tenth.

SMOKE -- Cigarette, cigar and pipe smokers were recorded as = 1, while non-smokers were recorded as = 0.

MEDICATIONS -- Each subject's file was reviewed for use of any medications. Each of the medication categories previously described were evaluated in relation to the information obtained from the file records. Usage of any medication in each category was recorded as presence = 1; while nonusage of any medication in each category was recorded as absence = 0. Medications were further categorized into either modifiers of cardiovascular function (CMOD) = 1, or non-modifiers of cardiovascular function (CMOD) = 0.

PERCENTAGE BODY FAT -- PFAT was estimated using each subject's sum of three skinfold sites, gender and age in accordance with the tables supplied by Jackson and Pollock, 1978 and Jackson, Pollock and Ward, 1980. Values were recorded to the nearest one-tenth.

DESIRABLE BODY WEIGHT -- DESWT was estimated by using each subject's height in inches, age and gender in accordance with the table developed by the Health and Nutrition Examination Survey,

1971-74 (Goldbeck, 1984). Due to subjects wearing athletic shoes, one inch was subtracted from each subject's height for determination of this variable only. Values were recorded to the nearest pound.

RELATIVE WEIGHT -- RELWT was calculated by the computer using the formula: weight (pounds)/ DESWT X 100. Values were recorded to the nearest one-tenth pound (Goldbeck, 1984).

Experimental Procedures

Screening -- All records in the Virginia Tech Intervention Center files were reviewed for all possible candidates. All subjects who met the previously described requirements set by the experimenter were used in the study.

Determination of Dependent Variables -- Classification of health/disease and categories of health/disease were based upon the physician referral and recorded by the experimenter as previously described. All blood pressure measurements, all heart rates, all MET levels, ratings of perceived exertion, peak $\dot{V}O_2$, cholesterol level, triglyceride level, HDL-C level, skinfold assessment, height, weight, and smoking habits were obtained from the GXT summary sheet or GXT worksheets. Medication information was obtained from the physician referral, physician GXT interpretation sheet, and the GXT worksheets. The primary purpose for each subject's prescribed medication was

determined by the experimenter based upon the physician referral and/or medical history information available in the subject's records. Arrhythmias and heart blocks were determined by the testing or referring physician and verified by the experimenter. All ECG measurements were performed by the experimenter from the actual ECG tracings from the GXT. Percentage body fat, desirable body weight, relative body weight, and total serum cholesterol/HDL-C ratio were obtained by the experimenter using the subject information in the file records and the estimation tables previously described.

Determination of Typical and Atypical Exercise Blood Pressure Responses -- Atypical systolic blood pressure response to exercise was defined as either a decrease in pressure > 4 mmHg, no change in pressure beyond ± 4 mmHg, or a rise ≥ 30 mmHG between the last two measured blood pressure responses during the GXT. All measurements corresponded to the last two completed stages of exercise. All other systolic responses were considered to be typical. Atypical diastolic blood pressure response to exercise was defined as a level ≥ 110 mmHg in the final completed stage of exercise or an increase ≥ 10 mmHg between the last two measured blood pressure responses. All measurements corresponded to the last two completed stages of exercise. All other diastolic responses were considered to be typical.

Exercise Blood Pressure Response Classification by Groups -- Four blood pressure response groups were established from the systolic and diastolic responses to exercise. Refer to above section for criteria applied to separate typical and atypical SBP and DBP changes. Each subject was placed into one of these groups. These groups were:

1. Typical systolic and typical diastolic (TSTD or S_TD_T).
2. Typical systolic and atypical diastolic (TSATD or S_TD_{AT}).
3. Atypical systolic and typical diastolic (ATSTD or $S_{AT}D_T$).
4. Atypical systolic and atypical diastolic (ATSATD or $S_{AT}D_{AT}$).

Statistical Procedures

The data were analyzed by computer using Statistical Analysis System (SAS). Descriptive statistics were used to describe the sample population and to describe the incidence of atypical systolic and diastolic blood pressure responses to exercise. The statistics used included means, standard deviations, frequency counts, and percentages.

Chi-square analyses were used to determine statistically significant differences between the four blood pressure response groups on categorically measured variables

such as gender, health status, signs and symptoms of exertional intolerance, etc. The one-way analysis of variance (ANOVA) was used to determine statistically significant differences between the four blood pressure response groups on continuous variables such as age, blood pressures, functional capacity, blood lipid levels, etc. In the ANOVA analysis, if a significant F ratio was revealed ($p < .05$), then the Tukey post-hoc procedure was performed in order to determine where the differences occurred.

Appendix B

INFORMED CONSENT

INFORMED CONSENT FOR PATIENTS WITHOUT KNOWN HEART DISEASE
CARDIAC & INTERVENTION CENTER AT VIRGINIA TECH

1. EXPLANATION OF TESTS AND BENEFITS TO BE EXPECTED

IN ORDER TO DETERMINE AN APPROPRIATE PLAN OF MEDICAL MANAGEMENT, I HEREBY CONSENT TO VOLUNTARILY ENGAGE IN AN EXERCISE TEST TO DETERMINE THE STATE OF MY HEART AND CIRCULATION. I ALSO CONSENT TO HAVE A BLOOD SAMPLE DRAWN FOR BLOOD CHEMISTRY ANALYSIS AND TO THE PERFORMANCE OF A LUNG FUNCTION TEST AND A BODY FAT ANALYSIS. THE INFORMATION THUS OBTAINED WILL HELP MY PHYSICIAN IN ADVISING ME AS TO THE ACTIVITIES IN WHICH I MAY ENGAGE.

BEFORE I UNDERGO THE TEST, I WILL BE INTERVIEWED BY A PHYSICIAN. MY RECORDS WILL ALSO BE REVIEWED TO DETERMINE IF ANY CONDITION EXISTS THAT WOULD CONTRA-INDICATE THE PERFORMANCE OF THE TEST. THE TEST WHICH I WILL UNDERGO WILL BE PERFORMED ON A QUINTON TREADMILL WITH THE AMOUNT OF EFFORT INCREASING GRADUALLY. THIS INCREASE IN EFFORT WILL CONTINUE UNTIL SYMPTOMS SUCH AS FATIGUE, SHORTNESS OF BREATH, OR CHEST DISCOMFORT MAY APPEAR. AT THAT POINT THE TREADMILL WILL BE SLOWED OR STOPPED.

DURING THE PERFORMANCE OF THE TEST, THE PHYSICIAN OR HIS TRAINED OBSERVER WILL OBSERVE MY PULSE, BLOOD PRESSURE, AND ELECTROCARDIOGRAM. A SAMPLE OF MY EXHALED AIR MAY BE COLLECTED IN ORDER TO MEASURE OXYGEN CONSUMPTION.

2. RISKS

THERE EXISTS THE POSSIBILITY OF ADVERSE CHANGES OCCURRING DURING THE TEST. THESE COULD INCLUDE ABNORMAL BLOOD PRESSURE, FAINTING, DISORDERS OF HEART RHYTHM, AND VERY RARE INSTANCES OF HEART ATTACK. EVERY EFFORT WILL BE MADE TO MINIMIZE THESE BY PRELIMINARY EXAMINATION AND BY OBSERVATIONS DURING THE TEST. EMERGENCY EQUIPMENT AND TRAINED PERSONNEL ARE AVAILABLE TO DEAL WITH THE UNUSUAL SITUATIONS WHICH MAY ARISE.

3. CONFIDENTIALITY AND USES OF INFORMATION

THE INFORMATION WHICH IS OBTAINED IN THIS TEST WILL BE TREATED AS PRIVILEGED AND CONFIDENTIAL AND WILL NOT BE RELEASED OR REVEALED TO ANY PERSON WITHOUT MY EXPRESS WRITTEN CONSENT. IF A PARTICIPATE IN THE HEART DISEASE INTERVENTION PROGRAM, I FURTHER UNDERSTAND THAT ANY INFORMATION ABOUT MY PARTICIPATION THEREIN OR ABOUT MY FUTURE HEALTH OR WORK STATUS WILL ALSO BE TREATED AS PRIVILEGED AND CONFIDENTIAL. THE INFORMATION OBTAINED, HOWEVER, MAY BE USED FOR STATISTICAL OR SCIENTIFIC PURPOSES WITH MY RIGHT OF PRIVACY RETAINED.

4. RELEASE

I HEREBY RELEASE VIRGINIA TECH AND ALL ITS OFFICERS, AGENTS, AND EMPLOYEES FROM ALL LIABILITY AND RESPONSIBILITY FOR ANY INJURY, ILLNESS, OR SIMILAR OCCURRENCE, INCLUDING HEART ATTACK OR ITS RESULTANT COMPLICATIONS, WHICH MIGHT ARISE OUT OF OR RESULT FROM MY PARTICIPATION IN THESE TESTS OR IN THE HEART DISEASE INTERVENTION PROGRAM.

5. INQUIRIES AND FREEDOM OF CONSENT

I UNDERSTAND THAT MY PARTICIPATION IN THESE TESTS IS VOLUNTARY AND THAT I AM FREE TO WITHDRAW AT ANY TIME. I HAVE READ THIS FORM AND I UNDERSTAND THE TEST PROCEDURES THAT I WILL PERFORM AND CONSENT TO PARTICIPATE IN THIS TEST AND IF THE ATTENDING PHYSICIAN SO ADVISES, IN THE HEART DISEASE INTERVENTION PROGRAM.

SIGNATURE OF PARTICIPANT	DATE
WITNESS (PROGRAM STAFF)	PHYSICIAN SUPERVISING TEST

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