INFLUENCE OF SYSTOLIC BLOOD PRESSURE ON ECG ST SEGMENT RESPONSES IN EXERCISE TESTS OF ADULTS WITHOUT DIAGNOSED CHD

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(ABSTRACT)

Records from treadmill maximal graded exercise tests (GXTs) for 61 patients from the Virginia Tech Intervention Center were screened for changes in systolic blood pressure. These blood pressure responses were standardized according to exercise demand (ΔSBP/MET) between three different levels of the exercise test. Subject records were chosen on the basis that they did not reflect a physician diagnosis of coronary artery disease (CAD) and were not taking antihypertensive medications. The ΔSBP/MET responses were stratified as follows: low to moderate (ΔBP/MET1) = difference between a systolic blood pressure at a moderate intensity stage minus the first stage systolic blood pressure, adjusted for the corresponding changes in metabolic demand (MET); moderate to high (ΔBP/MET2) = difference between systolic blood pressure at the maximal stage minus the moderate intensity stage per MET change; and low to high (ΔBP/MET3) = difference between systolic blood pressure at the maximal stage minus the first stage per MET change. Subjects were separated (STΔ and NoSTΔ) according to whether or not they had exercise induced ST segment shift of 1 mm ($\geq$ 0.1 mV) at maximal exercise. The two groups were similar in physical characteristics, except the NoSTΔ group had a significantly higher BMI (Body Mass Index), were a few years younger and exhibited a lower RPP at maximal effort. Discriminant Function Analysis was used to predict group classification of individual

patients (STΔ or NoSTΔ). Based on predictions using physical characteristics alone, (age, BMI, TC), age, BMI and TC (Total Blood Cholesterol) could correctly predicted classification in 66% of the cases. The set of age, BMI, TC and ΔBP/MET3 (low to high) generated a prediction with 77% correct classification. Thus, ΔBP/MET level alone was not the primary variable to explain predictive accuracy for clinically important ST changes in exercise testing. However, in accordance with the Bayesian principle, this hemodynamic exercise response is adjusted for overall metabolic demand in the test and coupled to markers of pre-test coronary risk, the ability to predict ST response is improved.

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Chapter I

INTRODUCTION

Statement of the Problem

Coronary Artery Disease (CAD), the leading cause of death in the United States, is presently incurable. In most Western countries, atherosclerosis, one of the important precipitation factors in primary hypertension and CAD, begins during childhood, with 75% of males developing CAD 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 CAD morbidity/mortality in the country (Froelicher, Thompson, Longo, Triebwasser & Lancaster, 1976). However, it appears practical to evaluate the screening techniques presently available for detection of subclinical or latent CAD to enhance the current trend toward lower incidence of CAD.

Since CAD 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, recent emphasis in "prevention-oriented" investigations has been given to improving the specificity, predictive value, and sensitivity of screening techniques for detecting individuals at "high-risk" for future CAD 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 CAD. Sensitivity refers to the ability of a given

test to identify "true-positive" cases (positive test indication of CAD in a patient with CAD), while specificity refers to a test's ability to detect the "true-negatives" (no indication of CAD in a patient without CAD) (ACSM 1995). 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. Perhaps a greater importance in screening techniques is the "risk ratio," which incorporates sensitivity, specificity, and predictive value. This 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 on 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 CAD are not known, a number of risk factors have long been recognized as valid and easily assessed indices for determining the likelihood of CAD or its early development. Among these are resting hypertension, hyperlipidemia, smoking, obesity, family history, gender, sedentary lifestyle and tension/stress (ACSM 1995). However, several researchers have determined that simple identification of these factors is especially inadequate when screening an asymptomatic population for latent CAD (Froelicher et al., 1976; Froelicher, Thomas, Pillow, & Lancaster, 1974; Jackson, Squires, Grimes, & Beard, 1983). Therefore, realizing that it is undoubtedly advantageous, although more difficult, to detect those at high risk for CAD 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) responses to exercise has been demonstrated to identify groups of men at high risk for CHD (Froelicher et al., 1974). Unfortunately, an abnormal ECG response does not precisely predict a future cardiac event, nor does a normal exercise ECG response rule out this possibility.

In healthy individuals, graded treadmill exercise evokes a moderate and progressive rise in systolic blood pressure. Deviations in systolic blood pressure responses from the normal pattern, particularly those accepted as indicative of "exertional hypotension" have been studied extensively by clinical investigators. (Morris, 1978; Thomson & Kelemen, 1975 and Li, Riggins & Anderson, 1979) Findings from these studies had a connection with the diagnosis of coronary artery disease (CAD), hypertension and cardiac vascular disorders, as well as in the prognosis of cardiac morbidity and mortality. Investigators have concluded that certain atypical responses in systolic blood pressure during and immediately after graded exercise testing are more sensitive to change than ECG changes for identifying patients with coronary heart disease.

Some investigators (Bruce, 1977; Amon, 1984; Hossak & Bruce, 1985) have concluded that certain atypical systolic blood pressure responses during and immediately after graded exercise tests may be actually more sensitive than exercise electrocardiographic changes and angina for identifying patients with coronary artery disease. There is evidence supporting the utility of such responses in predicting future resting hypertension.

In the administration of symptom-limited exercise testing, interpretation of blood pressure changes is of immediate concern to clinicians who must decide upon test termination. Continuation of the test provides the opportunity to gain valuable clinical information affecting patient treatment protocols or to improve the accuracy of cardiorespiratory fitness determinations. Conversely, favoring test termination is the recognition that presentation of certain systolic blood pressure deviations carries acute risks (Froelicher, 1994). With the substantial increase in the administration of graded exercise tests for both apparently healthy adults and diseased populations, further examination of systolic blood pressure response patterns is needed.

Mean arterial blood pressure is the product of cardiac output and peripheral vascular resistance. Systolic pressure rises in a linear fashion as a function of exercise intensity, but diastolic pressure either declines moderately or remains unchanged from resting values (ACSM 1991). The highest systolic blood pressure should be achieved at maximal workload. Despite studies showing discrepancies between non-invasively and invasively measured blood pressure, the product of heart rate and systolic blood pressure, determined by cuff and auscultation, correlates with measured myocardial oxygen consumption during exercise (ACSM 1994). Usually, a patient's angina pectoris and abnormal ST-segment depression are precipitated at the same double product (systolic blood pressure multiplied by heart rate multiplied by 10^{-3}). This product also is a non-invasive estimate of the maximal workload that the left ventricle can perform. Although automated methods of measuring blood pressure may correlate with manual methods, they have not

been proven accurate for exercise testing, particularly for the detection of exertional hypotension.

Exercise-induced hypotension (EIH) has been demonstrated in most studies to predict a poor prognosis for relative to cardiac mortality. Although the prognosis of EIH has not been specifically examined after a myocardial infarction, an abnormal systolic blood pressure response indicates an increased risk for cardiac events. Exercise induced decreases in systolic blood pressure can occur in patients with coronary artery disease, valvular heart disease, cardiomyopathies, and arrhythmias. EIH is usually related to myocardial ischemia or a ventricle significantly compromised by myocardial infarction; prognostically, it is most often defined as a drop of systolic blood pressure during exercise below the standing pre-exercise value, and indicates a significantly increased risk for premature cardiac mortality (Froelicher, 1994).

Blood pressure responses to graded exercise has also been shown to have predictive validity with respect to future development of resting hypertension and CHD, even on the absence of other predisposing risk factors (Dahms et al., 1978; Irving, Bruce, & DeRouen, 1977; Jackson et al., 1983; 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 hyper-response, especially in systolic pressure, and the development of resting hypertension within 2-5 years. In addition, Irving and associates (1977) have found that absence of systolic blood pressure increases during exercise, probably due to left ventricular dysfunction, is a more accurate

predictor of mortality due to CAD than ST segment changes in the exercise electrocardiogram. Other researchers (Morris, Phillips, Jordan & McHenry, 1978; Thomson & Kelemen, 1975) have reported that a decrease in exertional systolic blood pressure with exercise is a reliable sign of multiple vessel coronary artery disease. With respect to diastolic pressure, Froelicher (1983) suggested that a rising diastolic blood pressure in treadmill exercise is indicative of CAD, while Irving et. al. (1977) found fourth and fifth phase Korotoff sound difficult to detect during exercise, therefore diminishing the useful interpretation of exercise diastolic responses. Using exercise blood pressure responses in combination with CAD risk factors to predict the future development of resting hypertension and CAD may prove invaluable in improving the clinical management of apparently healthy individuals by possibly preventing the occurrence of CAD. Bruce et al. (1985) concluded that certain atypical systolic blood pressure responses during and immediately after GXTs may be actually more sensitive in identifying patients with CAD that exercise electrocardiographic changes.

Atypical blood pressure responses to exercise may not arouse concern in all types of individuals or under all exercise conditions. Sudden systolic hypotension in exercise may occur as a result of certain medications, left ventricular impairment, or coronary artery narrowing in the diseased individual, but may occur naturally in healthy individuals during maximal exhaustive exercise (Morris et al., 1978; Thomson & Kelemen, 1975). With respect to immediate test safety, these hypotensive responses represent a major concern in the diseased individual. Also, systolic hypertensive responses to exercise in those with clinical heart disease have been considered beneficial

and associated with very low mortality risk (Irving et al., 1977), whereas hypertensive responses 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, 1995), judgment of test safety may be improved.

Research Objectives

Thus, the purpose of this study was to determine whether the progression of systolic blood pressure might contribute to prediction of presence or absence of clinically important ST segment shifts in the ECG responses of adults undergoing graded exercise testing. A second objective was to determine if those manifesting atypical blood pressure responses to exercise also exhibited relatively more predisposing CAD risk, or if their exercise tests show any signs or symptoms of abnormal ST responses. The specific objectives were to:

1. To examine the differences in predisposing CAD risk factors and exercise systolic blood pressure per MET level responses in groups according to ST segment shift in the hopes of creating a prediction tool for the exercise testing setting;

2. To determine whether the progression of systolic blood pressure might contribute to prediction of presence or absence of clinically important ST segment shifts in the electrocardiographic responses during GXTs.;

3. To determine the possible influence of different predictors including physical and exercise variables during a GXT on ST segment electrocardiographic responses.

Significance of the Study

Mean arterial blood pressure is the product of cardiac output and peripheral vascular resistance. Systolic pressure rises in a linear fashion as a function of exercise intensity, but diastolic pressure either declines moderately or remains unchanged from resting values. (ACSM, 1995) The highest systolic blood pressure should be achieved at maximal workload. Despite studies showing discrepancies between non-invasively and invasively measured blood pressure, the product of heart rate and systolic blood pressure, determined by cuff and auscultation, correlates with measured myocardial oxygen consumption during exercise. (ACSM, 1994) Usually, a patient's angina pectoris and abnormal ST-segment depression are precipitated at the same double product (systolic blood pressure multiplied by heart rate multiplied by 10^{-3}). This product is also a non-invasive estimate of the maximal workload that the left ventricle can perform. Although automated methods of measuring blood pressure may correlate with manual methods, they have not been proven accurate, particularly for the detection of exertional hypotension.

Exercise-induced hypotension (EIH) has been demonstrated in most studies to predict a poor prognosis of coronary heart disease. Although the prognosis of EIH has not been specifically examined after a myocardial infarction, an abnormal systolic blood pressure response indicates an increased risk for cardiac events. Exercise induced decreases in systolic blood pressure

can occur in patients with coronary artery disease, valvular heart disease, cardiomyopathies, and arrhythmias. EIH is usually related to myocardial ischemia or a ventricle compromised by myocardial infarction, is best defined as a drop of systolic blood pressure during exercise below the standing pre-exercise value, and indicates a significantly increased risk for premature cardiac mortality (Froelicher, 1994).

Research Hypothesis

Ho: The progression of systolic blood pressure in graded exercise tests, with or without the inclusion of other pre-exercise characteristics, does not provide a means to predict those who will have ST segment shifts at peak exercise in adults without diagnosed coronary artery disease.

Delimitations

The following delimitations were imposed:

1. The study was confined to the records of male and female adults who participated in the Virginia Tech Cardiac Therapy and Intervention Program between April, 1977 until August, 1995.

2. The study was confined to participants without a recorded physician diagnosis of CAD or hypertension.

3. The study was confined to participants who were not receiving any prescribed anti-hypertensive, beta-blocker, calcium channel blocker, long-acting nitrate, or digitalis medications.

4. The data for each subject was confined to the physician referral information and to the results from their second maximal effort graded exercise test (GXT) given at the Laboratory for Health and Exercise Sciences.

5. The study was confined to participants tested at the Virginia Tech Cardiac Therapy and Intervention Center on Monday, Wednesday or Friday mornings between 7:00 and 8:00 am using individualized graded exercise treadmill protocols which increased every 2-3 minutes by 1-3 METs (predicted).

Limitations

The following factors were viewed as important constraints that limited interpretation of the data:

1. The possibility that some inconsistency occurred with respect to different technicians recording systolic blood pressure during the graded exercise tests.

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

Definitions and Symbols

DATE - the date of the follow-up test (6 month or a year) by month and year

DEMOGRAPHICS-

AGE- age in years at the time of the test

GENDER - gender of subject

WT - weight in kilograms

HT- height on centimeters

BMI- body mass index [WT(kg)/ HT(cm^2)

Hx Smoking - history of smoking

BLOOD CHEMISTRY -

CHOL- cholesterol

BLOOD PRESSURE MEASUREMENTS

Hx HTN - history of hypertension (from physician referral)

Rest HTN - resting hypertension (>140/90)

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)

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)

BPst - Blood pressure measured while patient is standing at rest.

BPsu - Blood pressure measured while patient is supine before exercise.

EXERCISE BLOOD PRESSURE GROUPINGS

ΔBP/MET1 - low to moderate = systolic blood pressure at a submaximal level minus the first level; divided by corresponding MET level.

ΔBP/MET2 - moderate to high = systolic blood pressure at maximal level minus moderate level; divided by corresponding MET level.

ΔBP/MET3 - low to high = maximal systolic blood pressure level minus first level; divided by corresponding MET level.

EXERCISE TESTING-

GXT- graded exercise test

HRst - Heart rate measured while patients is standing at rest.

HRsu - Heart rate measured while patient is supine before 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.

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. (ACSM 1991)

RPE - rating of perceived exertion; a subjective response to the subjects being tested as to his/her overall physical feeling during the GXT.

ECG MEASUREMENTS-

ECG - electrocardiogram

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

J60 - one and a half boxes over from the J point on the ECG

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

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 resting blood pressure measurements were made in the supine body position, unless otherwise stated.

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

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

6. 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.

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

8. It is assumed that all GXTs were maximal effort or symptom-limited.

Chapter II

REVIEW OF THE LITERATURE

Introduction

This review, divided into three major areas, progresses from a discussion of blood pressure and regulation at rest to that during exercise and finally a discussion on the significance of various systolic blood pressure responses. 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 and whether the progression of systolic blood pressure might contribute to prediction of presence or absence of clinically important ST segment shifts in the ECG responses of adults undergoing graded exercise testing. Topics include: responses of healthy and hypertensive adults, influences of physical training, theories of blood pressure control, significance of various systolic responses, clinical standards associated with testing, and the use of exercise testing in detection of latent hypertension.

The third section discusses the significance of excessive rises and hypotensive responses of systolic blood pressure, the blood pressure standards for clinical exercise testing and the prospects for detecting future hypertension through exercise testing.

Blood Pressure and Its Regulation
Under Non-Exercise Conditions

Basic Considerations

Blood pressure is the force exerted by the blood against the vessel wall (Taylor & Best, 1991). 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 sound) or Phase V (disappearance of sound) Korotkoff sounds. Although opinions of various researchers differ, the American Heart Association (AHA, 1985) and the World Heart Association have accepted the point of muffling as the definitive diastolic measurement, as it more closely correlates with direct measurements.

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 (Taylor & Best, 1991). However, resting systolic values as high as 140 mmHg and diastolic values less than 90 mmHG have widely been considered normal (Wilson & Meyer, 1981). Hypertension, or high arterial blood pressure, has been arbitrarily defined by many authorities as a systolic blood pressure of 160 mmHg or greater and/or a diastolic pressure exceeding 95 mmHg (Sparrow et al., 1982; Levenson & Safar, 1982). Blood pressures intermediately between 140/90 mmHg and 160/95 mmHg have been designated as "borderline" hypertension (Froelicher, 1994). In the past the term "labile" hypertension has been confused with the definition of borderline hypertension . 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; Wolthuis, Froelicher, Fischer, & Triebwasser, 1977).

Environmental Factors

There are several environmental factors that are known to influence blood pressure at rest. Resting blood pressure may rise simply due to the nervousness and anxiety associated with the measurement (Stegmann, 1981). Resting values are influenced by altitude and temperature. The effects of gravity during standing and tilt table test are particularly distressing because on the table, heart rate increases and systolic blood pressure falls until the subject faints (Brooks & Fahey, 1984).

An investigation involving 4,634 men and women established an effect on systolic and diastolic pressures based on seasons (Khaw, Barrett-Conner & Suarez, 1984). There was a significant difference between the highest value 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 who also reported the lowest pressures in the summer months.

In a study limited to 10 subjects, Reilly, Robinson, and Minors (1984) examined blood pressures 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.

Control of Blood Pressure in Normotension

Taylor and Best (1991) 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 and 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 artial receptors operate somewhat differently. The artial 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 in the urine (Taylor & Best, 1991).

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 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 and epinephrine, renin-angiotensin, and vasopressin vasoconstriction mechanisms. The norepinephrine-epinephrine hormones are released into the blood from the adrenal medulla following sympathetic nervous system stimulation. These hormones cause essentially the same effects as direct sympathetic stimulation, (i.e., excitation of the heart and constriction of 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 the hypothalamus, also acts as a vasoconstrictor when arterial pressure becomes too low, but has less power than angiotensin II (Taylor & Best, 1991).

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 mechanisms 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 quantities 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). 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 (1991), it was determined that essential hypertension is associated with normal or low levels of circulatory renin, rather that high renin levels.

Investigators cited by Guyton (1991) 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

Theories of Control

When exercise is initiated, possible 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 more arterioles and capillaries. This local control is the most important factor in attainment 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 the major part of the vascular dilation accompanying muscle activity. In the inactive muscles, the sympathetic vasodilator fibers are inactive, and hormonal activity contributes to vasoconstriction in these muscles.

As exercise continues, 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).

Although the specific mechanisms by which exercise blood pressure is regulated are currently uncertain, a number of hypotheses have been presented. 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 abundant in slow-twitch fibers. In addition, they observed a higher percentage of fast-twitch fibers in patients with essential hypertension.

Responses of Healthy Adults

Normally, there is a linear rise in the systolic blood pressure with gradual increases in dynamic exercise (Froelicher, 1983). 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 (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) reported 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) which involved three progressive age decades (25-34, 35-44, and 45-54 yrs.). Maximal systolic pressure increased slightly across the three age groups (164-212, 164-214, and 162-216 mmHg, respectively), as did maximum diastolic pressure (60-90, 64-94, and 68-96 mmHg, respectively).

Bruce et al. (1974) reported average resting blood pressure of healthy men to be 123 ± 13 mmHg systolic and 77 ± 10 mmHg diastolic (values are mean ± SD). 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 observed 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 further decline at minute five. This approximated average pre-exercise values, with systolic 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 pressure responses similar to those of normotensive adults, whereas hypertensive adults who are older or have more severe hypertension tend to exhibit greater rises in both systolic and diastolic blood pressure than normotensive adults (Aoki et al., 1982). 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 bring about erroneous values.

Smith et al. (1992) used multiple stepwise regression analysis that revealed that pre-exercise systolic blood pressure was a major determinant of maximal exercise systolic blood pressure indicating that higher baseline blood pressure predicted higher blood pressure readings. When compared with normotensive subjects, hypertensive patients had lower measures of maximal oxygen uptake and exercise heart rates. This may indicate lower cardiac performance at maximal exercise and explain the reduced capacity of hypertensive patients with left ventricular hypertrophy to increased systolic blood pressure with exercise.

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 readings of hypertension exhibited significantly steeper systolic pressures than normotensive controls. Exercise systolic blood pressure 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.

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 pressure in the two older groups revealed a greater rise than in the normotensive subjects of the same age. 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 depicted limits in this investigation of air crewmen.

Physical Training Influences

Numerous studies have been conducted to test the advantages and disadvantages of physical training on normotensive and hypertensive individuals and CHD patients, yet conflicting data fall short of proving that an increase in regular physical activity is actually beneficial for all types of blood pressures (Tipton, 1984). Fox and Naughton (1972) dispute that the benefits

may be as much or more in the area of an improved quality of life as in the extension of life. Tipton (1984) 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 is 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 another investigation by Boyer and Kasch (1970) were consistent with his claim only with respect to blood pressure. They concluded that hypertensive patients may be trained without undue risk and that interval exercise is of enhanced value in the therapy of hypertensive middle-aged men.

Likewise, Brooks and Fahey (1984) state that endurance training tends to reduce resting and submaximal exercise systolic, diastolic, and mean blood pressures. Although the mechanism for the response pattern with training closely imitates the hemodynamic changes 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 inactivity. Nevertheless, he advocated a gentle progression of physical activity for both apparently healthy and at risk patients, claiming that a cardiac emergency 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.

Significance of Various Systolic Blood Pressure Responses

Blood Pressure Standards for Clinical Exercise Testing

In the fifth edition of Guidelines for Graded Exercise Testing and Exercise Prescription, ACSM (1995) 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 (1995) has determined that a failure of systolic pressure 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 260 mm Hg 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 115 mmHg may be an indication to terminate exercise testing.

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 judgment.

Investigators have witnesses 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 rise in diastolic pressure. These atypical blood pressure responses have been associated with 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 blood pressure response appears to be an excessive increase in pressure. Filipovsky et al. (1992) found that exercise induced increase in systolic blood pressure was positively correlated with resting systolic blood pressure, whereas the correlation of exercise heart rate increase with resting heart rate was negative. They concluded that the magnitude of the exercise-induced increase of systolic blood pressure, but not of heart rate, may represent a risk factor for death from cardiovascular as well

as non cardiovascular causes, independently of resting blood pressure and heart rate.

Mundal et al. (1994) concluded that an early rise of systolic blood pressure during exercise adds prognostic information on cardiovascular mortality among otherwise healthy middle aged men with mildly elevated casual blood pressure. Excessive systolic responses to exercise have been reported to occur in hypertensive subjects (Irving et al., 1977; Sannerstedt, 1981). Amery et al. (1967) found that older hypertensive patients demonstrated a greater rise in systolic pressure with exercise that normotensive subjects. 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.

Naimark et al. (1990) had findings that indicated evidence of target organ involvement associated with increased exercise systolic blood pressure lend further support to the notion that blood pressure levels during exercise have important diagnostic and therapeutic implications. Aoki et al. (1982) that a greater sensitivity in the intrinsic factors of arterial smooth muscle to contraction in essential hypertension may be a responsible factor in excessive systolic responses. 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, Yankowitz, and Lancaster (cited by Wolthuis et al., 1977) reported in 1975 that although systolic pressure in hypertensive patients has been

observed to rise above certain limits, such elevations were not associated with any complications or significant 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 that excessive increases in systolic pressure. Iskandrian et al. (1992) found that exercise induced hypotension cannot be explained by the extent of coronary artery disease or ischemia, that it is probably due to ischemic activation of mechanoreceptors, a mechanism similar to those of other types of neurally medicated hypotension. 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, and connective tissue in the vascular wall.

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 Thomson 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 ST segment depression and angina. Furthermore, these decreased systolic pressures could not be attributed to any pharmocologic effects. Morris, Phillips, Jordan, and McHenry (1978) reported that to is not necessary for systolic blood pressure 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 Thomson 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.

Prospects for Detecting Future 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 to 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 to 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 and blood perfusion of body tissues, normally averages 120/80 mmHg in young adults at rest. Resting blood pressure may increase during periods of anxiety, winter and

spring seasons of the year, and late evening, and as a function of age, body weight, and body fat. Resting blood pressure may be lower in women, physically fit or younger individuals, and when individuals are placed in the head down tilt position. Resting blood pressure is regulated by the reciprocal interaction between various short-term, intermediate, and long-term mechanisms, such as the tone of the sympathetic nervous system, baroreceptor reflex activity, central nervous system ischemic response, and various hormones.

It is known that blood volume and cardiac output are normal in resting hypertension, and that total peripheral resistance is greatly enhanced in this state. Nonetheless, the malfunctioning mechanisms involved are yet to be fully explained. 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" and effects of differences in muscle fiber type endowment have been suggested.

With respect to blood pressure responses with dynamic exercise, 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 excessive increases and decreases 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 260 mmHg or higher, a diastolic rise of more than 20 mmHg, or a diastolic rise above 110 mmHg. The most distressing exercise blood pressure responses involve a failure of systolic pressure to rise or a decrease in systolic with increasing in exercise intensity. These responses are indicative of left ventricular dysfunction and severe multiple vessel disease. Excessive diastolic increases have been associated with triple vessel disease.

Chapter III

JOURNAL MANUSCRIPT

INFLUENCE OF SYSTOLIC BLOOD PRESSURE ON ECG ST SEGMENT RESPONSES IN EXERCISE TESTS OF ADULTS WITHOUT DIAGNOSED CHD

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INFLUENCE OF SYSTOLIC BLOOD PRESSURE ON ECG ST SEGMENT RESPONSES IN EXERCISE TESTS OF ADULTS WITHOUT DIAGNOSED CAD

by

Lucy E. Carter

(ABSTRACT)

Records from treadmill maximal graded exercise tests (GXTs) for 61 patients from the Virginia Tech Intervention Center were screened for changes in systolic blood pressure. These blood pressure responses were standardized according to exercise demand (ΔSBP/MET) between three different levels of the exercise test. Subject records were chosen on the basis that they did not reflect a physician diagnosis of coronary artery disease (CAD) and were not taking antihypertensive medications. The ΔSBP/MET responses were stratified as follows: low to moderate (ΔBP/MET1) = difference between a systolic blood pressure at a moderate intensity stage minus the first stage systolic blood pressure, adjusted for the corresponding changes in metabolic demand (MET); moderate to high (ΔBP/MET2) = difference between systolic blood pressure at the maximal stage minus the moderate intensity stage per MET change; and low to high (ΔBP/MET3) = difference between systolic blood pressure at the maximal stage minus the first stage per MET change. Subjects were separated (STΔ and NoSTΔ) according to whether or not they had exercise induced ST segment shift of 1 mm ($\geq$ 0.1 mV) at maximal exercise. The two groups were similar in physical characteristics, except the NoSTΔ group had a significantly higher BMI (Body Mass Index), were a few years younger and exhibited a lower RPP at maximal effort. Discriminant Function Analysis was used to predict group classification of individual

patients (STΔ or NoSTΔ). Based on predictions using physical characteristics alone, (age, BMI, TC), age, BMI and TC (Total Blood Cholesterol) could correctly predicted classification in 66% of the cases. The set of age, BMI, TC and ΔBP/MET3 (low to high) generated a prediction with 77% correct classification. Thus, ΔBP/MET level alone was not the primary variable to explain predictive accuracy for clinically important ST changes in exercise testing. However, in accordance with the Bayesian principle, this hemodynamic exercise response is adjusted for overall metabolic demand in the test and coupled to markers of pre-test coronary risk, the ability to predict ST response is improved.

Introduction

A number of factors derived from epidemiologic and clinical studies have been long recognized as valuable predictors for estimating relative risks for coronary artery disease (CAD). The graded exercise test (GXT) serves as a "gatekeeper" or medical screening procedure in efforts to further evaluate patients at risk of CAD who have suspicious symptoms. More specifically, ST segment displacement in the electrocardiogram (ECG) during exericse is suggestive of myocardial ischemia and has been demonstrated to identify groups of men at high risk for CAD[1]. Unfortunately, an abnormal ST response in the exercise ECG does not precisely predict the presence or absence of disease, nor does an absence of this ECG response rule out CAD.

In healthy individuals, graded treadmill exercise evokes a moderate and progressive rise in systolic blood pressure. Deviations in systolic blood pressure responses from the normal pattern, particularly those accepted as indicative of "exertional hypotension" have been studied extensively by clinical investigators [2,3,4]. In this context, exertional hypotension may help to differentiate those likely to have more severe coronary artery disease (CAD) and left ventricular functional impairment. Other investigators [5,6,7] have concluded that certain atypical systolic blood pressure responses during and immediately after graded exercise tests may actually be more sensitive than exercise electrocardiographic changes and angina for identifying patients with coronary artery disease. Early detection of a predisposition toward development of abnormal blood pressure responses in apparently healthy individuals may lead to more effective evaluation of CAD risk and/or medical decisions to institute preventive measures.

In the practice of symptom-limited exercise testing, interpretation of blood pressure changes is of immediate concern to clinicians who must decide upon test termination. Continuation of the test toward maximal stress levels provides the opportunity to gain valuable clinical information for diagnosis and improves the determination of functinal capacity. Conversely, advancing test intensity in the presence of certain systolic blood pressure patterns increases acute risks to the patient[8]. Peak exercise data reveals a tendency for higher hemodynamic responses and more indicators of ischemia and dysrhythmias[9]. With the substantial increase in the administration of graded exercise tests with apparently healthy adults and diseased populations, further examination of the clinical significance attached to certain systolic blood pressure response patterns seems warranted.

Thus, the purpose of this investigation was to examine the exercise systolic blood pressure responses to graded treadmill exercise in a group of low to moderate risk men and women without diagnosed CAD. A second objective was to determine if exercise blood pressure responses, with or without normalization for metabolic demand (METs), might be useful in predicting the presence or absence of important ST segment shifts at peak exercise.

Methods

Subjects. Records of program clients who had no previous diagnosis of CAD, and completing at least two GXTs while in the program at the Virginia Tech Cardiac Therapy and Intervention Center from April, 1977 until August, 1995. Subjects were excluded if they were receiving anti-hypertensive medications (i.e., beta-blocker, calcium channel blocker, diuretic, or nitrate) at the time of their test.

Demographic variables. Records included results of symptom-limited treadmill exercise tests (GXTs) that had been administered using individualized protocols with increments of 1-3 METs for each 2-3 minute stage, with test endpoints based upon criteria recommended by the American College of Sports Medicine [8]. The 12-lead ECG was recorded prior to exercise and at regular interval 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 recovery period. Blood pressure was taken by auscultation using a mercury sphygmomanometer by trained technicians. The Phase IV Korotkoff sound was accepted as the indicator of diastolic pressure. Rating of perceived exertion (RPE) also was determined at the end of each stage of exercise. Signs and symptoms of exertional intolerance by the attending physician were assessed throughout the tests. Current health history, height, weight, skinfold measurements, and smoking history were assessed 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 test and decided when to terminate exercise. Subjects gave informed consent.

Measurement of BP and ECG. Resting blood pressure measurements were made prior to exercise, with the subjects in standing position. Exercise blood pressures were measured during the last minute of each stage of exercise. Recovery blood pressures were measured at 2, 4, and 6 minutes post-exercise.

The criterion for an abnormal ST response was a horizontal or downsloping shift of the ST segment to a level of 0.1 mV or more at 60 msec after the J point (J_{60}). Measurements of ST segment in the standing posture and at peak exercise were done using standard clinical procedures[10].

Measurements were made only from lead V_5 recordings, averaging two complexes in a series for which the ECG baseline was stable.

Data Analysis. Two groups were identified, according to presence or absence of clinically significant ST responses in exercise, i.e., $\geq$ 1 mm shifts in J_{60}. A series of the t-tests for independent samples were used to examine difference in demographic and certain clinical characteristics for the STΔ and NoΔST subsets. For categorical variables, group comparisons were made using chi-square analysis.

Data was analyzed by Statistical Graphics Program (StatGraphicsPlus), on a computer at the laboratory of Virginia Tech. Discriminant Function Analysis was performed to determine sets of predictors that might be used to classify observations into two or more groups based on the specified predictors. This provided the basis for predicting which membership group a new case is most likely to fall into, or to obtain a small number of useful discriminating variables.

Results

Characteristics for the two groups (STΔ and NoSTΔ) are presented in Table 1. Mean values for the variables presented were similar for the two groups. The values for BMI (Body Mass Index) and TC (Total Blood Cholesterol) were somewhat higher in the NoSTΔ group and age somewhat lower than in the STΔ group.

Considering singular predictors, age was the only significant discriminator between the two groups. Discriminant Function revealed that use of the physical characteristics of age, BMI and TC, yielded a predictive accuracy of 66% (66% of cases correctly classified). The rise in systolic blood

pressure, any adjustment for MET demand, when added to the three variable prediction model, had no effect whatsoever. However, when ΔBP/MET1 was included, predictive accuracy increased to 72%; with ΔBP/MET2 variable, accuracy decreased to 67%. Highest four variable predictive accuracy was obtained from the model: age, BMI, TC and ΔBP/MET3, i.e., adding the systolic blood pressure response across the entire exercise test, adjusted for overall MET demand raised the correct classification rate to 77%. This finding was associated with a sensitivity (true positives) of 81% and a specificity (true negatives) of 76%. Using Discriminant Function, calculations were made as to whether subjects were members of the STΔ or NoSTΔ groups using zero as the cutoff point (see Figure 1). The STΔ subjects were predicted on the positive side of zero and the NoSTΔ subjects the negative side of zero. A Receiver Operating Characteristic (ROC) Analysis demonstrated that the four variable model predicted group classification better than chance alone; the area under the prediction performance curve was 0.76 ($p < 0.01$).

Discussion

The purpose of this study was to determine whether the progression of systolic blood pressure might contribute to prediction of presence or absence of clinically important ST segment shifts in the ECG responses of adults undergoing graded exercise testing. The inclusion of other pre-exercise characteristics, with the addition of systolic blood pressure rise across the entire graded exercise test, did provide a means to predict those who will have ST segment shifts at peak exercise in adults without diagnosed coronary artery disease.

The groups in this study were similar in physical characteristics, with the STΔ group being older. The groups also were similar in cardiovascular risk factors. The TC levels were close, and the only difference was that the NoSTΔ group had a higher BMI level. The groups were similar in peak exercise responses and indicators of myocardial demand. Both the STΔ and the NoSTΔ groups could be best predicted after the highest level of the test. Additional group differences may have been concealed as a result of the small number of subjects in the group.

ST depression occurring at above average maximal heart rates, peak systolic pressures and average normal durations of maximal exercise and clearing rapidly immediately after such exertion is usually a "false positive" response. This phenomenon of physiological imbalance between demand and supply represents an excessive pressure rate demand on the left ventricle in the presence of a virtually normal coronary blood supply[11].

The results indicated a relationship between descriptive factors and lipid values (i.e. age, TC, BMI) and predicting a ST shift without the exercise test. However, some investigators have found that exercise blood pressure along with other exercise values are more predictive of future hypertension than just resting values alone [14,15].

Other research reported that maximal values for systolic blood pressure tended to be somewhat higher with increasing age[11], which was also evidenced in the noninvasive research which involved three progressive age decades (25-34, 35-44, and 45-54 yr.)[12]. Maximal systolic pressure increased slightly across the three age groups (164-212, 164-214, and 162-216 mmHg,

respectively), as did maximum diastolic pressure (60-90, 64-94, and 68-96 mmHg, respectively).

Systolic pressure is normally expected to taper off as maximal effort is approached. A thorough pre-test screening incorporates 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, and blood lipid values are assessed along with diet, stress levels, and stress tolerances. 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 CAD. The information obtained in the present study, also may lead to improved identification of cardiovascular problems.

When trying to predict the probability of an ST shift, all variables of the exercise test should be considered. Although this present study viewed blood pressure responses throughout the entire exercise test, responses from submaximal data also may prove to be beneficial for future research. In research of other investigators, it was concluded that an early rise of systolic blood pressure during exercise adds prognostic information on cardiovascular mortality among otherwise healthy middle aged men with mildly elevated casual blood pressure[14]. However, an additional issue that merits further study would be recovery blood pressures in efforts to predict abnormal ST responses. The normal decline in systolic blood pressure during the recovery phase of treadmill exercise was examined and discovered that blood pressures

in patients with more severe coronary heart disease do not decline normally and that some recovery systolic blood pressure values actually exceed the peak exercise values[6]. They concluded that the ratios of early recovery systolic blood pressure to the peak exercise systolic blood pressure are more sensitive than exercise electrocardiographic changes and angina for identifying patients with CAD.

In trying to apply the findings of the present study in the clinical setting, first it should be acknowledged that screening the patient for CAD risk status is the most important. Practitioners should begin with screening with baseline characteristics such as age, total cholesterol, body composition. Once the GXT is progressing, it then becomes more relevant to examine the ΔSBP/MET progression, along with monitoring for signs and symptom appearance. Both the STΔ and the NoSTΔ groups could be best predicted after the highest level of the test.

Thus, in the present study we concluded that when using ΔSBP/MET level, used along with physical characteristics could give a 77% correct response to having a cardiovascular event. Using exercise blood pressure responses in combination with CAD risk factors to predict the future development of resting hypertension and CAD may prove invaluable in improving the clinical management of apparently healthy individuals by possibly preventing the occurrence of CAD. In accordance with the Bayesian principle, this hemodynamic exercise response is adjusted for overall metabolic demand in the test and coupled to markers of pre-test coronary risk, the ability to predict ST response is improved.

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Table 1. Group Classification[a] (NoΔST and ΔST) of Overall Sample

	NoΔST	ΔST	T-ratio	p level
Pre-Exercise Test Characteristics				
%MALE	33/45=73	11/16=69		
%FEMALE	12/45=27	5/16=31		
AGE, yrs	46.7 ± 1.5	53.6± 2.0	2.80	0.01
BMI, kg/m^2	26.7 ± 0.7	24.7 ± 0.8	-1.92	0.12
CHOL, mg/dL	221.2 ± 7.2	218.7 ± 8.1	-0.22	0.85
SBPst, mmHg	123.4 ± 5.1	121.6 ± 6.2	-0.38	0.45
HRst, b/min	76.1 ± 2.8	74.3 ± 3.9	-0.26	0.37
Exercise Responses				
SBPpk, mmHg	184.2 ± 3.9	181.1 ± 6.3	-0.41	0.68
HRpk, b/min	167.5 ± 2.9	165.3 ± 3.3	-0.49	0.63
RPPpk, $(X10^{-2})$	295.1 ± 14.0	316.0 ± 9.2	-1.27	0.24
ΔSTpk(J60), mV	0.0 ± 0.1	-0.1 ± 0.3	-3.55	0.01
RPEpk[b]	16.8 ± 0.3	17.0 ± 0.3	0.44	0.66
BP/MET3[c]	6.7 ± 0.6	5.7 ± 0.5	1.43	0.21

[a] Values are Means,and Standard Errors
[b] RPE was obtained from the 6 - 20 Borg scale
[c] BP/MET3 = Change in systolic blood pressure from low to high response in exercise test, adjusted for corresponding increase in MET demand.

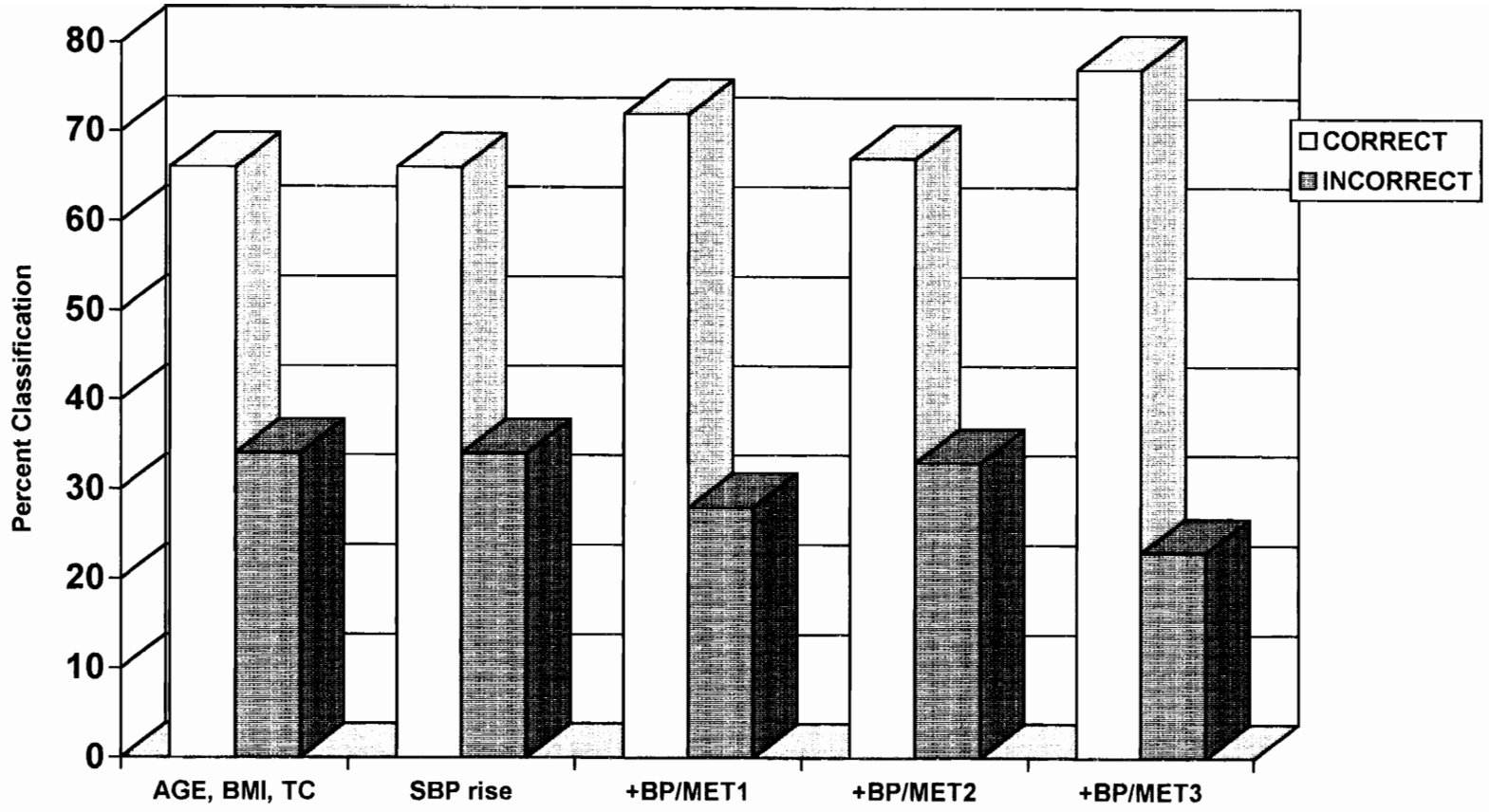

Figure 1. Exercise BP as Predictors of ST Shift

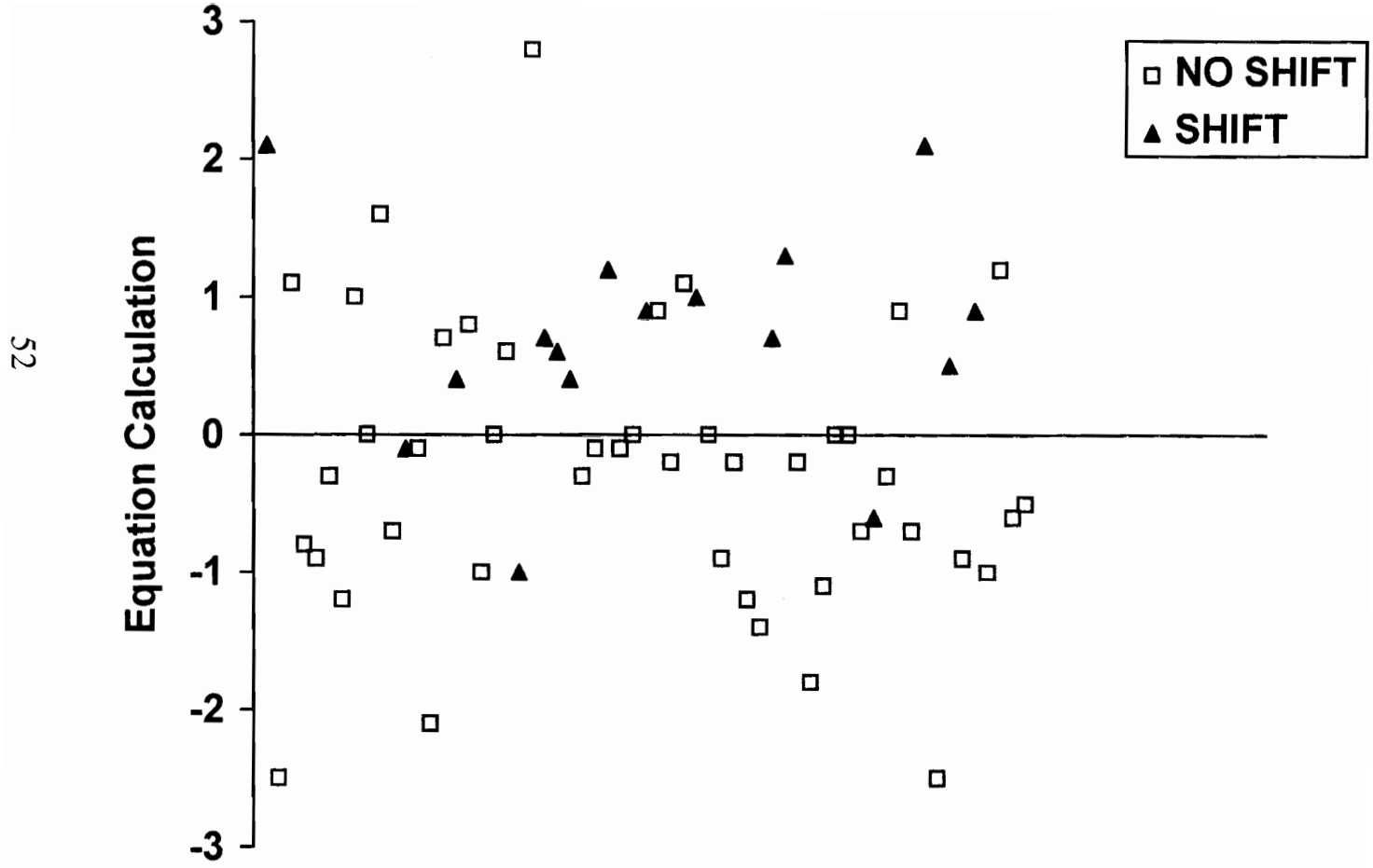

Figure 2. Prediction Success of ST Segment Shifts

Table 2. Discriminant Function Analysis for Group Classification

Equation for Group (StatGraphics Plus)

Constant	- 1.363
BP/MET3	0.159
AGE	0.082
BMI	- 0.094
CHOL	- 0.049

Total = 61 Correctly Classified = 47

Percentage of Cases Correctly Classified (%) = 77

ROC Analysis

Area Under Curve = 0.76

p level < 0.01

Chapter IV

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

Summary and Conclusions

Records from treadmill maximal graded exercise tests (GXTs) for 61 patients from the Virginia Tech Intervention Center were screened for changes in systolic blood pressure. These blood pressure responses were standardized according to exercise demand (ΔSBP/MET) between three different levels of the exercise test. Subject records were chosen on the basis that they did not reflect a physician diagnosis of coronary artery disease (CAD) and were not taking antihypertensive medications. The ΔSBP/MET responses were stratified as follows: low to moderate (ΔBP/MET1) = difference between a systolic blood pressure at a moderate intensity stage minus the first stage systolic blood pressure, adjusted for the corresponding changes in metabolic demand (MET); moderate to high (ΔBP/MET2) = difference between systolic blood pressure at the maximal stage minus the moderate intensity stage per MET change; and low to high (ΔBP/MET3) = difference between systolic blood pressure at the maximal stage minus the first stage per MET change. Subjects were separated (STΔ and NoSTΔ) according to whether or not they had exercise induced ST segment shift of 1 mm ($\geq$ 0.1 mV) at maximal exercise. The two groups were similar in physical characteristics, except the NoSTΔ group had a significantly higher BMI (Body Mass Index), were a few years younger and exhibited a lower RPP at maximal effort. Discriminant Function Analysis was used to predict group classification of individual

patients (STΔ or NoSTΔ). Based on predictions using physical characteristics alone, (age, BMI, TC), age, BMI and TC (Total Blood Cholesterol) could correctly predicted classification in 66% of the cases. The set of age, BMI, TC and ΔBP/MET3 (low to high) generated a prediction with 77% correct classification. Thus, ΔBP/MET level alone was not the primary variable to explain predictive accuracy for clinically important ST changes in exercise testing. However, in accordance with the Bayesian principle, this hemodynamic exercise response is adjusted for overall metabolic demand in the test and coupled to markers of pre-test coronary risk, the ability to predict ST response is improved.

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 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 diagnosis and preventive treatment.

This study was conducted to determine the incidence of atypical exercise blood pressure responses in a group of adults without diagnosed CHD. The study was further designed to determine a classification of systolic blood pressure responses in exercise according to a certain MET demand.

Descriptive group statistics were generated using Minitab for Windows for all the variables. The variables were then separated out and discriminant function analysis was performed using Minitab for Windows. The purpose of these preliminary analyses were to identify which variables would be entered

into the final discriminant function analysis. The purpose of the last analysis was to determine which variables discriminate between the group that is at risk of having a cardiovascular event and those who are not at risk.

Discriminant function analysis between the at risk group (STΔ) and the one not at risk (NoSTΔ) was performed on the GXT data. The following GXT data was used: event, age, BMI, TC, and BP/MET3. Age was a significant predictor between the two groups. Seventy six percent of the control (NoSTΔ) group and eighty one percent of the at risk group (STΔ) were correctly classified in their respective groups. Results of the study revealed that using four different predictors could give a 77% correct response to having a cardiovascular event. The different predictors were: age, BMI, cholesterol, and BP/MET3. It was concluded that those four predictors gave the best percentage in trying to determine if an individual was at risk of having an ST shift while exercising. Also concluded was that BP/MET measurements could not be used alone as predictors in the present study.

Recommendations for Future Research

One aspect for researchers to study would be recovery blood pressures. Amon et al. (1984) looked at the normal decline in systolic blood pressure during the recovery phase of treadmill exercise and discovered that blood pressures in patients with coronary heart disease do not decline normally and that some recovery systolic blood pressure values actually exceed the peak exercise values. They concluded that the ratios of early recovery systolic blood pressure to the peak exercise systolic blood pressure are more sensitive than exercise electrocardiographic changes and angina for identifying patients with CAD.

Although this present study viewed blood pressure resonses throughout the entire exercise test, submaximal data may prove also to be beneficial for future research. Wolthuis et al. (1977) found that the blood pressure begins to plateau near maximal levels of the graded exercise test as maximal effort is attained in apparently healthy individuals.

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

Recommendations for Practitioners in Adult Fitness

Practitioners at the Laboratory for Health and Exercise Sciences at Virginia Tech and at other clinical settings should keep placing extreme importance upon precise measurement taking and thorough record keeping. A thorough pre-test screening incorporates 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, and blood lipid values are assessed along with diet, stress levels, and stress tolerances. 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.

Practitioners should begin with screening with baseline characteristics such as age, total cholesterol, body composition. After that is attained, they should start watching ΔSBP/MET closely during the exercise test. Once the data has been collected, practitioners should begin sorting on basis of results to help make decisions on need for physician supervised GXT. They should lend support from standards setforth by ACSM with respect to medical supervision. The ACSM recommendation that exercise tests of asymptomatic adults without CHD risk factors be administered by qualified exercise test personnel. The recommendation for those adults with CHD is that a physician is required. Apparently healthy individuals into supervised exericse programs may require minimal supervision during exercise testing and training, even in the presence of CHD risk factors or atypical blood pressure responses.

Thus, the present study concluded that when using ΔSBP/MET level, used along with physical characteristics could give a 77% correct response to having a cardiovascular event. Using 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. Therefore, when blood pressure changes to exercise are interpreted in conjunction with preliminary clinical findings plus accepted standards for test termination judgment of test safety may be improved.

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Appendix A

Appendix A

Detailed Methodology

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 participants, classified as noncardiac and achieving a minimum of four completed exercise stages, were selected. Of the 105 male and female subjects initally reviewed, 31 were on hypertensive medication. An additional two were eliminated due to missing values for SBP. All 61 subjects were tested at the Virginia Tech Intervention Center by their laboratory staff and a center affiliated physician. Refer to Table 1 for the characteristic profile of the final subjects (N=61).

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, wach subject signed an informed consent form prior to testing. Each candidate who met the experiment-set requirements for the study was included in the sample.

General Methods

Measurements and Techniques

Subject Classification - Based upon the physician referral, andy subject with no diagnosed coronary artery disease was classified as noncardiac. Any subject diagnosed with coronary artery disease, based upon the

physician referrral, was classified as cardiac and was dropped from the study.

Categories of Disease - Each subject's file records were reviewed to determine which of the following conditions existed as documented in the physician 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 ausultation using a mercury manometer, standard blood pressure cuff, and stethoscope.

Exercise Blood Pressure - Exercise blood pressure was measured by auscultation during the last minute of each stage of exercise and at the maximal level during the GXT.

ECG Measurements - J_0 and J_{60} were measured at rest and during exercise. All measurements were made in lead V^5, recorded in millimeters and later converted to millivolts. All ECG values were estimated within one-half millimeter.

Rating of Perceived Exertion - Subjective RPE was assessed using Borg's 15- (range = 6 to 20) point scale. 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 exercise stage.

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, dypsnea or shortness of breath, leg fatigue, general fatigue, ot any other factors resulting in test termination.

Serum Cholesterol - Blood sample were drawn from each subject following a twelve-hour fast. Sample were drawn wither 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.

Height - On conjunction with the GXT, an anthropometer was used to measure each subject's height while wearing atletic or 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 wearingshoes. 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 is so, they were asked to disclose the quantity, frequency, and type of smoking. Subjects who had not smoked in more than two years wer considered non-smokers and those who were still currently smoking were considered smokers.

BMI (Body Mass Index) - Each subject's BMI was calculated using their weight in kilograms divided by their height in meters squared.

Statistical Procedures

The overall subject sample was described using means and standard errors of the mean. Two groups were identified, according to presence or absence of clinically significant ST responses in exercise; ≥ 1 mm shifts in J60. A series of the t-tests for independent samples were used to examine difference in demographic and certain clinical characteristics for the STΔ and NoΔST subsets. Group comparisons also were made using chi-square analysis for categorical variables such as gender, and signs and symptoms during exercise.

Discriminant analysis was mainly used to classify observations into two or more groups based on the specified predictors. This provided the basis for predicting which membership group a new case is most likely to fall into, or to obtain a small number of useful discriminating variables.

Receiver Operating Characteristic (ROC) Analysis, from the Epistat program on the Virginia Tech laboratory computer, demonstrated that the four variable model predicted group classification better than chance.

Appendix B

Definitions and Symbols for Computer Data Coding

Subject = Each subject was given and identification number

AGE = Age of subject in years

GENDER = Male (0) or Female (1)

BMI = Body Mass Index

CHOL = Total Blood Cholesterol

Hx HTN= History of hypertension (0 = no Hx; 1 = yes Hx)

BPpre = Resting hypertension before test (0 = no; 1 = yes)

SMOKE = Smoker (1) or Non-smoker (0)

SYMP = Signs or symptoms during exercise test (0 = No; 1 = Yes)

SBPst = Systolic blood pressure standing before exericse

DBPst = Diastolic blood pressure standing before exercise

HRst = Heart rate standing before exercise

J60rst = Measurement of ST segment at 60 msec after the J point at rest

J60pk = Measurement of ST segment at 60 msec after the J point at peak

J60ch = Change in J60 between rest and peak

STch = Whether subject had ST depression of 1 mm or more, 0 for no shift and 1 for a ST shift

RPEpk = Rate of Perceived Exertion at peak (scale of 6 - 20)

SBPpk = Systolic blood pressure peak (in mmHg)

HRpk = Heart rate peak (in b/min)

RPPpk = Rate Pressure Product at peak (10^{-2})

SBPch1 = Systolic blood pressure change between moderate and low levels of the exericse test.

METch1 = Met level change between the moderate and low levels of the exercise test

BP/MET1 = Low to moderate response of systolic blood pressure per MET change

SBPch2 = Systolic blood pressure change between moderate and high levels of the exercise test

METch2 = Met level change between the moderate and high levels of the exercise test

BP/MET2 = Moderate to high response of systolic blood pressure per MET change

SBPch3 = Systolic blood pressure change between moderate and high levels of the exercise test

METch3 = Met level change between the high and low levels of the exercise test

BP/MET3` = Low to high response of systolib blood pressure per MET change

Equation = Calculation for prediction of group membership (NoSTΔ and STΔ)

SUBJECT	AGE	GENDER	BMI	CHOL	Hx HTN	BPpre	SMOKE
1	72	0	22.6	159	0	0	0
2	23	0	30.4	208	0	0	0
3	53	1	23.1	240	0	0	0
4	31	1	23.3	146	0	0	1
6	44	0	23.6	247	0	0	0
7	46	0	25.4	219	0	0	0
8	41	0	40.4	152	0	0	0
9	57	0	24.1	194	0	0	0
10	62	0	27.8	246	0	1	0
11	64	0	21.3	181	0	0	0
12	46	0	25.3	204	0	0	0
13	49	0	22.8	236	0	0	0
14	56	0	28.3	236	0	0	0
15	34	1	31.6	234	0	0	0
16	56	0	23.2	240	0	0	0
17	44	1	25.1	225	0	0	0
18	59	1	27	293	0	0	0
19	40	0	29.1	195	1	0	0
20	52	0	22.7	192	0	0	0
21	50	0	24.3	176	0	0	1
22	44	0	32.5	237	0	0	0
23	57	1	21.6	250	0	0	0
24	53	1	24	243	0	0	0
25	52	0	26.8	212	0	0	0
26	52	0	26.6	179	0	0	0
27	45	0	25.1	208	0	0	0
28	47	0	29.6	185	0	0	0
29	63	0	22.9	252	0	0	0
30	43	0	25.9	173	0	0	1
31	49	0	21.1	271	0	0	0
32	62	0	26	247	0	1	0
33	49	0	26.2	269	0	0	0
34	46	0	28.4	275	0	0	0
35	66	1	26.8	258	0	1	0
36	58	0	24.3	225	0	0	0
37	47	0	22.5	217	0	0	0
38	38	0	27.6	165	0	0	0
39	47	0	30	165	0	0	0
40	22	0	19.2	180	0	0	0
41	33	0	25.7	166	0	0	0
42	51	1	29.8	203	0	0	0
43	59	0	24.4	240	0	0	1
44	45	0	24.4	190	0	0	0
45	38	0	34.1	222	0	0	0
46	58	1	42.9	201	0	0	0
47	55	1	30	192	0	0	0

48	51	1	22.2	307	0	0	0
49	49	0	24.9	267	0	1	1
50	43	0	23.2	276	0	1	0
51	42	0	33.3	245	0	1	0
52	61	1	23.4	220	0	0	0
53	48	1	28.8	261	0	1	0
54	55	1	19.8	185	0	0	0
55	36	0	28.3	416	0	1	1
56	45	1	22.5	173	0	0	0
57	39	0	25.6	230	0	0	1
58	56	0	21.8	208	0	0	0
59	41	0	30.6	194	0	1	0
60	53	1	20.5	237	0	0	0
61	42	0	27.8	179	0	0	0
62	39	0	25.1	207	0	0	0

SYMP	SBPst	DBPst	HRst	J60rst	J60pk	J60ch	STch
0	122	68	48	0	-1.5	-1.5	1
0	116	78	70	0.5	0.5	0	0
0	82	70	85	0	0	0	0
0	108	70	92	0	0	0	0
0	108	72	86	0	0.5	0.5	0
0	120	80	54	1	0.5	-0.5	0
0	122	80	69	0	-0.5	-0.5	0
0	112	72	76	0.5	0	-0.5	0
0	144	94	80	0	-0.5	-0.5	0
0	110	82	68	0	0.5	0.5	0
0	132	90	75	0	0.5	0.5	0
0	120	88	68	1	-2	-3	1
0	124	84	71	0	0	0	0
0	132	82	98	0	-0.5	-0.5	0
0	124	74	54	0	-0.5	-0.5	0
0	120	80	68	0.5	-1	-1.5	1
0	132	76	62	0	0	0	0
0	138	90	62	0	-0.5	-0.5	0
1	108	78	80	0.5	0.5	0	0
0	122	78	94	0	0	0	0
0	108	90	80	0	-1.5	-1.5	1
0	122	84	78	-0.5	-0.5	0	0
0	120	80	81	0	-1	-1	1
1	116	70	41	0	-1.5	-1.5	1
0	110	74	100	0	1	1	1
0	110	80	84	-0.5	0	0.5	0
0	112	74	66	0	0.5	0.5	0
1	126	88	78	0.5	-1.5	-2	1
1	108	78	68	0	0	0	0
0	132	88	68	0	0	0	0
0	178	82	108	0	-1	-1	1
0	116	90	91	0	0	0	0
0	128	82	68	0	0	0	0
0	142	92	78	0	0.5	0.5	0
1	118	66	78	0	-1	-1	1
0	122	80	59	0	0	0	0
0	120	86	70	0	0	0	0
0	108	90	79	0	0	0	0
0	124	70	75	0	0	0	0
0	114	74	97	0.5	0	-0.5	0
1	114	80	100	0	1	1	1
0	134	88	60	0.5	-2.5	-3	1
0	118	78	86	0	0	0	0
0	124	70	66	0	0.5	0.5	0
0	124	84	83	0	-0.5	-0.5	0
0	136	84	100	-0.5	-0.5	0	0

0	118	80	75	0	-0.5	-0.5	0
0	150	100	86	0	-0.5	-0.5	0
0	164	102	75	0	1	1	1
0	148	96	71	0	0	0	0
0	120	82	68	0	0	0	0
0	140	92	88	0	0.5	0.5	0
1	112	72	64	0	-2	-2	1
0	140	84	103	0	0	0	0
0	122	80	62	0	-1.5	-1.5	1
0	130	88	60	0	0	0	0
0	94	80	81	0	-1	-1	1
0	140	90	85	0	-0.5	-0.5	0
1	120	96	83	0	0	0	0
1	116	90	75	0	-0.5	-0.5	0
0	124	80	60	0	0	0	0

RPEpk	SBPpk	HRpk	RPPpk	SBPch1	METch1	BP/MET1	SBPch2
17	130	135	175.5	12	3.6	3.3	10
17	192	188	361	32	5.6	5.7	22
18	138	170	234.6	28	3.1	9.0	22
19	166	167	277.2	25	6.2	4.0	32
15	154	165	304	6	5.6	1.1	16
19	186	187	347.8	44	6.9	6.4	14
19	210	187	392.7	24	3.1	7.7	48
13	176	152	305	28	2.4	11.7	22
13	224	142	318	-4	2.4	-1.7	10
13	162	125	203	20	6.2	3.2	22
19	188	188	353	2	4.1	0.5	12
16	150	166	249	18	3.9	4.6	10
16	198	174	334	30	5.6	5.4	16
13	178	176	306	16	6.9	2.3	24
17	182	160	302	18	3.9	4.6	36
18	168	158	265	24	2.1	11.4	38
18	186	117	210	10	2.1	4.8	38
16	206	177	364.3	20	6.2	3.2	36
17	128	169	216	8	5.6	1.4	6
19	192	190	353.4	26	4.1	6.3	34
17	188	176	331	26	5.6	4.6	32
19	194	188	315.8	34	2.1	16.2	38
19	196	170	333.2	24	3.6	6.7	34
16	192	165	316.8	46	5.6	8.2	26
19	182	180	327	30	6.1	4.9	40
17	176	166	292.2	34	6.2	5.5	12
17	182	167	303.9	50	5.2	9.6	14
15	192	168	275	12	3.2	3.8	28
13	162	180	291	16	3.1	5.2	16
17	178	150	295.5	17	4.1	4.1	16
15	218	168	366	28	4	7.0	18
17	192	178	306	18	2.9	6.2	50
13	212	150	310	34	6	5.7	34
16	198	175	329	14	2.4	5.8	16
17	172	138	237	8	4.1	2.0	44
11	170	127	215	20	4.1	4.9	26
17	194	171	331.7	36	6	6.0	14
17	178	162	288	38	5.6	6.8	20
17	214	170	364	60	6.2	9.7	34
18	172	187	321.6	10	6.2	1.6	30
16	164	177	290.3	20	1	20.0	12
17	188	170	319.6	16	2.9	5.5	34
18	162	180	392	18	5.2	3.5	22
19	156	125	299.5	6	2.1	2.9	14
18	160	157	239	14	4.1	3.4	8
15	162	176	285	18	3.9	4.6	4

18	148	166	282.2	18	3.1	5.8	8
19	204	156	315	4	4.1	1.0	16
16	236	170	401	34	6.2	5.5	24
17	280	188	526.4	42	4.9	8.6	60
19	166	150	249	24	4.1	5.9	8
19	188	177	332.8	22	4.1	5.4	14
18	190	156	296	40	3.1	12.9	28
19	224	194	434.6	30	7.9	3.8	30
17	162	166	252	18	2.1	8.6	20
17	190	180	392.4	30	6.2	4.8	18
19	170	182	288	20	5.6	3.6	36
17	224	170	374	40	7.9	5.1	34
17	180	136	244.8	16	4.1	3.9	34
17	174	188	327.1	36	4.1	8.8	20
17	182	188	382	18	4.1	4.4	34

METch2	BP/MET2	SBPch3	METch3	BP/MET3	Equation
4.2	2.4	22	7.8	2.8	2.1
4.7	4.7	54	10.3	5.2	-2.5
2.1	10.5	5	5.2	1.0	1.1
3.5	9.1	56	9.7	5.8	-0.8
4.7	3.4	22	10.3	2.1	-0.9
5.8	2.4	58	12.7	4.6	-0.3
5.6	8.6	72	8.7	8.3	-1.2
6.5	3.4	50	8.9	5.6	1
3.2	3.1	6	5.6	1.1	0
4.8	4.6	42	11.1	3.8	1.6
3.1	3.9	14	7.2	1.9	-0.7
4.7	2.1	28	8.6	3.3	-0.1
4.5	3.6	46	10.1	4.6	0.1
3.1	7.7	40	10	4.0	-2.1
5.9	6.1	54	9.8	5.5	0.7
4.1	9.3	62	6.2	10.0	0.4
3.5	10.9	48	5.6	8.6	0.8
5.4	6.7	56	11.6	4.8	-1
4.6	1.3	14	10.2	1.4	0
5.6	6.1	60	9.7	6.2	0.6
3.3	9.7	58	8.9	6.5	-1
2	19.0	72	4.1	17.6	2.8
4.3	7.9	58	7.9	7.3	0.7
3.3	7.9	72	8.9	8.1	0.6
6	6.7	70	12.1	5.8	0.4
3.4	3.5	46	9.6	4.8	-0.3
4.1	3.4	64	9.3	6.9	-0.1
4.7	6.0	40	7.9	5.1	1.2
2	8.0	32	5.1	6.3	-0.1
2.1	7.6	33	6.2	5.3	0.2
4.3	4.2	46	8.3	5.5	0.9
2.4	20.8	68	5.3	12.8	0.9
3.7	9.2	68	9.8	6.9	-0.2
3.2	5.0	30	5.6	5.4	1.1
3.8	11.6	52	7.9	6.6	1
5.2	5.0	46	9.3	4.9	0.1
4.9	2.9	50	10.9	4.6	-1
3.3	6.1	58	8.9	6.5	-0.1
7.5	4.5	94	13.7	6.9	-1.2
5.8	5.2	40	12	3.3	-1.4
2.1	5.7	32	3.1	10.3	0.7
3.1	11.0	50	6	8.3	1.3
4.1	5.4	40	9.3	4.3	-0.2
2	7.0	20	4.1	4.9	-1.8
2.1	3.8	22	6.2	3.5	-1.1
1.2	3.3	22	5.1	4.3	0

2.1	3.8	26	5.6	4.6	0
5.6	2.9	20	9.7	2.1	-0.7
4.9	4.9	58	11.1	5.2	-0.6
3.2	18.8	102	8.1	12.6	-0.3
5.6	1.4	32	9.7	3.3	0.9
3.8	3.7	36	7.9	4.6	-0.7
3.1	9.0	68	6.2	11.0	2.1
6.2	4.8	60	14.1	4.3	-2.5
3.1	6.5	38	5.2	7.3	0.5
3.5	5.1	48	9.7	4.9	-0.9
6.8	5.3	56	12.4	4.5	0.9
5.8	5.9	74	13.7	5.4	-1
2.1	16.2	50	6.2	8.1	1.2
6.5	3.1	56	10.6	5.3	-0.6
3.8	8.9	52	7.9	6.6	-0.5

Appendix C

SUBJECT _

DATE OF TEST: _____ HT_____ WT: _____ BMI_____
AGE: _____ TC_____

BPst: _____ mmHg BPsu_____mmHG
HRst: _____ bpm HRsu:_____bpm
HxHTN_____ RestHTN_____ Smoking_____

Risk Factors:_____
Medications:_____
_____

S	METS	SP (mph)	GR (%)	MIN	ST	HR	BP
1							
2							
3							
4							
5							

Total Time: _____min Protocol:_____ Symptoms_____

STrest_____STseg: _____Jpt:_____ J60:_____

RPEpk_____RPP: _____PredVO2: __ ml/kg/min

Calculations:

Stage 1: _____METS/ _____ = ___ Low-Mod. Response

Stage 2: _____METS/ _____ = ___ Mod.- Hi Response

Stage 3: _____ METS/ _____ = ___Low-Hi Response

VITA

Lucy Ellen Carter was born on May 1, 1969 in Boulder, Colorado to Nancy and Harold Carter. After graduating from Boulder High School in May, 1987, Lucy went on to Colorado State University in Fort Collins, Colorado. She decided to study exercise and sports science with a concentration in Wellness Program Management as her major. Lucy graduated in December of 1991 after finishing an internship in Durham, North Carolina for Duke University in their corporate fitness program.

After the internship, Lucy worked odd jobs at restaurants and she received a job at a local health club. Lucy realized she wanted to further her education in cardiac rehabilitation and applied to Virginia Tech. Lucy was accepted in April, 1993 and is currently completing her degree requirements. She hopes to attain the Exercise Specialist Certification from the American College of Sports Medicine in the near future.

In her free time, Lucy enjoys all sports, especially basketball and football and loves all sorts of music, and reading. Hopefully, she will have a paying job when all this is done, which will make her parents particularly happy.