To the Graduate Council:

I am submitting herewith a dissertation written by Donald J. Torok entitled "The Blood Pressure Response in Sprinters and Distance Runners to Isometric and Dynamic Exercise." I have examined the final copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Education.

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THE BLOOD PRESSURE RESPONSE IN SPRINTERS AND DISTANCE RUNNERS TO ISOMETRIC AND DYNAMIC EXERCISE

A Dissertation

Presented for the

Doctor of Philosophy

Degree

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Donald J. Torok

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DEDICATION

This dissertation is dedicated to my parents

Mr. George E. Torok

and

Mrs. Clara G. Torok

who have always supported and stood by me in all of my endeavors.

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Over the course of my lifetime, there have been many individuals and situations that have influenced and guided not only my own academic growth, but also have nurtured and instilled the love that develops between student and mentor. One can never remember everyone or everything that has had an impact on their life, for every experience will somehow or somewhere influence our view of the world. As I take this next step along the path of my life, I would like to thank a few individuals for the part that they have played in my life.

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ABSTRACT

The purpose of this study was to examine the cardiovascular responses of sprinters and distance runners to isometric (IE) and dynamic (DE) exercise in an attempt to understand the role that training and muscle fiber type play in the hemodynamic adjustments to exercise. Eighteen normotensive college-age males were selected as subjects based upon prior running performance and grouped accordingly: sprinter (n=6), distance runner (n=6), or active control (n=6). Each subject completed a DE (cycle ergometry) test (6 minutes) at 20%, 40%, and 60% of $\dot{V}O_{2peak}$, three minutes of isometric handgrip at 30% of MVC, and a muscle biopsy. Blood pressure (BP), heart rate (HR), cardiac index (CI), oxygen uptake, and blood lactate were measured and systemic vascular resistance (SVR) was calculated during each stage of DE, while BP and HR were measured during each minute of IE. Needle biopsies of the vastus lateralis muscle revealed a significant difference in capillary density (capillaries per mm^2) between the sprinters and distance runners (323 ±23 vs. 409 ±27, p=0.0308) and approached significance for type I fibers (46.5 ±4.4% vs. 64.8 ±6.7%, p=0.06). The IE challenge elicited higher BP responses (SBP, DBP, & MABP) at minute-3 in the sprinters, which was due to their higher HR response. During DE, there were no significant BP or HR differences between the groups. However, at 60% of $\dot{V}O_{2peak}$, the distance runners had a higher cardiac index (p=0.0002) and a lower systemic vascular resistance (p=0.0157) than the sprinters. The latter response is consistent with the greater capillary density in the distance runners. Taken together, these results indicate that fiber type, capillary density, and/or training influence the hemodynamic responses to exercise, even when intensity is normalized to a subject's maximal capacity.

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CHAPTER I

INTRODUCTION

In the United States, hypertension is one of the foremost long-term health problems facing its citizenry. The American Heart Association estimates that over 50 million Americans are afflicted with this condition [1]. Since hypertension is a primary risk factor for cardiovascular disease, early identification of individuals predisposed to this condition would be of great benefit. While resting blood pressure (BP) can significantly predict future hypertension [2-5], there is evidence that exaggerated blood pressure responses to exercise improve upon those predictions [2, 6-11].

An exaggerated BP response to exercise, in normotensive individuals, has been found to be a significant predictor of future hypertension. Prospective studies have shown that exaggerated BP responses to submaximal [6-9] and maximal [2, 10, 11] exercise are associated with a 2-4 fold increase in the risk of future hypertension [9]. The significance of a "hyper-reactive" BP response is further supported by the fact that several groups at increased risk of developing hypertension exhibit this response. Blacks [12-16], borderline hypertensives [11, 17-19], and normotensive offspring of hypertensive parents [20, 21] show greater increases in blood pressure during exercise than age- and sex-matched controls.

Despite the potential importance of the exaggerated BP response, the factors responsible for this phenomenon are still largely undetermined. However, there is evidence that skeletal muscle characteristics may influence BP control during exercise. Specifically, skeletal muscle fiber type (and associated differences in vascularity) may influence the level of peripheral resistance in skeletal muscle. Type I (slow-twitch) fibers

have greater oxidative capacity, fatigue resistance, and vascularity than type II (fast-twitch) fibers. Juhlin-Dannfelt et al. [22] found a positive correlation between the % type II fibers and leg vascular resistance at rest. In addition, Frisk-Holmberg et al. [23], showed that % type II fibers were negatively correlated to leg blood flow during dynamic exercise. Subsequently, Fitton [24] reported that sprinters (presumed to have a greater percentage of type II fibers) had a 2-fold greater increase in the blood pressure response to both isometric and dynamic exercise, compared to distance runners. These studies suggest that there is a relationship between fiber type and the blood pressure response to both dynamic and isometric exercise.

The primary purpose of this study was to extend this line of research by examining the cardiovascular responses of sprinters and distance runners to isometric and dynamic exercise. These groups differ in type of training and potentially, in a genetic predisposition, that should influence fiber type and capillary density. Muscle biopsies were used to assess the muscle fiber type and capillary density of each group. The changes in mean arterial pressure (MABP), heart rate (HR), and (for dynamic exercise) cardiac index (CI), and systemic vascular resistance (SVR) responses were utilized to examine the role that muscle fiber type and capillary density may play in the hemodynamic adjustments to exercise.

Hypotheses

The following hypotheses were tested:

ISOMETRIC EXERCISE

Hypothesis 1: The distance runners will have a lower resting HR than the sprinters and control group.

Hypothesis 2: The HR response to each minute of isometric exercise will be greater in the sprinters than in the distance runners and the controls.

Hypothesis 3: The systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial blood pressure (MABP) will not be different in these three groups at rest.

Hypothesis 4: The systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial blood pressure (MABP) will be greater in the sprinters during each minute of isometric exercise than in the distance runners and the control group.

DYNAMIC EXERCISE

Hypothesis 5: The distance runners will have a lower resting HR than the sprinters and controls.

Hypothesis 6: There will be no difference in the HR response at 20, 40, and 60% of $\dot{V}O_{2peak}$ in the three groups.

Hypothesis 7: The SBP, DBP, MABP, cardiac index (CI), systemic vascular resistance (SVR) will be greater in the sprinters at 20, 40, and 60% of $\dot{V}O_{2peak}$ than in the distance runners and the control group.

CHAPTER II

REVIEW OF LITERATURE

The purpose of this review is to examine the cardiovascular and hemodynamic responses to different stressors as they relate to blood pressure regulation. Special emphasis is directed to research examining the blood pressure response during dynamic and isometric exercise and the possible role of muscle fiber type on blood pressure regulation.

Hypertension

Hypertension is one of the foremost long-term health problems facing the American public. With a prevalence rate affecting around 30% of the total population, and the incidence being twice as great in blacks than whites, hypertension research has a wide-based interest group [4, 5, 25, 26]. The pathophysiology responsible for the hemodynamic and cardiovascular changes in hypertension continues to be a perplexing problem.

Blood pressure is the product of cardiac output ($\dot{Q}$) and total peripheral resistance (TPR). This enables a variety of cardiovascular and hemodynamic factors to influence blood pressure. Changes in cardiac output are caused by changes in either stroke volume or heart rate. While venous capacitance affects the stroke volume, the blood viscosity, elasticity of the arterial delivery system, and the degree of the resistance of the arteriolar bed affect TPR. All of these factors influence blood pressure.

Hypertension or the state of elevated blood pressure has been defined by a variety of different criteria. The Joint National Committee III (1985) [26] recommended that 140/90 mmHg be used as the threshold for hypertension. Blood pressure values above this

reading are considered hypertensive, and below this, normal. Hypertensives can then be further categorized by diastolic blood pressure (DBP) reading into:

90-104 mmHg	mild hypertension
105-114 mmHg	moderate hypertension
≥ 115 mmHg	severe hypertension

or systolic blood pressure (SBP) readings:

140-159 mmHg	borderline isolated systolic hypertension
≥ 160 mmHg	isolated systolic hypertension

The World Health Organization uses the following criteria for blood pressure classification:

SBP <140 mmHg & DBP< 90 mmHg	normotensive
SBP 141-159 mmHg & DBP 91-94 mmHg	borderline hypertension
SBP ≥ 160 mmHg & DBP ≥ 95 mmHg	hypertensive

The American College of Sports Medicine [27] uses a variety of groupings to identify hypertension:

Blood pressures:

<140/90 mmHg	normotensive
≥140/90 mmHg	borderline to mild hypertension
≥150/95 mmHg	mild to moderate hypertension
≥160/100 mmHg	moderate to severe hypertension
≥170/110 mmHg	uncontrolled hypertension

There are two major classifications of hypertension. Primary or essential hypertension is responsible for about 90% of all cases of hypertension [27]. The mechanisms responsible for this type of hypertension remain unknown. The other category, known as secondary hypertension, is related to damage or dysfunctioning of a organ within the body or some identifiable endocrine problem. In essential hypertension, an elevation in vascular resistance is the dominant physiological finding.

Epidemiology

A number of different screening programs have been used to define the magnitude and scope of hypertension. The data from these studies have provided information on both the prevalence and incidence of hypertension. Prevalence refers to the total number of occurrences at a point in time, while incidence refers to the number of new cases that occur within that group. The collection of incidence data requires greater time, but provides some important associations and developments.

The Community Hypertension Evaluation Clinic (CHEC) Program, [25] examined over one million adults in the United States between 1973 and 1975. Both systolic and diastolic blood pressure differed as a function of sex and race. In blacks over 30 years old, SBP was higher than in whites, and men had higher systolic blood pressures than women across all race groups up until age 50 in blacks and age 65 in all other race groups. The same general trend was evident for DBP with the exception that it reached a peak at around age 60. The prevalence of hypertension (DBP $\geq$ 90 mmHg) rose from 39.9/1000 for adults under 20 years of age to 348.8/1000 for those 60 to 64 years old. In the 30-39 year old age group, blacks had a 43% higher prevalence of hypertension than whites. An alarming fact disclosed by this study was that 55% of people were either untreated, uncontrolled, or unaware of the fact that they had hypertension. The black male group had

about 77% undiagnosed, untreated, or uncontrolled cases, while only about 62% of the white males fell into this category.

The Hypertension Detection and Follow-up Program (HDFP) was designed to help increase awareness and to look at ways to improve hypertension control [5]. Fourteen clinical centers across the United States were selected as test sites. The first screening revealed that mean DBP was higher in blacks than in whites across all age groups, and values for men were higher than women. Blacks had a 2 to 4.5 fold increase in the prevalence of hypertension (DBP ≥95 mmHg) compared to whites. After the initial screening, 76% of the people with hypertension participated in a second screening. During this second screening, 29% of the blacks and 39% of the whites did not exhibit a hypertensive condition (DBP < 90 mmHg). This points to the importance of getting more than one blood pressure measurement to establish the presence of the disease.

The results of the Subcommittee on Definition and Prevalence of the 1984 Joint National Committee [26] provided updated information on the prevalence of hypertension. Hypertension was defined as blood pressure values ≥ 140/90 mmHg or the person being on antihypertensive medication. The prevalence was higher for men than women (33% vs. 27%) and blacks than whites (38% vs. 29%). The prevalence of hypertension increased with age. The results showed that one out of every three people in the United States had hypertension. The prevalence increased to over 50% for those over 55 years of age. Only about 54% of the people with hypertension were aware that they had a problem.

The National Health and Nutrition Examination Survey I (NHANES I) Epidemiologic Follow-up Study (NHEFS) examined the current prevalence and incidence of hypertension [4]. This follow-up study was done 7 to 12 years after the original data were collected. Only about 40% of the individuals in either the lowest or highest quintile for SBP remained in that group during the follow-up. The incidence of hypertension between men and women was very similar. The incidence of hypertension increased about

5% for each increase of 10 years in whites and about 10% for each 10 years in blacks. In hypertensive adults over 50 years of age, the number surviving 12 years later was 48% for white men, 68% for white women, 63% for black women, and 51% for black men.

The Framingham Heart Study was used by Leitschuh et al. [3] to examine the progression of hypertension (SBP ≥ 140 mmHg/DBP ≥ 95 mmHg) over a 26 year period. Hypertension developed in 33% of the men and 42% of the women with normal blood pressure (SBP< 140 mmHg /DBP <85 mmHg) and in 71% of the men and 78% of the women with high-normal blood pressure (SBP <140 mmHg/DBP 85-89 mmHg). Those individuals with a high-normal blood pressure were at a greater risk of developing hypertension (2.1 times for men and 1.9 times for women). The risk factors associated with hypertension for individuals with normal BP were baseline SBP and DBP, and relative weight. Women in the normal group also had serum cholesterol as a risk factor. Diastolic blood pressure was a risk factor for men in the high-normal range.

In two different intervention studies, [28, 29], blood pressure was reduced with a reduction of sodium and alcohol, an increase in exercise and weight loss while on a low fat diet, or one of these combinations: reduced calories, reduced sodium, reduced calories and sodium, or increased potassium. The incidence of hypertension was only 8.8% in the intervention group compared to 19.2% for the control group, after the 5-year follow-up period [28]. Both studies show promise in the potential of reducing the incidence of hypertension.

Horan and Lenfant identified risk factors that have been related to the development of hypertension [30]. Included in this list of risk factors were age, obesity, excessive sodium intake, excessive alcohol intake, and a sedentary lifestyle. The identification and use of different potential predictors of hypertension was reviewed. These potential predictors were grouped into 5 categories: demographic (race, gender, and industrialized society) , clinical (rapid heart rate, obesity, biochemical markers, BP in children, left

ventricular mass), genetic (restriction fragment length polymorphisms (RFLPs), challenge response (exaggerated BP response to exercise, stress) and laboratory (cation transporters, hormones) predictors.

Exaggerated BP Response to Exercise

The use of exercise to examine the body's ability to adjust and regulate blood flow is not a new methodology. An increase in blood pressure is a normal response to an exercise stimulus, but some individuals have an exaggerated blood pressure response. First and foremost, it is important to start with a working definition of an exaggerated blood pressure response. Wilson and Meyer [10] used a maximal blood pressure of ≥225/<90 mmHg as a definition of an exaggerated blood pressure. Others have used >200/100 mmHg [6], SBP≥200 or an increase in DBP [31], SBP>200 mmHg and/or an increase in DBP of 10 mmHg to > 90 mmHg [7], ≥230/≥110 mmHg [8], or a systolic BP response that is one to two standard deviations above the mean and/or a diastolic increase of 10 mmHg above the resting measurement [9] . Like the multiple definitions used to define hypertension, the exaggerated blood pressure response during exercise encompasses a variety of definitions, which all point to an abnormal response.

The blood pressure response during a maximal treadmill test was used by Wilson and Meyer [10] to predict future development of hypertension (SBP ≥140 mmHg/DBP ≥90 mmHg). Over 3800 subjects were classified according to both resting BP and BP during treadmill testing. A maximal BP of <225/<90 mmHg was classified as normal and anything above that as an exaggerated response. The interesting finding of this study was that 12.4% of the subjects who had normal resting BP, had an exaggerated BP response to the exercise testing. This group with the exaggerated BP response had a two-to-three fold greater probability of developing hypertension than those with a normal BP response. The

BP response during the exercise test provided a better predictor of future hypertension than resting BP. Family history of hypertension was an additional risk factor in the development of hypertension.

The blood pressure response to submaximal cycle ergometer testing in normotensive, borderline hypertensive, and hypertensive subjects was examined by Franz [6]. The normotensive male group (resting BP 131±11/80±7 mmHg) showed a linear increase in both heart rate (HR) (98±11 to 126±13) and SBP (155±12 mmHg to 188±14 mmHg) to a series of absolute workloads (50 watts to 100 watts in 10 watt stages of one minute each). DBP showed a slight increase from 86±7 mmHg to 92±8 mmHg. Females showed the same trends as the males. Age did influence the BP and HR responses of the subjects as the older subjects had higher SBP and DBP responses before, during, and after exercise, and lower HRs over the same periods. The borderline hypertensive group had resting blood pressures of ~ 150/92, while the hypertensive group has blood pressures of 172 ±7/104 ±4 mmHg. In the borderline group, 58% revealed an exaggerated blood pressure response (>200/100 mmHg) during the exercise test, which was similar to the response of the hypertensive group. At a follow-up testing session 3.8 years later, 97% of the borderline hypertensives who had an exaggerated BP response developed hypertension, and 68% of those with a negative response demonstrated a normal response. This response pattern is similar to the work of Wilson and Meyer [10]. The hypertensive group, on average, had higher blood pressure responses across all stages of the exercise test, but the resting value could not be used to determine the extent of this BP response. This study must be viewed with some caution as all subjects performed the same absolute work rate and this would account for some of the differences in the BP response. However, exercise testing is clearly an asset in the future identification of hypertension.

An exaggerated blood pressure response (SBP>200 mmHg and/or an increase in DBP of 10 mmHg to > 90 mmHg) in male normotensives (≤140/90 mmHg) to a submaximal cycle ergometry test was used by Dlin et al. [7] to predict the development of hypertension. A group of 75 individuals with an exaggerated BP response were matched for age, BMI (weight/height^2), family history of hypertension, HR, aerobic fitness, and smoking with a group that showed a normal response to exercise. In a follow-up (3 to 14 years), 10.6% of the subjects with an exaggerated BP response became hypertensive, while none of the matched controls became hypertensive. The exercise SBP response was able to explain about 18% of the final resting SBP and the exercise DBP response 14% of the final resting DBP. Due to the young age of the subjects (25 years old at start), there appeared to be good evidence that an exaggerated BP response to exercise might aid in the identification of people at risk for hypertension.

Exercise test (90% of age-predicted HR) results were used by Jackson et al. [8] to evaluate the possibility of predicting future hypertension (≥140/≥90 mmHg). An exaggerated BP response during exercise (either cycle or treadmill) was defined as ≥230/≥110 mmHg. Of the 4,856 individuals tested, 3.4% had an exaggerated BP response to the exercise test. The important fact is that of the subjects with an exaggerated BP response during the exercise test, 32% had a normal resting BP. During a 2 to 4 year follow-up period, 52% of the patients with an exaggerated BP responses who returned had hypertension, while only 15% of a control group who had a normal BP during exercise developed hypertension. This three fold greater development of hypertension in the exaggerated BP group lends support to the use of exercise testing in the early identification of people at risk for the development of hypertension. Exercise mode did not have an effect on the exercising BP, but the BP responses of women were generally less than that of men.

Jette et al. [9] reviewed a number of the earlier studies that examined the use of an exaggerated BP response to exercise to predict future hypertension. Despite a great deal of variability, the risk of developing sustained hypertension was 2 to 10 times greater in those individuals who had an exaggerated BP response to exercise. The authors examined the blood pressure responses in normotensives (<140/90 mmHg at rest) during the Canadian Aerobic Fitness Test (CAFT). An exaggerated BP response was viewed as a systolic BP response that was two standard deviations above the mean and/or a diastolic increase of 10 mmHg above the resting measurement. This test allowed the assessment of BP during a submaximal exercise bout of about 65-70% of one's estimated maximal aerobic power while minimizing the risk of potential complications that could be experienced during higher intensity exercise. Estimates of the prevalence of an exaggerated BP response in the Canadian population were made from a representative sample of 15,519 males and females between the ages of 7 to 69 years. From the 1981 population figures, it was estimated that about 176,000 Canadians between the ages of 20-69 years who exhibited an exaggerated BP response to the CAFT would have developed sustained hypertension by 1988. This represents about one-third of the individuals with an exaggerated BP response.

Post-exercise blood pressure was measured to evaluate its potential as a predictor of subsequent hypertension in currently normotensive subjects [31]. Over 700 normotensive (SBP<150/DBP<90) subjects were given an exercise test on a cycle ergometer to 70% of age-predicted maximum HR, and supine BP was recorded 30 seconds later. The subjects were followed over the next 15 years. During the follow-up examination, 33% of the subject population had developed hypertension, with 34% of this group having had exaggerated BP values (SBP≥200 or an increase in DBP) during exercise. Of the subjects with a normal BP response to exercise, only 11% developed hypertension. This submaximal exercise test and the criteria for an exaggerated BP response were able to correctly predict the development of hypertension in 17% of the subjects. This submaximal

test did not have the same predictive strength as a maximal test, but it did point out some individuals who were at risk.

Chaney and Eyman examined the BP response to dynamic (maximal treadmill test) and isometric [60 second maximal voluntary contraction (MVC)] exercise in 100 normotensive (<140/90 mmHg) white men [2]. During the 14 year follow-up period, 16 of the 100 subjects developed hypertension. Using the BP responses measured at rest and during the various exercise tests, resting DBP was determined to be the best single predictor for the development of hypertension. People with high normal resting BPs or an unusually exaggerated BP response to dynamic or static exercise were more prone to develop hypertension. Combining the resting DBP with the isometric DBP response provided a better means of identification of subjects at risk for hypertension than resting DBP alone. This combination of BP values reduced the false classification of future hypertension from 31% to 20%.

Guerrera et al. [11] investigated the BP response measured during maximal cycle ergometry in borderline hypertensives (140/90 to 160/95) to the subsequent development of hypertension over a two year period. Subjects performed a maximal exercise test to age-predicted maximal HR or until an excessive BP (>250/>130 mmHg) occurred. After this first testing session, this group of 28 males was divided into two groups: normal exercise response (n=13) and an exaggerated exercise response (n=15) (maximal BP >220/ >105). All subjects were observed over the next 2 years with casual BP measurements taken every 3 months. These groups were similar in age, smoking habits, family history, resting SBP, resting DBP, resting HR, height, and weight. Only two people in the normal exercise response group became hypertensive, while 7 had a reduction in resting BP and were now normotensive. In the exaggerated exercise response group, 10 became hypertensive, while the other 5 remained borderline hypertensive. This provided rather good evidence for the use of maximal exercise testing in the evaluation of the risk for hypertension.

Fiber Types

The identification of muscle fiber type gained popularity with the work of Saltin et al.[32]. Skeletal muscle fibers can be categorized into two basic groups: slow twitch (type I) or fast twitch (type II). This basic designation is based upon the performance and metabolic characteristics of the fibers. The fiber types of endurance runners were compared to sprinters, and sedentary individuals to examine differences in fiber composition, metabolic profile, capillary density, and training adaptations. The endurance runners possessed a greater percentage of type I fibers (more than 60% type I), while sprinters exhibited less than 30% type I fibers, and the control subjects had about 54% type I fibers [33]. The classification of the muscle fibers can be subdivided into type I, type IIa, and type IIb fibers utilizing the myosin ATPase stain following preincubation at three different pH values [34]. This differentiation of the type II fibers into type IIa and type IIb is based upon the level of myofibrillar ATPase stability [33]. The type IIa fibers are more fatigue resistant than the type IIb fibers. The generally smaller type I fibers (~ 5300 μm^2) in men provides an excellent environment for oxygen exchange, while the generally larger type II fiber (~ 5600-6100 μm^2) has a greater diffusion distance.

The variability in distribution of muscle fiber types was studied by Elder et al. [35]. The overall mean percentages of the different fiber types were very similar in different muscles of the same subject. The vastus lateralis exhibited the lowest between-site variation of the four muscles studied (soleus (7.67%), triceps (8.12%), biceps (7.61%), vastus lateralis (6.63%)). Three sample sites within the muscle were found to be necessary to reduce the between-site standard deviation to under 5% for the vastus lateralis, four sample sites for the triceps, and five sample sites for both the biceps and soleus. The mean % of type II fibers in this study was found to be 57% for the vastus lateralis (range 43-69%), 55% for the biceps (range 42-72%), and 39.5% for the triceps (range 25-57%).

There were no differences in the distribution of type II fibers in either superficial or deep samples from the muscle bellies of the vastus lateralis, biceps, or triceps. Although there was both between site and within site variability in muscle fiber type, the degree of variability could be reduced with multiple sites and samples of 200-300 fibers.

Gollnick et al.[36] studied the muscle fiber composition of trained and untrained men. Muscle samples from the vastus lateralis and deltoid were compared to determine the variability of fiber type within the same individual. Fiber types were classified as type I or type II using myosin ATPase activity. The average percentage of type I muscle fiber was similar in both muscle samples (vastus lateralis had 40% type I fibers, and deltoid had 45% type I fibers). The depth and site of the muscle biopsy was presented as an important variable in the comparison of fiber type variability. A 4.6% difference in fiber type distribution was discovered within different biopsy sites of the same vastus lateralis muscle (with variation of biopsy site of 4 cm above or below the standard 12-16 cm above the patella site). There was a relationship between the distribution percentage of type I fibers and the area occupied within the muscle by that fiber type.The cross-sectional area of trained muscle fibers tended to be greater than untrained fibers and the type II fiber area tended to be 20% larger than type I fibers in most subjects. The oxidative capacity (assessed by SDH activity) of both type II and type I muscle fibers was higher in the trained subjects. This provided for a more economical utilization of the glycogen stores and an enhanced ability to oxidize fatty acids.

Costill et al.[37] examined muscle biopsies from the lateral head of gastrocnemius in elite distance runners, trained middle distance runners, and untrained men. The elite runners had higher percentages of type I fibers (79%) than the middle distance runners (62%) or untrained men (58%), and a greater type I cross-sectional area than either group (83% vs. 62% vs. 60%). The muscle's oxidative capacity, as represented by succinate dehydrogenase (SDH) activity, was 3.4 and 2.8 fold greater in the elite and middle distance

runners than in the untrained group. This significantly greater SDH level in the distance runners was not related to the percentage of type I fibers (r=0.22). The magnitude of the cross-sectional fiber area was of special importance. The elite runners had cross-sectional areas of type I fibers that were 29% greater than the type II fibers. A later study [38] showed that endurance training increased the area occupied by the type I fibers by 10%, but there was no change in the percentage of type I fibers.

Costill et al. [39] showed that distance runners (both males and females) have a greater percentage of type I fibers than sprinters, throwers, and jumpers. Regardless of training, the size of the different fibers were generally smaller in women, and in the untrained. The range in the percent of type I fibers differed greatly between the male sprinters (21-27%), distance runners (63-74%), and untrained controls (38-73%).

The muscle fiber profile of endurance athletes was compared to that of a control group by Tesch et al. [40]. Again there is a trend showing that the control group had more type II fibers (61%) than that found in the endurance athletes (40% type II fibers).

Capillary Density

Capillary density has been determined by a number of methods: a) capillaries per fiber, b) capillaries per mm^2, c) number of capillaries around a specific fiber type, d) number of capillaries around a fiber type per fiber type area. Each of these different methods presents a little different picture, and the method selected can have a big impact on the how the situation is viewed.

In the capillaries per fiber method, the total number of capillaries adjacent to each fiber type is averaged for a specific area of the muscle sample. The capillary density of the different fibers appears to favor an increased blood supply to the type I fiber. The type I fibers are associated with a greater number of capillaries (3-4 cap/fiber) and usually are a little smaller in cross-sectional area than the type IIa fibers (2.5-3 cap/fiber) and the type IIb

fibers (2-3 cap/fiber) [33]. The number of capillaries associated with a specific fiber type becomes more apparent when examining a homogeneous fiber type area. In this situation, the type II fibers had 1-4 capillaries per fiber, while the type I fibers had 4-11 capillaries per fiber [33]. The capillary density expressed as the number of capillaries per cross-sectional area of a specific fiber type provides an indication of the availability of oxygen and nutrients for the muscle fiber.

The relationship of capillary density to muscle fiber type was examined by Andersen [41] in men. The number of capillaries around type I fibers (4.9) and type IIa fibers (4.5) was similar, and both were greater than that found around the type IIb fibers (3.5). The number of capillaries per fiber area for the type IIa fibers and the type IIb fibers was found to be less than that for the type I fibers. Aerobic training (20 min, at ~80% $\dot{V}O_{2max}$, 4 times/week) for seven weeks was responsible for an increase in capillary density from 289 cap/mm^2 to 356 cap/mm^2.

The capillary density issue was also examined in the trained and untrained vastus lateralis muscles of 23 men [42]. The key issue was the number of capillaries per fiber area and the association between fiber diameter and mitochondrial content. The trained athletes averaged 41% more capillaries per fiber (~5.87 vs. ~4.43). This represented less sharing of blood supply between fibers in the trained group. When the data were expressed relative to the number of capillaries per mm^2, the trained athletes still maintained ~40% greater capillary number. The overall diameter of the muscle fibers was not different between the trained and untrained subjects (49.1μm ±1.1 vs. 48.8 μm ±2.5). The number of capillaries around a muscle fiber was related to the mitochondrial content of the muscle fiber. The type I fibers had a greater capillary density than either the type IIa or type IIb fibers.

Muscle biopsies of the vastus lateralis of trained and untrained young men were studied to compare the effects of training on capillary density [43]. The trained men had

about a 30% greater muscle fiber area than that of the untrained subjects (440 ±36.5 fibers/mm^2 vs. 557 ± 30.0 fibers/mm^2). The number of capillaries per square millimeter were similar between the two groups (~600 capillaries/mm^2). With the same number of capillaries per square millimeter in the two groups, the larger muscle fibers in the trained men result in their having more capillaries per muscle fiber. This represents an average of about 1.5 capillaries per fiber in the trained group and only 1 capillary per fiber in the untrained group. The effects of training appear to result in an increased fiber size and greater capillary number.

The results of endurance training were demonstrated by the comparison of the number of capillaries in trained and untrained athletes [40]. The number of capillaries per fiber was significantly greater in the endurance athletes (3.11 cap/fiber) than in the control group (2.16 cap/fiber). The number of capillaries per mm^2 was less in the control group (306±29) than in the endurance trained athletes (401±61).

The influence of endurance training on capillary density was also examined in trained and untrained females [44]. The average number of capillaries per fiber was 52% greater in trained than untrained females (1.69 ±0.13 vs. 1.11 ±0.07). A positive relationship was noted between the number of capillaries found around a muscle fiber (4.42±0.31 cap/fiber vs. 3.04 ±0.17 cap/fiber) and the mitochondrial content of that muscle fiber. The muscle fibers with the greatest mitochondrial content had larger cross-sectional areas in both men and women.

Henrich et al. [45] presented data pointing to rarefaction of the capillary bed in the skeletal muscle bed of hypertensive humans. The quadriceps muscle of the normotensive group revealed a 37% greater capillary density than in the hypertensive group. The pectoralis major muscle showed an even greater level of rarefaction with a 51% difference (232 ±28 capillaries/2.5 mm^2 vs. 477 ±30 capillaries/2.5 mm^2). Since vascular resistance is associated with the fourth power of the inner radius of the vessels, this reduction of the

surface area of the capillary bed may affect the vascular resistance. The magnitude of this effect might not be very noticeable at rest or during mild exercise, however, as the vascular system comes under greater stress during more intense exercise, the limitations of the capillary bed would become more apparent.

Muscle Fiber Type as a Determinant of BP Responses

The early work of Juhlin-Dannfelt et al. [22] provided the first association between muscle fiber type and hypertension. The fiber type of the vastus lateralis muscle of age-matched normotensives (n=22) and hypertensive (n=19) subjects was utilized for making comparisons related to blood pressure and blood flow at rest. The muscle fiber composition (myofibrillar ATPase staining) was characterized into red, high-oxidative, low-glycolytic, slow-twitch (ST) or type I fibers, and white, low-oxidative, high-glycolytic, fast-twitch (FT) or type II fibers. A significant negative correlation was found between the percentage of type I fibers and the mean arterial pressure during both sitting and standing. No significant relationship was found between fiber type and leg blood flow, but leg vascular resistance fell in both groups as the percentage of type I fibers increased. Subjects with higher percentages of type II fibers had higher vascular resistance and higher blood pressures. This association between the percentage of type II fibers and increased vascular resistance suggested a potential interaction between fiber type and blood pressure.

Frisk-Holmberg et al. [23], provided a look at the interaction between muscle fiber type and blood flow during dynamic exercise. Twelve healthy, physically active (relative $\dot{V}O_{2max}$ between 46.7 ml· kg^{-1}· min^{-1} to 65.1 ml· kg^{-1}· min^{-1}) males exercised at approximately 50% of $\dot{V}O_{2max}$ on a cycle ergometer for about 10 minutes. Muscle fiber type (vastus lateralis) averaged 49% ±4% type I fibers (range 26 to 66% type I fibers) in these subjects. The exercise intensity was responsible for more than a six fold increase in

leg blood flow compared to resting conditions. Leg blood flow (infusion of indocyanine green dye) during exercise was greater in those subjects with a higher percentage of slow twitch muscle fibers. This relationship was not apparent during the resting conditions of Juhlin-Dannfelt et al. [22]. The leg blood flow during resting conditions represents flow to other than muscle (e.g. skin and subcutaneous tissues). The relationship between the percentage of type I fibers and leg blood flow points to a possible difference in the vascular bed and blood supply to the exercising muscle. The possibility exists that this greater blood flow is somehow related a greater capillary density associated with the type I fibers.

Frisk-Holmberg et al. [46], found a positive relationship between mean arterial blood pressure (MABP) and the percentage of type II fibers (vastus lateralis muscle) during supine rest. This investigation used 17 essential hypertensive patients (HT) and 17 age-matched normotensive (NT) control subjects. An isometric exercise test (33% of maximum voluntary contraction (MVC) for one minute) was utilized as a stressor to the cardiovascular system. The hypertensive patients had significantly higher values than the normotensive controls for the following resting variables: weight, systolic, diastolic, and mean arterial pressures, and resting heart rate. There was a greater percentage of type II fibers in the HT group ($p<0.1$). There was a correlation between the percentage of type II fibers and the MABP in the supine resting condition ($r=0.68$, $p<0.01$ for HT and $r=0.47$, $p<0.1$ for NT). During the isometric test, the maximal change in MABP (17±7 mmHg vs. 20±11 mmHg) and the percentage of type II fibers showed a positive correlation ($r= 0.62$, $p<0.01$ for HT and $r=0.47$, $p<0.1$ for NT). When the relationship between the percentage of type II fibers and ΔMABP was observed by age groups, a stronger relationship was evident. This ΔMABP was greatest in those subjects with the greatest percentage of type II fibers. This positive relationship between the percentage of type II fibers and the rise in BP with isometric exercise provides room for some speculation about the importance of fiber type in the cardiovascular adjustments to exercise. During isometric exercise, the BP

response could be triggered by high levels of metabolites from the type II fibers. The change in HR (4 ±3 b/min vs. 6 ±6 b/min) did not differ significantly between the NT and HT groups during the isometric exercise.

Fitton [24] studied the BP response in sprinters and distance runners to both dynamic and static exercise. During cycle ergometry, the male sprinters had greater DBP and MABP responses at 40, 60, and 80% of $\dot{V}O_{2max}$. During static exercise (30% MVC to fatigue), the male sprinters had greater SBP, DBP, and MABP responses across all fatigue intervals compared to the endurance runners. This study lends support to the notion that muscle fiber type does play a role in the blood pressure response to both static and dynamic exercise. The assumption of a type II predominance in sprinters and the greater pressor response to dynamic and isometric exercise provides some evidence that this phenomenon is not just a coincidence.

CHAPTER III

METHODS

Subjects

Healthy, white males (ages 18-30 years) were recruited from the University of Tennessee-Knoxville student body and faculty, and the surrounding community. The subject pool was comprised of 18 normotensive individuals (6 sprinters, 6 distance runners, and 6 control subjects). The nature of the study was explained, and the subjects were asked to sign an informed consent form (see Appendix C) prior to starting the study. Subject selection was based upon a normotensive screening blood pressure, a negative medical history (see Appendix A), and meeting the appropriate running standards. The subjects also filled out a questionnaire on parental history of hypertension (see Appendix B) prior to completion of testing.

Criteria for exclusion of subjects:

1. Casual, supine blood pressure > 140/90 mm Hg
2. Contraindications to exercise testing (see Appendix A)
3. Sprinters slower than 11.3 seconds for 100 meters, 25 seconds for 200 meters or 52 seconds for 400 meters, and distance runners slower than 16:45 for 5,000 meters.

Testing Sessions

Anthropometric Measures. Height (m), weight (kg), and three skinfolds (mm) were measured. Body mass index (BMI) was calculated using the formula: BMI=weight

(kg)/ height (m^2). Percent body fat was estimated from the sum of three skinfolds (chest, triceps, and subscapular) [47].

Blood Pressure. Resting (after 20 minutes) supine blood pressure measurements and heart rates were measured during screening, prior to the maximum oxygen uptake test and the graded exercise test, and a resting (20 minutes), seated BP was measured prior to the isometric handgrip test. The blood pressure was measured in the non-dominant arm with the use of an automated BP monitor (Colin STBP 780, Colin Medical Instruments, Plainfield, N.J.). The monitor receives a signal from the ECG and only monitors for Korotkoff sounds after each QRS complex, which reduces noise artifact. Appropriate cuff sizes were selected, and systolic and diastolic pressures, and heart rate were recorded. A mercury sphygmomanometer was connected to the cuff, and a stereo headset was used to simultaneously monitored the STBP 780 recordings by the investigator The fourth phase Korotkoff sound was used to measure DBP. The average of three resting (after 20 minutes) blood pressure measurements and heart rates were used to determine the resting values.

Isometric Handgrip Protocol. Maximal isometric handgrip strength (kg) was measured with a handgrip dynamometer after the measurement of the resting blood pressure on the day of screening. The best of three trials with the dominant arm was considered the maximal isometric strength.

On the day of the isometric exercise test, a 21-gauge teflon catheter was inserted into an antecubital vein of the non dominant arm. The subject then rested in the seated position for 20 minutes. Three blood pressure measurements and HRs were taken and the average of these three values was used as the resting measure. A blood sample was then drawn for measurement of blood lactate. The blood sample (50 μL) was placed into whole blood lactate tubes from Yellow Springs Instruments. The subject performed an isometric

contraction at 30% maximal voluntary contraction (MVC) for 3 minutes. Blood pressure measurements and HRs were taken at the end of each minute of isometric exercise. The subject viewed the handgrip dynamometer and received verbal encouragement from the experimenters during the test. A post-test blood sample (50 μL) was taken within the first minute post-exercise and stored in the same manner as the pre-test sample. Samples were analyzed with a Yellow Springs Instrument Model 27 Industrial Analyzer (Yellow Springs, Ohio) at the end of the testing session.

Maximal Oxygen Uptake Protocol. Each subject performed a maximal oxygen uptake test on a cycle ergometer. The subject began unloaded cycling for one minute, and the work rate was increased by 35 W each minute until he could no longer maintain the pedal cadence. O_2 uptake and CO_2 production were measured with an automated gas-collection system using the Rayfield REP-200C interface and software (Rayfield Equipment, Waitsville, Vt.), and an Apple II-plus computer. Percentages of expired O_2 and CO_2 were measured with an Applied Electrochemistry S-3A oxygen analyzer and CD-3A carbon dioxide analyzer (Sunnyville, Calif.) during each minute of the test. Standard gases analyzed by the Scholander technique [48] were used to calibrated the analyzers prior to testing. Peak oxygen uptake was taken as the highest oxygen uptake recorded during the test. The subject's blood pressure and heart rate were recorded during the last 30 seconds of each stage of the exercise test. A blood sample (finger stick) was taken in the second minute of the recovery period in only the sprinter and distance groups to measure lactate. The blood sample (50 μL) was placed in whole blood lactate tubes which were analyzed at the completion of the exercise test.

Submaximal Dynamic Exercise Protocol. The subject reported to the lab after an overnight fast, and prior to caffeine ingestion and physical exertion. Following a 5-minute supine rest period, a 21-gauge teflon catheter was placed in an antecubital vein in the non-dominant arm to obtain blood samples for lactate analysis. An additional 20

minutes of supine rest preceded the beginning of the submaximal exercise testing session. Blood samples (50 μL) were taken at the end of each test period (supine rest, bike rest, 20%, 40%, and 60% of $\dot{V}O_{2peak}$. Each subject cycled at each stage for 6 minutes and blood pressure, heart rate, oxygen uptake, and cardiac output were measured during each stage. The oxygen uptake and carbon dioxide production were measured during each 30-seconds of the test and values at minute 4 (HR, BP, $\dot{V}O_2$, and $\dot{V}CO_2$) were taken as steady-state values. After a blood sample was taken at minute 4, cardiac output measurements were performed. The next stage was started after the completion of the cardiac output measurement.

Cardiac Output Measurements. The CO_2 rebreathing technique was used as a non-invasive means of measuring cardiac output [49]. Arterial pCO_2 was approximated from end-tidal pCO_2, measured with a rapid-response infrared CO_2 analyzer. The average of 10 end-tidal pCO_2 values taken prior to a rebreathing trial were used to estimate arterial pCO_2 according to the formula of Jones et al. [50].

The subject then rebreathed CO_2 and O_2 from a latex bag to allow a rapid equilibration of venous pCO_2. A valid equilibrium measure during a trial was determined by a "plateau" in pCO_2 according to the criteria of Jones et al [51] and Ashton and McHardy [52]. This required that within the first 6-8 seconds that the inspiration and expiration pCO_2 readings were within ±1 mm Hg of each other. If a trial had to be repeated, the end-tidal pCO_2 values were allowed to return to baseline before redoing the trial. If a clear plateau was not reached, the "downstream" correction factor of Jones [53] was used. An automated gas mixing apparatus was utilized to adjust the gas volumes and initial pCO_2 mixtures placed in the latex bag[53].

Arterial and venous CO_2 levels ($CaCO_2$ and $CvCO_2$) were estimated using a standard dissociation curve. The indirect Fick equation was used to calculate cardiac

output: $CO = \dot{V}CO_2 / (CvCO_2 - CaCO_2)$. The cardiac output values were also expressed as cardiac index (CI), with body surface area estimated by the method of DuBois [54].

Muscle Biopsies. The muscle biopsy method according to Bergström [55] was used with 4 mm and 5 mm biopsy needles. The sampling site was locally anesthetized before the sample was taken. The muscle sample was taken 18 to 20 cm above the patella, 5-6 cm lateral from the midline, from the vastus lateralis muscle of the right leg of each subject.

The sample was then frozen in isopentane that was cooled with liquid nitrogen. The samples were stored at -70°C until sectioned and stained for determination of fiber type and capillary density. Serial sections were cut in a cryostat (-20° C) and mounted on glass cover slides for histochemical analysis. The samples were stained for myosin ATPase by the method of Dubowitz [34] for determination of fiber type (see Appendix D). Capillary density was determined with the amylase (PAS) staining procedure of Pearse [56] (see Appendix E).

Muscle fiber type percentages were determined by counting all of the available fiber from a photomicrograph of the muscle sample. The average number of muscle fibers in the samples counted was 416 fibers.

Capillary density was determined by first taking a photomicrograph utilizing the same standard focal length for all of the samples. A 0.5 mm x 0.5 mm section of the photomicrograph was utilized to count the number of capillaries in the section. This values was then multipled by 4 to give our capillary density in capillaries per mm^2.

Data Analysis

The descriptive characteristics (height, weight, sum of skinfolds, body mass index, body surface area, peak oxygen uptake, age, resting SBP, DBP, MABP, HR, %

type II fibers, and capillary density) of the sprinters, distance runners, and controls were compared with a one-way ANOVA. Parental history of hypertension was analyzed with a Chi-squared test of independence.

A two-way ANOVA, (Group x Time) with repeated measures was used to analyze the dynamic and isometric exercise conditions. When a significant interaction was found, a Scheffe post-hoc test was used to determine where the differences were located. Differences were considered significant at $p<0.05$.

CHAPTER IV

RESULTS

Descriptive Characteristics

The descriptive characteristics of the three groups of six subjects is found in Table A1.* The distance runners weighed less, had less body surface area, and a lower body mass index (kg/m^2). The distance runners had a significantly greater capillary density and significantly less hand grip strength than the sprinters. The control group had a greater sum of skinfolds than either of the two experimental groups. There were no significant differences among the groups in resting HR, blood pressure measures (SBP, DBP, or MABP), parental history of hypertension ($p=0.7251$), or fiber types, although the percentage of Type I fibers did approach significance ($p<0.06$).

Maximal Oxygen Uptake Determination

During the graded exercise test used to determine maximal oxygen uptake, there were no significant differences between the groups for HR or blood pressure responses. The distance runners had a greater relative ($ml \cdot kg^{-1} \cdot min^{-1}$) maximal oxygen uptake (62.9 ± 1.5 $ml \cdot kg^{-1} \cdot min^{-1}$) than either the sprinter (46.9 ± 1.0 $ml \cdot kg^{-1} \cdot min^{-1}$) or control group ($46.5 \pm 2.0$ $ml \cdot kg^{-1} \cdot min^{-1}$) ($p<0.0001$).

Isometric Exercise

During isometric exercise the distance runner group maintained less absolute force than the sprint group (16.0 ± 0.9 kg vs. 19.8 ± 0.7 kg). Table A2 shows that the distance runners had a lower heart rate response than the sprint group across all three minutes of the

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* All tables and graphs may be found in the appendix.

isometric exercise (Graph B1). The blood pressure response reached significance during the third minute of the isometric exercise challenge. During this time period, the distance runners demonstrated lower systolic, diastolic, and mean arterial blood pressures than the sprint group (Graphs B2a-c). At the end of the three minutes of isometric exercise in the present study, the sprint group showed a 2-fold greater increase in HR, a 1.8-fold greater increase in SBP, a 1.5-fold greater increase in MABP, and a 1.3-fold greater increase in DBP compared to the distance runners.

Dynamic Exercise

There were no differences in the resting values between the groups before the start of the dynamic exercise protocol (see Table A3). There were no significant differences in the HR, or blood pressures at 20%, 40%, or 60% of $\dot{V}O_{2peak}$ (Graphs B3 & B4a-c). In addition, there were no differences in TPR during the exercise challenge. There was a trend towards a greater cardiac output in the distance runners ($p<0.09$). When adjusted for the differences in body surface area by utilizing cardiac index, there was a significant main effect that occurred at 60% of $\dot{V}O_{2peak}$, for cardiac index ($p<0.0006$) (Graph B5) and for SVR ($p=0.0001$) (Graph B6).

CHAPTER V

DISCUSSION

The primary purpose of this study was to examine the cardiovascular responses of sprinters and distance runners to isometric and dynamic exercise in an attempt to see the role that training and muscle fiber types (genetics) may have on the hemodynamic responses to exercise. The use of both isometric and dynamic exercise provided an opportunity to examine cardiovascular control mechanisms during conditions in which cardiac output and total peripheral resistance are altered in different ways.

Isometric Exercise

The sprinters exhibited a greater BP response to isometric exercise than the distance runners (Graph B2a-c), consistent with earlier studies [24, 46]. The lower BP response in the distance runners was caused by the relative bradycardia which was evident across the duration of the isometric exercise bout (Graph B1). In contrast, Frisk-Holmberg et al.,[46] did not find a correlation between muscle fiber type and the HR response to isometric exercise. This might have been due to the fact that Frisk-Holmberg et al. [46] only looked at the response after one minute of exercise, a time frame that may not have been sufficient for the magnitude of the response to become evident. The present data (Graphs B1 & B2a-c) and others [24, 57] demonstrate that the HR and BP response continues to increase throughout the duration of isometric exercise at intensities that result in muscle fatigue ($\geq$ 20% MVC) [57].

There are at least two possible explanations for the exaggerated BP response in the sprinters: the increased somatic pressor reflex due to changes in muscle metabolites, and the increase in central command that causes changes in HR. Frisk-Holmberg et al.[46]

proposed that individuals with a high percentage of type II muscle fibers exhibit "exaggerated sympathetic outflow and BP responses" due to a build-up of local metabolites. This somatic pressor reflex involves the stimulation of the group III and IV afferent fibers which results in an increase in muscle sympathetic nerve activity (MSNA) and vascular resistance [58]. On the other hand, central command contributes importantly to the increase in HR, but not an increase in MSNA, as shown in studies using neuromuscular blockade [58] and in patients with McArdle's disease [59]. Therefore, the exaggerated BP response in sprinters is more likely due to the increase in central command that resulted in their elevated HR response.

There are at least two potential explanations for this increase in central command. First, the sprinters maintained a greater absolute force during the isometric exercise bout than the distance runners (19.8 ±0.7 kg vs. 16.0 ±0.9 kg). The greater force maintained by the sprinters may have required the recruitment of a greater number of type II muscle fibers [60]. Secondly, since the sprinter group had more type II muscle fibers, which fatigue more easily [60], a greater effort (central command) would be required to maintain 30% MVC. The longer time to fatigue observed in the distance runners in Fitton's study [24] lends support to this fatigue factor. Although the individual effort was not quantified in the present study, the sprinters did appear to exhibit more effort in maintaining the exercise force (unrecorded observation).

Dynamic Exercise

In the present study, there was no significant differences between the groups in the BP response to dynamic exercise set at the same relative (% $\dot{V}O_{2peak}$) work rate (Table A3). This is in contrast with the work of Fitton [24], who showed a two-fold greater change in BP in sprinters compared to distance runners at 40%, 60% and 80% $\dot{V}O_{2max}$. However, in the present study there was evidence of a difference in the hemodynamic

mechanism controlling BP. At 60% $\dot{V}O_{2peak}$, the distance runners showed a higher cardiac index and a lower SVR than the sprinter group (Graphs B5 & B6). The difference in CI clearly resulted from the distance runners having higher maximal aerobic powers, such that 60% of this value represented a greater $\dot{V}O_2$ (per kilogram of body mass). There was a greater vasodilatory response in the distance runners as evidence by the lower SVR at 60% $\dot{V}O_{2peak}$. This greater vasodilatory response was associated with the greater percentage of type I muscle fibers in the distance runners. This is supported by the work of Frisk-Holmberg et al., [23] who found that leg blood flow during exercise was associated with the percentage of type I muscle fibers ($r=0.61$), and Juhlin-Dannfelt et al., [22] who found that resting total peripheral resistance was negatively correlated with the percentage type I fibers ($r=-0.61$).

Two possibilities are offered to explain the lower SVR seen in the distance runners at 60% $\dot{V}O_{2peak}$. The first is that the distance runners may have had lower levels of metabolic acidosis in the skeletal muscle, which could have led to a diminished BP response. Although the differences in blood lactate were not significant ($p>0.08$), the trend was sprinters > control subjects > distance runners (Graph B7). The fact that this difference in SVR did not become apparent until 60% of $\dot{V}O_{2peak}$ ties in with the use of type II skeletal muscle fibers and the activation of group III and IV muscle afferents [60-63]. The increased activation of type II muscle fibers does not normally occur until after about 40% $\dot{V}O_{2max}$. This would help to explain the absence of a difference in the SVR response before this time. Another factor related to this is the shifting of the lactate threshold such that it occurs at a higher relative oxygen uptake following endurance training [64]. Without the increased recruitment of type II muscle fibers and their increased glycolytic activity, there would be no reason to suspect an increased metabolic acidosis in the skeletal muscle.

An alternate explanation for the lower SVR of the distance runners is the greater vascularity of their skeletal muscles. The greater capillary density found in the distance runners is associated with the greater percentage of type I muscle fibers [33, 40-42, 44]. So, for similar BPs at the same relative % of $\dot{V}O_{2peak}$, the distance runners would have to have a lower SVR since their $\dot{Q}$ response was greater. The greater capillary density in the distance runners in the present study would lend support to this argument. Additionally, other studies have found that endurance training increases the maximal vasodilatory capacity of the resistance vessels [65-67]. These studies suggest that the total cross sectional area of the arterioles would be greater in the distance runners. The lower systemic vascular resistance and greater capillary density of the distance runners in the present study provides support for this. Therefore, at the same level of sympathetic nervous system tone, a trained individual would be likely to have a lower vascular resistance in the skeletal muscle.

Conclusion

In conclusion, it is important to remember that isometric and dynamic exercise are two very different stressors. During isometric exercise, there is an increase in $\dot{Q}$ and no change in TPR. This adjustment in $\dot{Q}$ is mediated by an increase in HR that is driven by central command, and results in an increase in BP. The degree of central command is associated with the percentage of type II muscle fibers. The body's ability to meet the increased metabolic demands of incremental, dynamic exercise are reflected in changes in $\dot{Q}$ and TPR. The magnitude of the BP response is associated with the blood flow to the active muscles and the resistance encountered by that flow. The cardiovascular benefits of endurance training are evident in lower HRs for the same work rate. The benefits of endurance training also take place within the vasculature of the muscles. The increase in capillary density and in the maximal vasodilatory capacity of the resistance vessels provides

a beneficial adaptation to the vascular system. These central and peripheral adaptations that take place with endurance training provide an explanation for the reduced BP responses during isometric exercise, and the lower SVR response during dynamic exercise in the distance runners.

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APPENDIXES

TABLE A-1

Descriptive Characteristics

	Control	Sprinter	Distance	p-value
Age	22.7 ± 0.8	25.2 ±1.5	25.5 ±1.5	0.2760
Height (cm)	181.9 ±2.5	179.2 ±2.1	173.7 ±2.3	0.0644
Weight (kg)	83.6 ±4.7	81.3 ±1.2	64.9 ±2.5 #	0.0015
Absolute VO_2 (L/min)	3.9 ±0.16	3.8 ±0.08	4.1 ±0.16	0.3767
Relative VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	46.5 ±2.02	46.9 ±1.00	62.9 ±1.54 #	0.0001
Max. Handgrip (kg)	59.1 ±3.0	65.8 ±2.4	53.2 ±2.8 *	0.0185
30% MVC	17.7 ±0.9	19.8 ±0.7	16.0 ±0.9 *	0.0185
Sum of Skinfolds (mm)	34.7 ±4.6 #	22.2 ±2.1	18.2 ±1.1	0.0038
kg/m^2	25.2 ±1.03	25.4 ±0.63	21.5 ±0.67 #	0.0056
Body Surface Area (m^2)	2.05 ±0.07	2.00 ±0.02	1.78 ±0.04 #	0.0024
% Type I	52.1 ±3.75	46.5 ±4.41	64.8 ±6.71	0.0600
% Type IIa	36.9 ±2.61	46.5 ±5.87	31.4 ±6.32	0.1499
% Type IIb	11.0 ±2.06	7.2 ±2.80	3.3 ±1.49	0.0722
Capillary Density (capillaries/mm^2)	327.2 ±15.5	322.7 ±22.5	408.8 ±26.8 *	0.0308
Resting HR (beats/min)	60.7 ±4.6	53.0 ±2.4	52.3 ±1.6	0.1455
Resting SBP (mmHg)	119.3 ±1.7	113.5 ±2.5	117.9 ±1.5	0.1216
Resting DBP (mmHg)	71.4 ±0.9	69.6 ±1.3	72.1 ±2.2	0.5067
Resting MABP (mmHg)	87.2 ±0.7	84.1 ±1.4	87.2 ±1.5	0.1667
Max. HR (beats/min)	191.5 ±3.9	176.3 ±3.5	176.8 ±5.0	0.0355
Max. SBP (mmHg)	211.3 ±3.6	213.8 ±7.4	231.7 ±4.9	0.0399
Max. DBP (mmHg)	84.0 ±2.5	84.3 ±4.6	78.0 ±3.3	0.3923
Max. MABP (mmHg)	126.0 ±2.4	127.1 ±3.7	128.7 ±3.7	0.8476

#=significantly different from other groups

*=significantly different from sprinters

TABLE A-2

Isometric Exercise

Group		Rest	Min-1	Min-2	Min-3
Sprinter n=6	HR	53.0±1.7	74.2±4.0	80.6±5.1	85.4±6.1
	SBP	114.5±2.2	130.7±4.7	151.7±6.8	166.3±7.8
	DBP	74.8±2.1	92.3±4.0	104.3±4.0	114±3.6
	MABP	87.9±1.9	105.0±4.2	120.0±4.8	131.3±4.8
Control n=6	HR	58.3±2.0	70.0±3.7	75.0±3.2	78.2±3.0
	SBP	117.5±1.1	133.5±3.1	141.0±4.6	151.3±4.1
	DBP	70.5±3.6	84.3±1.7	99.0±2.6	109.0±2.5
	MABP	86.0±2.4	100.6±1.9	112.9±3.0	123.0±2.8
Distance n=6	HR	52.5±2.4	60.8±4.5 *	62.7±4.3 *	67.8±4.9 *
	SBP	112.3±2.1	122.0±4.1	133.7±3.8	140.7±5.4 *
	DBP	71.2±2.4	82.3±4.0	93.3±4.1	101.7±2.7 *
	MABP	84.8±2.1	95.4±3.6	106.6±3.5	114.5±3.2 *

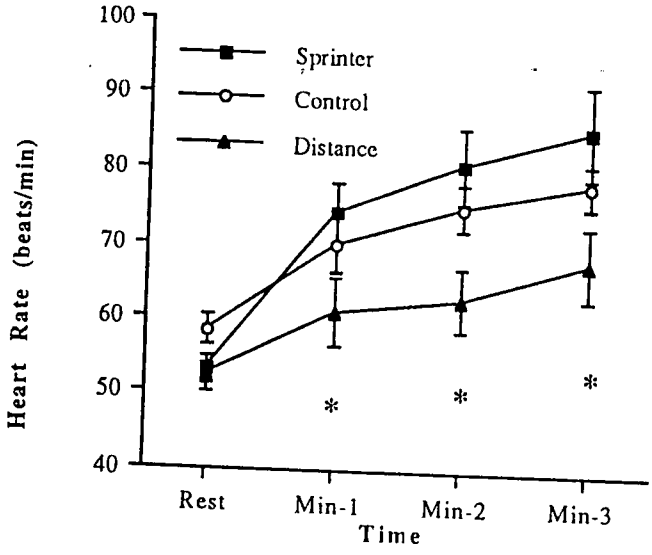
Means ± SE
* = Significantly Different from Sprinter Group

TABLE A-3

Dynamic Exercise

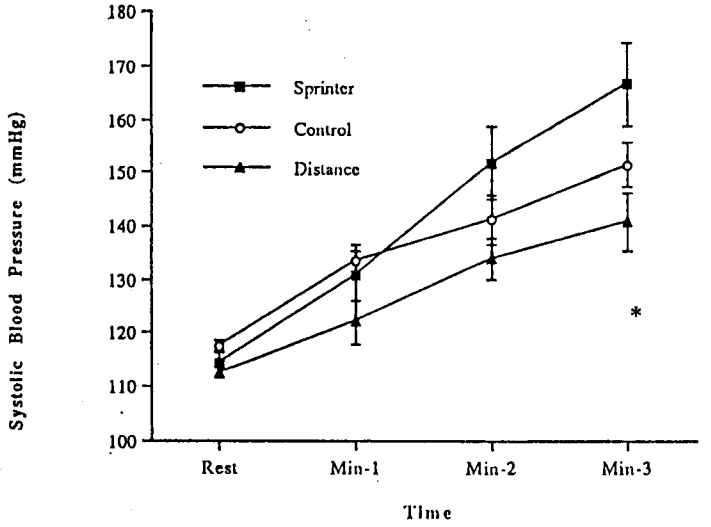
Group		Supine	Bike Rest	20%	40%	60%
Sprinter n=6	HR	51.0±3.1	64.3±3.8	81.3±2.7	108.2±4.4	139.0±6.2
	SBP	114.7±3.5	119.7±4.0	135.3±5.5	153.0±8.0	174.0±10.8
	DBP	70.7±2.3	82.3±0.6	82.7±2.2	82.0±2.1	82.7±2.3
	MABP	85.2±2.5	94.7±1.7	100.0±3.1	105.4±4.0	112.8±4.9
	CI		2.54±0.2	4.22±0.3	6.54±0.2	7.42±0.2
	SVR		38.4±3.2	24.8±2.90	16.2±1.0	15.3±0.9
	{LA}		0.73±0.04	0.89±0.07	1.31±0.11	2.76±0.29
	VO_2		0.38±0.02	0.86±0.03	1.54±0.05	2.28±0.09
Control n=6	HR	58.8±4.4	69.2±2.7	85.2±5.0	114.5±4.0	144.0±7.0
	SBP	118.8±2.2	125.3±2.0	136.7±1.5	154.3±4.5	172.7±5.6
	DBP	71.8±0.4	80.3±2.0	79.3±2.2	78.3±1.1	79.3±1.8
	MABP	87.3±0.9	95.2±1.2	98.3±1.6	103.4±1.7	110.1±2.5
	CI		2.55±0.2	4.39±0.3	6.41±0.3	7.78±0.3
	SVR		38.8±3.4	22.8±1.4	16.3±0.9	14.2±0.5
	{LA}		0.81±0.07	0.77±0.06	1.09±0.09	2.35±0.21
	VO_2		0.40±0.03	0.95±0.06	1.61±0.08	2.42±0.10
Distance n=6 $	HR	47.2±2.3	60.0±4.2	82.3±6.4	109.2±3.5	133.3±4.8
	SBP	116.3±2.1	122.3±3.0	141.7±2.9	159.3±4.1	185.3±6.4
	DBP	73.5±2.1	83.3±0.7	83.3±1.6	83.7±2.5	81.0±1.8
	MABP	87.6±1.6	96.2±1.1	102.6±1.6	108.6±2.6	115.4±3.2
	CI		2.89±0.4	4.82±0.3	7.27±0.3	9.57±0.3 #
	SVR		35.4±3.6	21.6±1.2	15.1±0.9	12.1±0.5 *
	{LA}		0.77±0.06	0.87±0.04	1.24±0.13	1.93±0.22
	VO_2		0.30±0.02	0.84±0.07	1.58±0.09	2.41±0.13

Means ± SE
* = Significantly Different from Sprinter Group
# = Significantly Different from Other Groups
$ = n=5 subjects



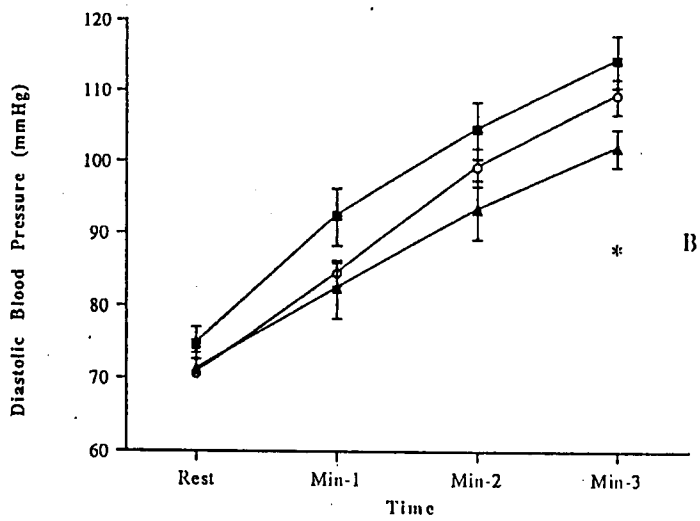
*=Sign. Diff from Sprinter group

B 1



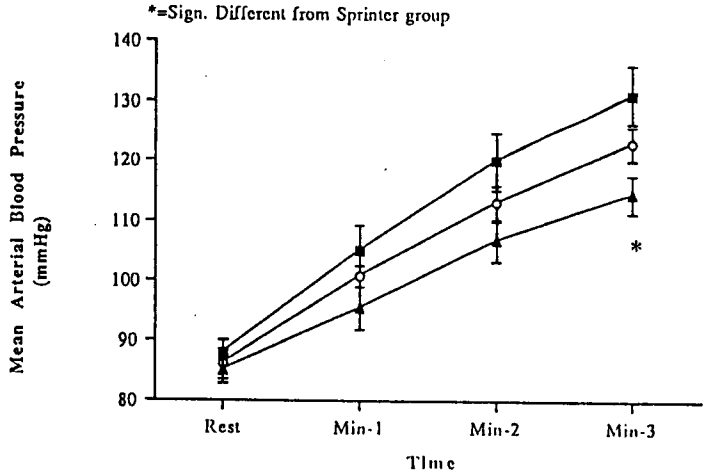
B2a

*=Sign. Different from Sprinter group



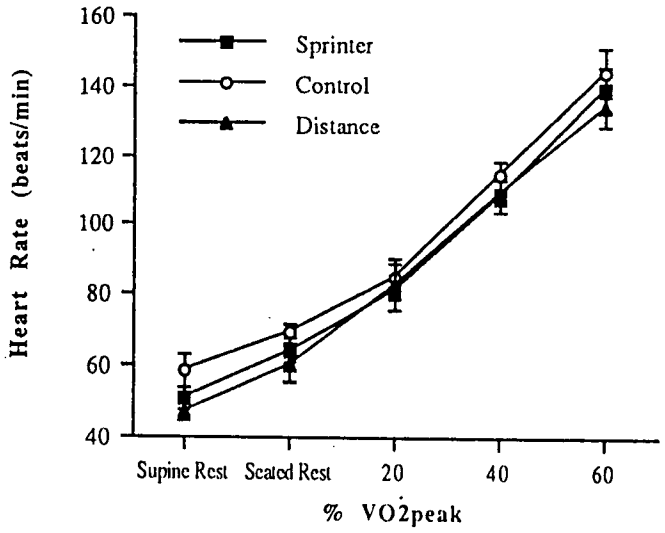
B2b

*=Sign. Different from Sprinter group

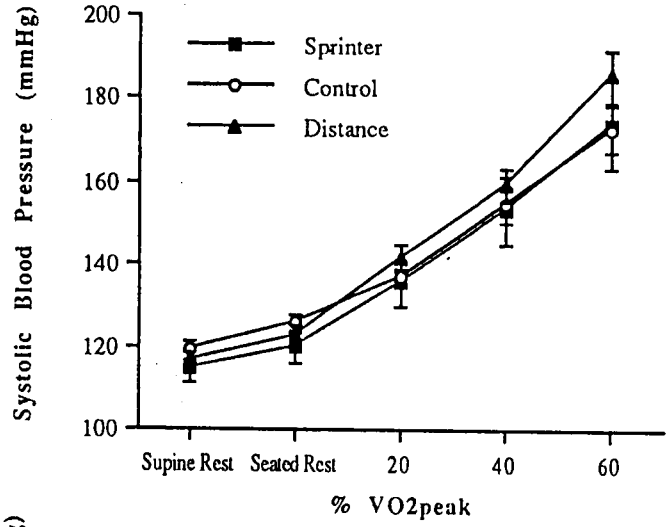


B2c

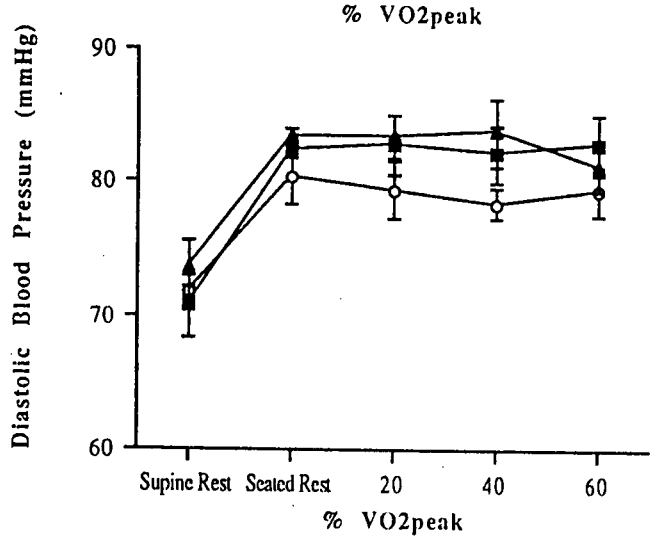
*=Sign. Different from Sprinter group


160
140
120
100
80
60
40
Heart Rate (beats/min)
Sprinter
Control
Distance
Supine Rest
Seated Rest
20
40
60
% VO2peak

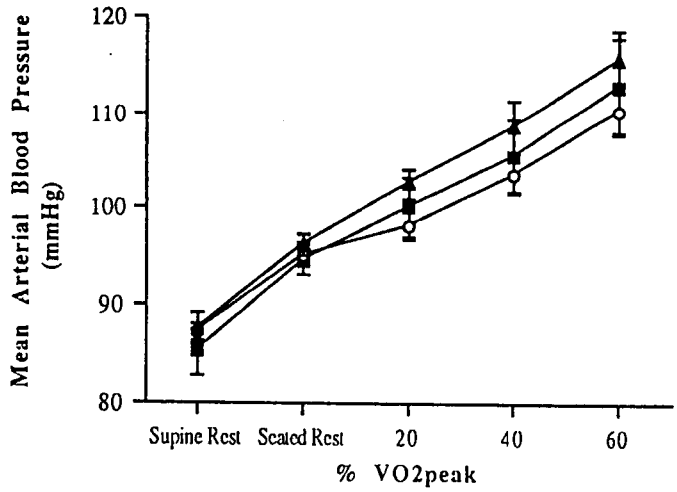
B 3


Systolic Blood Pressure (mmHg)
200
180
160
140
120
100
Sprinter
Control
Distance
Supine Rest
Seated Rest
20
40
60
% VO2peak

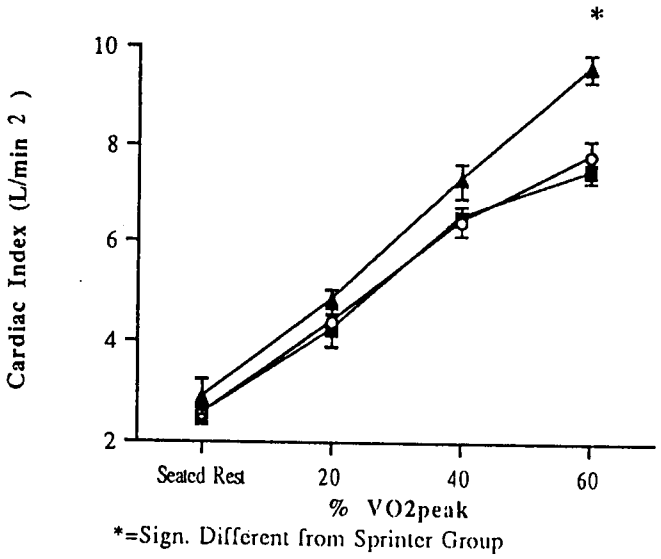
B4a


Diastolic Blood Pressure (mmHg)
90
80
70
60
Supine Rest
Seated Rest
20
40
60
% VO2peak

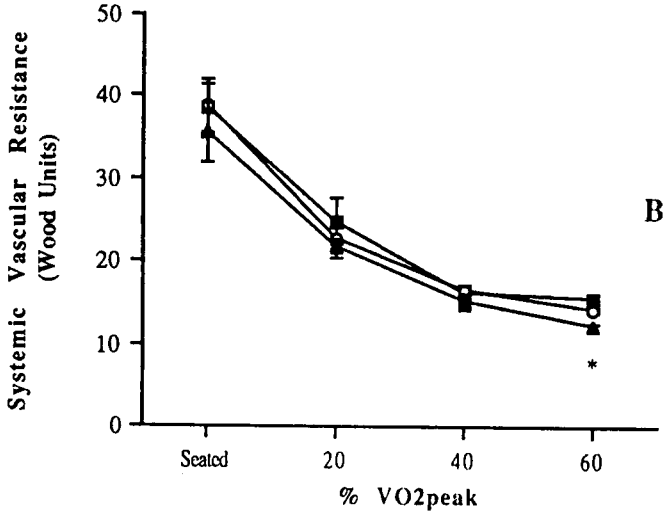
B4b


Mean Arterial Blood Pressure (mmHg)
120
110
100
90
80
Supine Rest
Seated Rest
20
40
60
% VO2peak

B4c

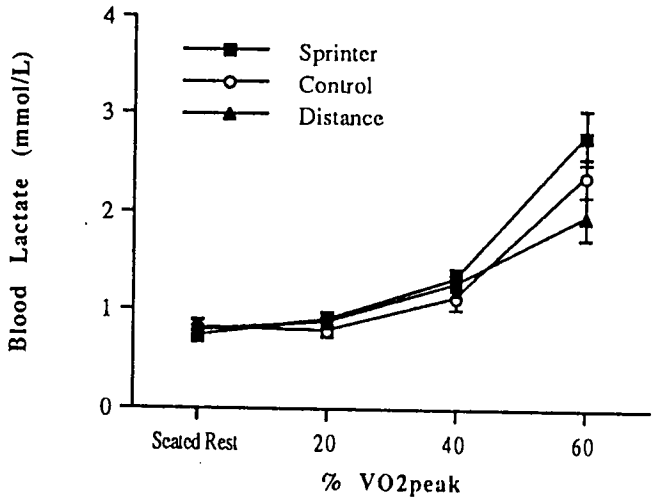

*
10
8
6
4
2
Cardiac Index (L/min 2)
Seated Rest
20
40
60
% VO2peak
*=Sign. Different from Sprinter Group

B 5



B 6

*=Sign. different from Sprinter & Control Groups


Blood Lactate (mmol/L)
4
3
2
1
0
Sprinter
Control
Distance
Seated Rest
20
40
60
% VO2peak

B 7

APPENDIX C-1

PERSONAL HISTORY FORM

Name _____ Age _____ Date _____

Past History: Have you ever had? Place a check if YES

	Yes	No	Don't Know
Free or easy bleeding (hemophilia)	( )	( )	( )
Rheumatic fever	( )	( )	( )
Heart murmur	( )	( )	( )
High blood pressure	( )	( )	( )
Any heart problems	( )	( )	( )
Irregular heart beat	( )	( )	( )
Varicose veins	( )	( )	( )
Lung disease	( )	( )	( )
Seizures	( )	( )	( )
Bronchitis	( )	( )	( )
Emphysema	( )	( )	( )
Diabetes	( )	( )	( )
Asthma	( )	( )	( )
Kidney disease	( )	( )	( )
Liver disease	( )	( )	( )
Hay fever	( )	( )	( )

Present Symptoms Review:

Have you recently had?	YES		YES
Chest pain	( )	Frequent urination	( )
Shortness of breath	( )	Blood in urine	( )
Heart palpitations	( )	Burning sensations	( )
Leg or ankle swelling	( )	Severe headache	( )
Coughing of blood	( )	Blurred vision	( )
Low blood sugar	( )	Difficulty walking	( )
Feeling faint or dizzy	( )	Weakness in arm or leg	( )
Numbness	( )		

Do you smoke? Yes No How many per Day? _____

APPENDIX C-2

FAMILY HISTORY OF HIGH BLOOD PRESSURE

NAME : _____ Date: _____

It is important for us to know if either of your parents have High Blood Pressure, because family history of high blood pressure may explain some of the individual blood pressure variation that occurs during exercise. The next time you talk to your parents, Please ask then the following questions:

1) Has your Mother ever been diagnosed as having Hypertension (High Blood Pressure) ?

Yes ____ No _____ Don't Know _____

2) Is your Mother currently taking blood pressure medication ?

Yes ____ No _____ Don't Know _____

3) Has your Father ever been diagnosed as having Hypertension (High Blood Pressure) ?

Yes ____ No _____ Don't Know _____

4) Is your Father currently taking blood pressure medication ?

Yes ____ No _____ Don't Know _____

All information will be kept confidential !

Home Address : _____

City, State Zip: _____

APPENDIX C-3

INFORMED CONSENT FORM

AMERICAN HEART ASSOCIATION:HYPERTENSION STUDY
MUSCLE FIBER COMPOSITION AND BLOOD PRESSURE RESPONSE TO EXERCISE

Address: Dept. of Human Performance and Sport Studies
College of Education
University of Tennessee-Knoxville
1914 Andy Holt Avenue/ Knoxville, TN 37996
(615) 974-5111 or 974-5091

You are invited to participate in a research study, the purpose of which is to examine the role of muscle fiber composition in the control of blood pressure during exercise. The findings are expected to influence our understanding of the mechanisms that control blood pressure. A description of the procedures that you will undergo are listed below:

EXERCISE BOUTS:

You will undergo a maximal bicycle test for the determination of maximal oxygen uptake, which will be measured using a computerized system. A small sample of blood (50 μL-about a drop) will be taken from a finger stick for the determination of the lactic acid level. Blood samples will be collected on each of the test days (screening, isometric, maximal oxygen uptake, and at each level during the submaximal exercise session). You will perform a maximal handgrip strength test for the determination of maximal handgrip strength. Then you will perform an isometric contraction of 30% of your maximal handgrip strength. On another day, you will report to the laboratory following an overnight fast. After 20 minutes of supine (lying) rest, a series measurements will be obtained. You will then perform cycle exercise at relative intensities of 20, 40, and 60% of your maximum (6 minutes at each stage). During this cycle exercise, your cardiac output and blood pressure will be measured.

NEEDLE BIOPSY:

On a separate day you will report to the laboratory for a needle biopsy procedure. This involves taking 2-3 small samples of muscle from your thigh, each one the size of a small pea. the skin will be shaved and cleaned thoroughly. After injecting a local anesthetic to cause numbness, a small incision (1 cm) will be made. A biopsy needle will be inserted into the muscle and the tissue sample removed. Afterwards, a butterfly bandaid will pull the incision together and a sterile bandage will be applied. You may experience a momentary sensation of pressure or pain when the sample is taken, followed by a dull ache for one to two days following the procedure. Care should be taken to keep the area clean and dry for several days, until the skin heals. You should refrain from exercise during this time and return to the laboratory 3-5 days after the muscle biopsy to be evaluated for any signs of infection.

The risks involved with exercise include abnormal blood pressure responses, fainting, musculoskeletal injury, disorders of the heart, and in very rare instances heart attack. Additional risks for the muscle biopsy procedure include infection and internal bleeding. However, there is only a minor chance that serious complications will occur. In the event that physical injury occurs as a result of the study, the University of Tennessee does not automatically provide reimbursement for medical care or other compensation. If physical injury is suffered in the course of research, or for more information, please notify the investigators in charge, Dr. David Bassett, Jr. or Dr. Edward Howley, at 974-5111. Benefits to be expected include financial payment ($100.00) and an assessment of your fitness. Your total time involvement would be about one hour/day on 5 separate days.

The information in this study will be provided to you. Data from this study may be used in research reports or presentations; however, your identity will not be disclosed.

You are free to decide whether or not to participate in this study and are free to withdraw from the study at any time.

Before you sign this form, please ask questions about any aspects of the study which are unclear to you.

Authorization: I, _____, have read the above and decided to participate in the research project described above. My signature also indicates that I have received a copy of this consent form.

_____Participant's signature Date: _____

_____Witness Date : _____

APPENDIX D-1

Procedure for the Determination of Muscle Fiber Type Myosin ATPase Stain

REAGENT PREPARATION:

Alkaline Stock Solution:
Add 2.253 g glycine, 2.40 g $CaCl_2$, 1.755 g NaCl, 1.08 g NaOH and bring to a final volume of 400 mL with distilled water. Adjust pH to 9.4 (make fresh).

Alkaline Preincubation Solution:
Take 135 mL of the alkaline stock solution and readjust the pH to 10.3 using 1N NaOH or 1N HCL as needed

Acid Preincubation Stock Solution:
Add 1.94 g NaOAc, 2.94 g sodium barbital (controlled substance) to 100 mL of distilled water. This is a 0.2 M stock barbital acetate buffer. Store in freezer.

Acid Preincubation Solution:
Add 31.25 mL acid preincubation stock, 62.5 mL of 0.1 M HCL, and 50 mL of distilled water in three different Columbia jars. Adjust each to its respective pH (4.3, 4.54, 4.6).

Incubation Solution:
Add 200 mg ATP to 135 mL of the alkaline stock solution and adjust to pH 9.4 with 1N HCL or 1N NaOH as needed. Be sure to label this solution as the Acid incubation. Add 20 mg ATP to 135 mL of the alkaline stock solution and adjust to pH 9.4 as before. Label this solution as the Alkaline incubation.

PROCEDURE:

1) Preincubation

Alkaline preincubation: Incubate labeled slides in the alkaline preincubation solution for fifteen minutes. Agitate the Columbia jar slightly during this time period. The alkaline preincubation solution should be at room temperature before the slides are put in for incubation.

Acid preincubation:Incubate labeled slides in the acid preincubation solution for five minutes. Agitate the Columbia jar slightly during this time period. The acid preincubation solution should be at room temperature before the slides are put in for incubation.

2) Incubation

a) Rinse the preincubation solution thoroughly from the slides with distilled water. (rinse twice)
b) Incubate the labeled slides in their respective incubation solution in the shaker bath for 45 minutes at 37° C.
c) Rinse the slides twice with distilled water after removing them from the shaker bath.
d) Incubate in 1% $CaCl_2$ for three minutes.

e) Rinse the slides twice with distilled water after removing them.
f) Incubate in 2% $CoCl_2$ for three minutes.
g) Rinse the slides twice with distilled water after removing them.
h) Incubate in 1% $(NH_4)_2S$ for one minute
i) Rinse the slides three times with distilled water after removing and allow them to dry.
j) Do not allow the slides to sit exposed to light for prolonged periods of time. Mount coverslips to slides with Paramount or Pro-texx. Store slides in darkness as exposure to light will induce fading of the stain.

APPENDIX D-2

Procedure for the Determination of Capillary Density

REAGENT PREPARATION:

SCHIFF'S REAGENT:

a) Place 1.25 g Paraosaniline (Sigma p-3750) in 200 mL of boiling distilled water and dissolve. Boil for four to five minutes.
b) Cool to 50° C and filter
c) Add 20 mL of 1 M HCL
d) Cool to room temperature and add 0.5 g sodium metabisulphite. Place in a refrigerator overnight and keep out of the light.
e) Add activated charcoal and shake for two minutes. Then filter.
f) Add a further 5 mL of 1 M HCL and 0.25 g sodium metabisulphite and store refrigerated and in the dark until ready to use. Final solution should be colorless.

CARNOY'S FIXATIVE:

Mix in the following ratio: 6 : 3: 1-Ethanol:Chloroform: Glacial Acetic Acid

1% PERIODIC ACID

To every 10 mL of distilled water add 0.1 g of Periodic Acid (Sigma A-7875). Make fresh.

0.5% AMYLASE

To every 10 mL of distilled water add 0.05 g Amylase (Sigma A-3176). Warm up to 37° C in a water bath 15 minutes before use.

STAINING PROCEDURE:

a) Place slides with muscle section in Carnoy's fixative for 10 minutes at room temperature.
b) Rinse three times with distilled water
c) Place slides in 0.5% Amylase solution in shaking water bath for 5 minutes at 37° C
d) Rinse three times with distilled water
e) Place slides in 1% Periodic Acid for 10 minutes
f) Rinse three times with distilled water
g) Place in Schiff's reagent for fifteen minutes (bring Schiff's reagent to room temperature before using).
h) Rinse in Tap water for 15 minutes.
i) Air dry!! Don't use ascending alcohols.
j) Mount coverslip on slide with paramount.

APPENDIX E

Listing of Abbreviations

ANOVA	Analysis of Variance
BMI	Body Mass Index
BP	Blood Pressure
ΔBP	Change in Blood Pressure (peak blood pressure- resting blood pressure)
BSA	Body Surface Area
CAFT	Candian Aerobic Fitness Test
CHEC	Community Hypertension Evaluation Clinic
CI	Cardiac Index
CO_2	Carbon Dioxide
DBP	Diastolic Blood Pressure
ECG	Electrocardiogram
EMG	Electromyographic Activity
FT	Fast-twitch or Type II Muscle Fibers
HDFP	Hypertension Detection Follow-up Program
HR	Heart Rate
HT	Hypertensive
MABP	Mean Arterial Blood Pressure
MSNA	Muscle Sympathetic Nerve Activity
MVC	Maximal Voluntary Contraction
NHANES I	National Health and Nutrition Examination Survey I
NHEFS	National Health Epidemiologic Follow-up Study
NT	Normotensive
$\dot{Q}$	Cardiac Output
QRS	ECG tracing representing the Depolarization of the Heart
RFLPs	Restriction Fragment Length Polymorphisms
SBP	Systolic Blood Pressure
SDH	Succinate Dehydrogenase
ST	Slow-twitch or Type I Muscle Fibers
SVR	Systemic Vascular Resistance [$mmHg \cdot L^{-1} \cdot min^{-1} \cdot m^{-2}$]
TPR	Total Peripheral Resistance
$\dot{V}O_{2max}$	Maximal Oxygen Uptake
$\dot{V}O_{2peak}$	Peak Oxygen Uptake

VITA

Donald Joseph Torok was born in Buffalo, New York on January 10, 1951. He attended elementary school in the city of Buffalo and graduated from Clarence Central High School in June, 1969. After a year of chemical engineering at the University of Cincinnati, he transferred to the State University of New York at Geneseo and received his Bachelor of Science degree in Sociology in May, 1973. In December of 1976, he received his Elementary Education Certification from the State University of New York at Geneseo. In September of 1977 he started teaching 4th and 5th grade at York Central School in Retsof, New York. In the fall of 1980 he became the assistant cross country and track coach at the State University of New York at Geneseo. In the fall of 1982 he became the head coach of cross country and track and part-time instructor at the State University of New York at Geneseo. After ten years of teaching at York Central, he entered Miami University in Oxford, Ohio to pursue a degree in Exercise Science. He received a degree of Master of Science in Exercise Science from Miami University in August of 1989. That same August, he entered The University of Tennessee, Knoxville and in December of 1992 received a Doctor of Philosophy degree in Exercise Physiology.

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