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THE EFFECT OF MUSCLE MASS AND INTENSITY ON BLOOD PRESSURE RESPONSE TO ISOMETRIC EXERCISES IN NORMOTENSIVE MALES

by

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Finally, recognition is extended to my best friend Jesus Christ for always being with me, just like He promised.

to mom and dad

CONTENTS

I

ABSTRACT

The purpose of this study was to determine the influence of muscle mass and exercise intensity on blood pressure response to isometric contraction. Twenty male subjects (mean age=27 yrs) performed one-arm handgrip (HG), one-leg extension (LE) and combined one-arm handgrip plus one-leg extension (COMB) in a random order of exercise for one minute at 30% and 40% of maximal voluntary contraction (MVC). Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and heart rate (HR) were recorded at rest and continually throughout the static contraction exercises using a Finapres 2300 BP monitor. Data were analyzed by ANOVA and linear regression coefficient analysis. The results indicated that the blood pressure responses were significantly higher ($p<0.05$) where COMB>HG and COMB>LE at both 30% and 40% MVC. However, for each mode of static exercise the hemodynamic responses at 40% MVC were significantly higher than the hemodynamic responses at 30% MVC. There were no significant differences in blood pressure responses between HG and LE within each intensity, except for SBP which was significantly greater at 40% MVC than at 30% MVC. HR response increased progressively with increasing size of muscle mass, (COMB>LE>HG) at both intensities. The HR responses for the three modes of contraction at 40% MVC were significantly higher than at 30% MVC. There was no significant difference in HR between rest and HG at 30% MVC. Analysis of the regression coefficient (slope) for SBP showed a significantly greater pressor response, where COMB>LE>HG at both 40% and 30% MVC. Likewise, DBP indicated similar results except that HG and LE were not significantly different at 40% MVC. These results indicate that the blood pressure response to static exercise can be attributed in part to the size of the muscle mass being recruited (ie. COMB) within the same intensity of MVC, and in part to the relative intensity at which the muscle is being contracted.

INTRODUCTION

Muscular exercise is accompanied by important changes in cardiovascular function which lead to an increase in blood flow to the working muscles (Perez-Gonzalez, 1981). Isometric exercise is physiologically characterized by a dramatic increase in systolic and diastolic blood pressure (pressor response) on the cardiovascular system (Nagle et al., 1988). Heart rate also increases during isometric exercise, although its influence is not as pronounced (Lind et al., 1964). The exact nature of the mechanisms underlying the pressor response during static muscular activity are not fully understood.

Since the human cardiovascular response during isometric exercise is attributed to both peripheral and central stimuli, a number of studies have been conducted to determine whether the reflex from the working muscle is related to the muscle mass involved in the contraction or the percentage of maximal tension achieved. Studies concerning the effect of muscle mass as opposed to the intensity generated have produced inconsistent results. Riendl et al. (1977) showed that muscle mass does not influence the cardiovascular response during isometric contraction of large and small muscle groups, but that intensity of exercise is the contributing factor influencing this hemodynamic response. A comparative investigation (Lind and McNicol, 1967) has provided similar findings although two more recent studies have indicated that the cardiovascular response during isometric exercise was dependent primarily upon the muscle mass utilized. Nagle et al. (1988) indicated a progressively greater blood pressure and heart rate response in healthy men as muscle mass increased when performing handgrip, left and right leg extension and dead lift at the same percentage of maximal voluntary contraction. Likewise, Misner et al. (1990) concluded that both pressor response and increased heart rate were affected by the size of the muscle mass in men and women. The majority of these studies were limited by the use of a sphygmomanometer and stethoscope to monitor blood pressure and heart rate. The inability to measure blood pressure response continuously restricts a full understanding of the exact response.

As such, there is a discrepancy in the literature pertaining to the role of muscle mass and exercise intensity on cardiovascular response to isometric stress. Therefore, the purpose of this study was to determine the influence of muscle mass and intensity on blood pressure response using continuous monitoring during isometric exercise.

METHODOLOGY

Subjects

Twenty moderately active male subjects ranging in age from 24 to 32 years participated in this study. All subjects provided informed written consent to participate prior to testing. All subjects met the following criteria: did not suffer from a physical ailment which could alter their performance, were non-smokers, had a resting blood pressure less than 140/90 mm Hg and exercise blood pressure less than 170/100 mm Hg following the initial stage of the Canadian Aerobic Fitness Test (CAFT) (Jetté et al., 1991), answered "no" to all questions on a modified PAR-Q and were not taking medication that would alter their cardiovascular response or physical performance. The mean age and physical characteristics of the subjects are shown in Table 1. The results showed a mean age 27$\pm$2.6 yrs, mean height 178$\pm$6.9 cm, mean weight 77$\pm$11.4 kg and BMI 24$\pm$3.2 kg/m^2.

Materials

The one-arm handgrip was performed on a dynamometer constructed from an air pressure gauge (H.O. Trerice Co., Detroit Michigan) connected by surgical tubing to a rubber squeeze bulb. The one-leg extension was performed using the Kin-Com 500H Isokinetic apparatus (Chattecx Corp.; Chattanooga, TN). Cardiovascular response was recorded continually throughout the isometric contractions using a Finapres 2300 BP monitor finger cuff (Ohmeda; Louisville, CO).

Study Protocol

The testing protocol was conducted on two consecutive days. At the initial testing session, subjects were thoroughly briefed as to the nature of the study and their involvement. Subjects were requested to sign an informed consent and complete a modified PAR-Q and a health questionnaire (Jetté et al., 1990) prior to participation. The subjects were then seated for a five minute period following which time resting heart rate and blood pressure were recorded using a calibrated aneroid sphygmomanometer and stethoscope. The subjects were then screened to ensure that they did not demonstrate an exaggerated blood pressure response to exercise by completing the initial stage of the CAFT (Jetté, Landry and Sidney, 1991). Participants between the ages of 24 and 29 years started at stage 5, while those between 30 and 32 years started at stage 4. Subjects walked up and down a two-step staircase (20.3 cm) to a six-count recorded musical rhythm for three minutes at a rate equivalent to 65 to 70% of the average aerobic power anticipated of a person 10 years older than the subject. At the completion of the 3-minute exercise, blood pressure was determined immediately following the 10-second post-exercise heart rate measurement {ie. between the 15^{th} and 45^{th} second of post-exercise}(Jetté, Landry and Sidney, 1991). An exaggerated blood pressure response was defined as the subject's systolic blood pressure exceeded 170 mmHg or showed a difference of 45 mmHg from the initial stage of the CAFT. If the subject's heart rate did not exceed a predetermined standard following the initial stage and no adverse signs and symptoms were observed or reported, the participant was allowed to proceed to the second and third stages of the CAFT. Predicted maximum aerobic capacity was calculated using the procedure of Jetté et al. (1976).

Following a twenty minute rest, subjects were then instructed on how to properly perform the one-arm handgrip on the Trerice dynamometer using their left hand and the one-leg extension exercise on the Kin-Com 500H Isokinetic apparatus using their dominant leg, while wearing the Finapres BP monitor (Hartmann and Bassenge, 1989). The Finapres BP monitor finger cuff was fitted on the subject's right hand on the second digit. The arm was supported by a sling to maintain finger and cuff at the level of the heart. They were further instructed to inhale and exhale during isometric exercises so as not to perform a Valsalva manoeuvre. MVC for handgrip and leg

extension exercises was determined from the highest of three repeated maximal contractions for each exercise. The duration of each maximal repetition was five seconds with a two minute rest between each contraction.

Day two served as the actual testing session for data collection to determine the influence of muscle mass and intensity on blood pressure response to isometric exercise. During the examination phase, subjects were seated and strapped on the Kin-Com apparatus. Following a five-minute period of rest, heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were measured for one minute using the Finapres BP monitor. Subjects then performed one-arm handgrip, one-leg extension and combined one-arm handgrip and one-leg extension tasks at 30% of their predetermined MVC for one minute. Each of the three tasks were performed once with a five-minute rest period between each exercise. The subjects were then provided with a ten minute rest period before being asked to perform the same three isometric tasks at 40% MVC. The order of exercises was randomized, where each subject performed the three selected isometric exercises in a different order at 30% and 40% MVC. The order was also randomized periodically betweeen subjects to control for an order effect.

Statistical Analysis

Descriptive analysis of the participants included the following means: age, height, weight, resting heart rate, resting blood pressures, predicted VO_2max, maximum handgrip strength and maximum leg extension strength. The hemodynamic response to the three muscle masses during isometric exercise were compared at two different intensities. A 3X2 factorial design analysis of variance (ANOVA) with repeated measures was conducted on the mean values for the one-minute exercise response to determine if there were any significant differences in heart rate and blood pressures with respect to the three muscle masses and two levels of intensity to the isometric stressors. In the presence of a significant F-ratio, Least Square Means technique was administered for post-hoc analysis in order to localize significant differences between the means. An alpha level of 0.05 was used as a standard for statistical significance.

Linear regression analysis was calculated for the dependent variables systolic and diastolic blood pressure for each of the three isometric exercises at 30% and 40% MVC to determine the slope of the pressor responses. The slope for each of the contractions was calculated from the mean blood pressure responses during the one minute of exercise for each subject using comparison of regression coefficients to determine if their were any significant differences between the pressor responses for the three respective isometric exercises at both 30% and 40% MVC (Edwards, 1984). An alpha level of 0.05 was used as a standard for statistical significance.

Results

Twenty healthy normotensive males participated in this study. The mean predicted VO_2max suggested a relatively fit group of subjects (mean=51$\pm$5.8 ml/kg/min). Mean values for the maximum one-arm handgrip and the maximum one-leg extension were 97$\pm$13.6 kPa and 592$\pm$109.9 N, respectively (Table 1).

The mean responses for heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) at rest and for each of the one minute of isometric contractions (HG, LE and COMB) at both 30% and 40% MVC are shown in Table 2. These responses, which were recorded every two seconds starting five seconds prior to exercise throughout the isometric contraction and fifteen seconds post-exercise, are graphically presented in Figures 1-4. The results indicated that SBP, DBP, MAP and HR values were significantly greater ($p<0.05$) where COMB>HG and COMB>LE at both 30% and 40% MVC. There were no significant differences in the DBP and MAP responses between HG and LE at both 30% and 40% MVC. However, there was a significant difference between HG and LE for SBP at 30% and 40% MVC. HR increased progressively with size of muscle mass, (COMB>LE>HG) regardless of static intensity. HR responses at 40% MVC were consistently higher than at 30% MVC for the same muscle mass. There was no significant difference in HR between rest (73$\pm$9.5 bpm) and HG (74$\pm$9 bpm) at 30% MVC. Furthermore, analysis of variance indicated non significant interaction effects of the static contractions between 30% and 40% MVC for all cardiovascular responses. There was no significant difference in SBP between COMB at 30% MVC (143$\pm$12 mmHg) and LE

at 40% MVC (143±11.6 mmHg). Similarly, DBP response for COMB at 30% MVC (88±10.5 mmHg) was not significantly different from HG (89±10.6 mmHg) and LE (90±11 mmHg) at 40% MVC. MAP response for COMB at 30% MVC (103±10.5 mmHg) was not significantly different from HG (103±10 mmHg) and LE (104±10.5 mmHg) at 40% MVC. Finally, no significant differences in HR were found between LE at 30% MVC (79±7.9 bpm) and HG at 40% MVC (80±10.1 bpm) as well as COMB at 30% MVC (82±8.9 bpm) and LE at 40% MVC (84±10.4 bpm).

Table 3 shows the results of the regression coefficient analyses on the slope of the SBP and DBP responses during the continuous one-minute contraction for the mean of the twenty subjects at both 30% and 40% MVC. The regression analysis showed a strong linear trend in SBP and DBP responses regardless of intensity of exercise or muscle mass recruited. The range of r-squared values for SBP and DBP were 0.87 to 0.98 and 0.95 to 0.99, respectively. Linear regression coefficient analysis for SBP indicated a significant pressor response ($p<0.05$), where COMB>LE>HG at both 30% and 40% MVC. The SBP slopes for HG, LE and COMB were 0.23, 0.32 and 0.52 at 30% MVC and 0.37, 0.48 and 0.72 at 40% MVC. Likewise, DBP showed similar results (COMB>LE>HG) at 30% and 40% MVC; however, no significant difference was found between HG and LE at 40% MVC. The DBP slopes for HG, LE and COMB were 0.19, 0.22 and 0.37 at 30% MVC and 0.35, 0.36 and 0.54 at 40% MVC.

Discussion

The purpose of this study was to determine the effect of muscle mass and intensity of exercise on the blood pressure response to isometric exercises in normotensive males. A number of studies have provided controversial results as to the effect of muscle mass in elevating blood pressure (pressor response) during isometric contraction. Lind and McNicol (1967) suggested that the magnitude of the increase in blood pressure during sustained contraction of the arms and legs in four healthy men was associated with the tension developed within the muscle, and was unrelated to the muscle mass. Their results indicated that when two or more muscle groups contracted isometrically at the same relative tension, the increase in blood pressure was the same, whether

they were contracted separately or combined. Neither the muscle mass recruited, nor the absolute tension generated had any bearing on the cardiovascular response. Conversely, McCloskey and Streatfield (1975) indicated from their study regarding blood pressure response during static contraction of the finger and hand in male and female subjects that muscle mass was a contributing factor in the pressor response. Their results showed that blood pressure was greater following handgrip contraction than after finger contraction. Likewise, Mitchell et al. (1981) confirmed that static contraction at 40% MVC elicited progressive elevations in blood pressure when the muscle mass was increased. Also, following blood circulatory occlusion in the muscle following contraction, the blood pressure again increased linearly with a larger contribution of muscle mass.

The cardiovascular responses to submaximal isometric exercise displayed by the subjects in this study were similar to more recent investigations (Misner et al., 1990; Seals, 1989 and Mitchell et al., 1980). Mean values for SBP, DBP, MAP and HR were progressively greater from HG to LE to COMB at 30% and 40% MVC, except the SBP response for HG and LE at 30% MVC which indicated a flat response. Also, all cardiovascular responses were higher at 40% MVC than at 30% MVC for the same exercise. Analysis of the regression coefficient (slopes) for SBP versus time and DBP versus time indicated significant pressor responses. At both 30% and 40% MVC, COMB>LE>HG, except that there was no significant difference in DBP for HG and LE at 40% MVC. Nevertheless, changes in blood pressure and heart rate responses during the three modes of isometric contraction appear to be closely related to the size of the muscle mass as well as the relative tension generated. This is consistent with previous studies which attribute the progressive increases in cardiovascular response during static exercise to both central and peripheral mechanisms (Shepherd et al., 1981; Perez-Gonzalez, 1981; Mitchell et al., 1981). Central command is influential in increasing the sympathetic outflow during static contraction or by stimulating the appropriate amount of motor neurons within the active muscle. Peripheral contribution is effective in providing feedback as to the tension being generated on the isometrically contracted muscle fibres and ergoreceptors detecting elevations in metabolic by-products as the percentage of MVC increases.

The extent to which both central and peripheral mechanisms regulate the pressor response to isometric exercise of various muscle mass is unclear (Seal et al., 1983). The central contribution associated with cortical irradiation is responsible during the initial stages of isometric exercise in recruiting the appropriate number of motor units from different muscle groups. It has been suggested that the number of motor neurons activated to produce a given force with arm muscles may be larger than when using the leg muscles, since the number of muscle fibres in a motor unit is less in the arms than in the legs (Feinstein et al., 1955). This could provide an explanation as to why there was no significant difference between HG and LE at either 30% or 40% MVC for DBP and MAP responses. Even though the size of the handgrip and leg extension muscle masses are distinctly different, central control mechanisms may have recruited a larger number of motor neurons from the forearm in comparison to that of the leg. If this was the case, increases in blood pressure during static handgrip contraction could have been attributed to a greater central influence when compared to the pressor response during static leg extension. The peripheral contribution within the exercising muscle in increasing blood pressure during static contraction is associated with receptors that are activated by metabolic by-products produced during static exercise. The by-products stimulate metabolic and chemical ergoreceptors in the active muscle which cause a peripheral reflex transferring information back to the central cortex (Mitchell et al., 1981). Likewise, muscle afferent fibres (mechanoreceptors III and IV) whose function is to induce cardiovascular adjustments as well as transmit information from the periphery to the spinal cord have been associated with isometric exercise (Shepherd et al., 1981). Peripheral mechanisms may very well have been involved in the blood pressure responses which were observed in this study. The progressive increases in blood pressure and heart rate could also be attributed to muscle afferents and metabolic receptors within the statically contracted muscle. Their contribution would also be further enhanced as muscle mass and intensity increase (Mitchell et al., 1981).

Previous studies have been limited in their ability to objectively monitor the cardiovascular response during isometric contractions by using a stethoscope and sphygmomanometer. In this investigation, the Finapres BP monitor made it possible to measure cardiovascular responses continually throughout static exercise. Regression analysis on the changes of SBP and DBP responses as a function of time indicated a strong linear trend at both 30% and 40% MVC.

Observation of Figures 1 and 2 suggest that SBP and DBP increase linearly throughout exercise, until cessation of contraction at which time the blood pressure returns to near resting values within fifteen seconds. The figures demonstrate that there was no "plateau" during one minute of static exercise for any mode of contraction or at either intensity. Perhaps the duration of contraction in this study was not long enough or intensity of exercise large enough to produce a plateau in blood pressure. Lind et al. (1964) performed handgrip contractions at intensities greater than 15% MVC and observed increases in heart rate, systolic and diastolic pressure and cardiac output. They concluded that the increased magnitude of these hemodynamic responses is determined by the intensity of contraction and its duration, but they did not report any blood pressure plateau. Although the subjects in this study performed at adequate intensities (ie. 30 and 40%) in producing a significant pressor response isometric contraction for a longer duration, perhaps to fatigue, possibly would have elicited a plateau in blood pressure. Further investigation would appear necessary to study the effect of longer durations of isometric contraction at various intensities to determine if a plateau in blood pressure would occur.

The present findings demonstrated that the pressor response to isometric contraction was dependent upon size of muscle mass and relative tension developed. Mitchell et al. (1981) suggested that blood pressure increases with a greater muscle mass due to central and peripheral involvement. Increased central drive would elicit a greater number of motor units, while a larger muscle mass would evoke more peripheral contribution from active muscle afferent nerve endings and metabolic ergoreceptors. It was suggested that the number of motor units recruited in the forearm for HG contractions is greater than that of the muscles used for leg extension. This would explain the similar changes in DBP and MAP responses between HG and LE, but not evident when compared to COMB. Therefore, cardiovascular responses to isometric exercise were directly related to muscle mass, but were moderated by the number of motor neurons recruited within the contracting muscle. The pressor response to isometric contraction followed an increasing linear trend for all muscle masses and intensities. In conclusion, the results of this study indicated that muscle mass and intensity of exercise are contributing factors in the pressor response to isometric contraction in normotensive males.

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TABLE 1: Group means for demographics, anthropometrics and exercise variables

Variables	Age (yrs)	Height (cm)	Weight (kg)	BMI (kg/m^2)	VO_2 Max (ml/kg/min)	Max HG (kPa)	Max LE (N)
X	27	178	77	24	51	97	592
SD	2.6	6.9	11.4	3.2	5.8	13.6	109.9

TABLE 2: Group means for variables during rest and exercise at 30% and 40% MVC

Variables		Rest	30% MVC HG	30% MVC LE	30% MVC COMB	40% MVC HG	40% MVC LE	40% MVC COMB
SBP *	X	124	134	133	143	139	143	151
	SD	8.7	11.5	10.2	12.0	11.9	11.6	13.2
DBP †	X	77	83	84	88	89	90	96
	SD	9.5	11.1	10.0	10.5	10.6	11.0	11.6
MAP †	X	90	96	98	103	103	104	112
	SD	8.8	10.4	9.0	10.5	10.0	10.5	12.5
HR ††	X	73	74	79	82	80	84	87
	SD	9.5	9.0	7.9	8.9	10.1	10.4	10.8

Note:

* Significant differences found between all variables at $p<0.05$, except: 30% HG vs 30% LE and 30% COMB vs 40% LE.

† Significant differences found between all variables at $p<0.05$, except: 30% HG vs 30% LE, 30% COMB vs 40% HG, 30% COMB vs 40% LE and 40% HG vs 40% LE.

†† Significant differences found between all variables at $p<0.05$, except: 30% HG vs REST, 30% LE vs 40% HG and 30% COMB vs 40% LE.

TABLE 3: Group means for linear regression slope of the SBP and DBP with respect to muscle mass and intensity

Exercises	Slope	Point of Origin	Linear Trend(r^2)
SBP (30% MVC) †			
HG	0.23	127.35	0.87
LE	0.32	124.46	0.98
COMB	0.52	127.45	0.97
SBP (40% MVC) †			
HG	0.37	127.71	0.97
LE	0.48	129.16	0.97
COMB	0.72	129.63	0.96
DBP (30% MVC) †			
HG	0.19	77.11	0.96
LE	0.22	77.74	0.95
COMB	0.37	77.52	0.97
DBP (40% MVC) *			
HG	0.35	79.24	0.99
LE	0.36	78.99	0.97
COMB	0.54	80.68	0.97

Note:

† Significant difference found at the $p<0.05$ level of significance.

* No significant difference found at the $p<0.05$ level of significance between HG and LE at 40% MVC.

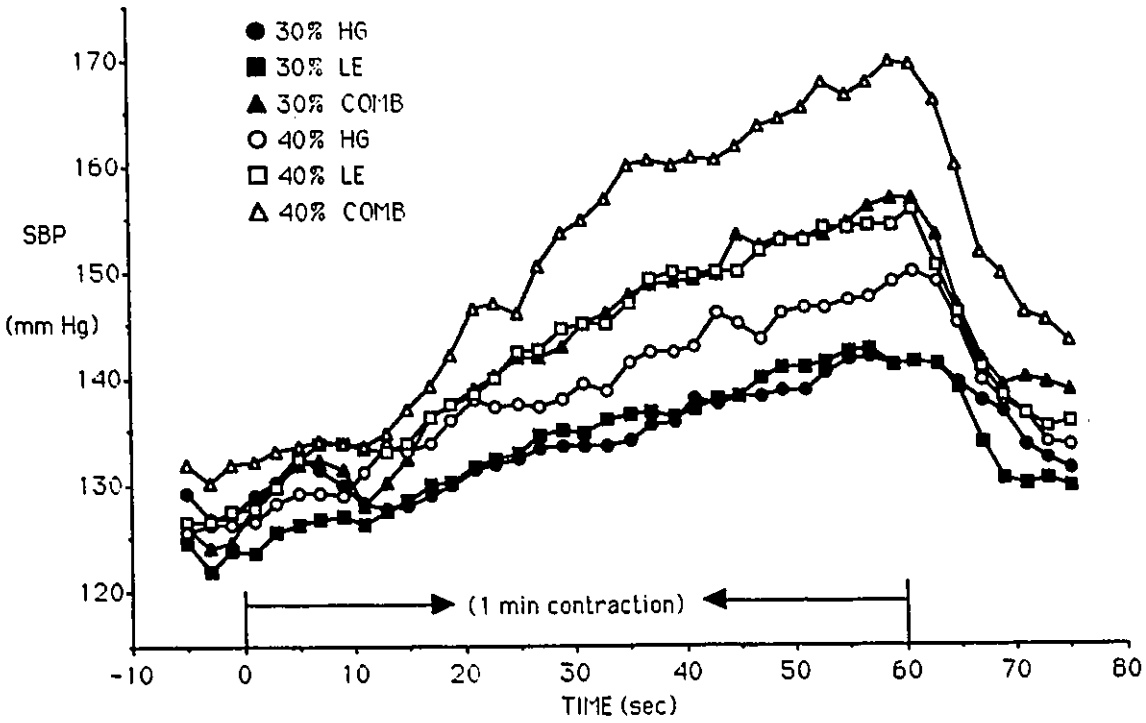

Figure 1: Continuous SBP response (2 sec interval) for isometric contractions (HG, LE and COMB) at 30% and 40% MVC.

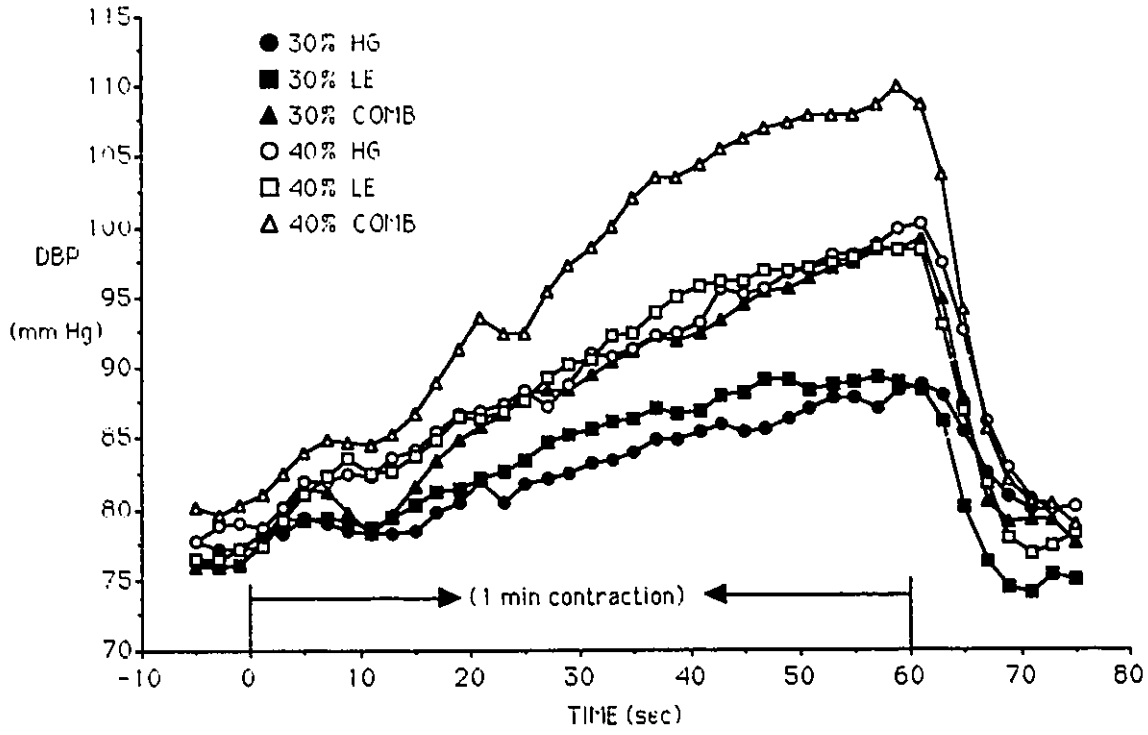

Figure 2: Continuous DBP response (2 sec interval) for isometric contractions (HG, LE and COMB) at 30% and 40% MVC.

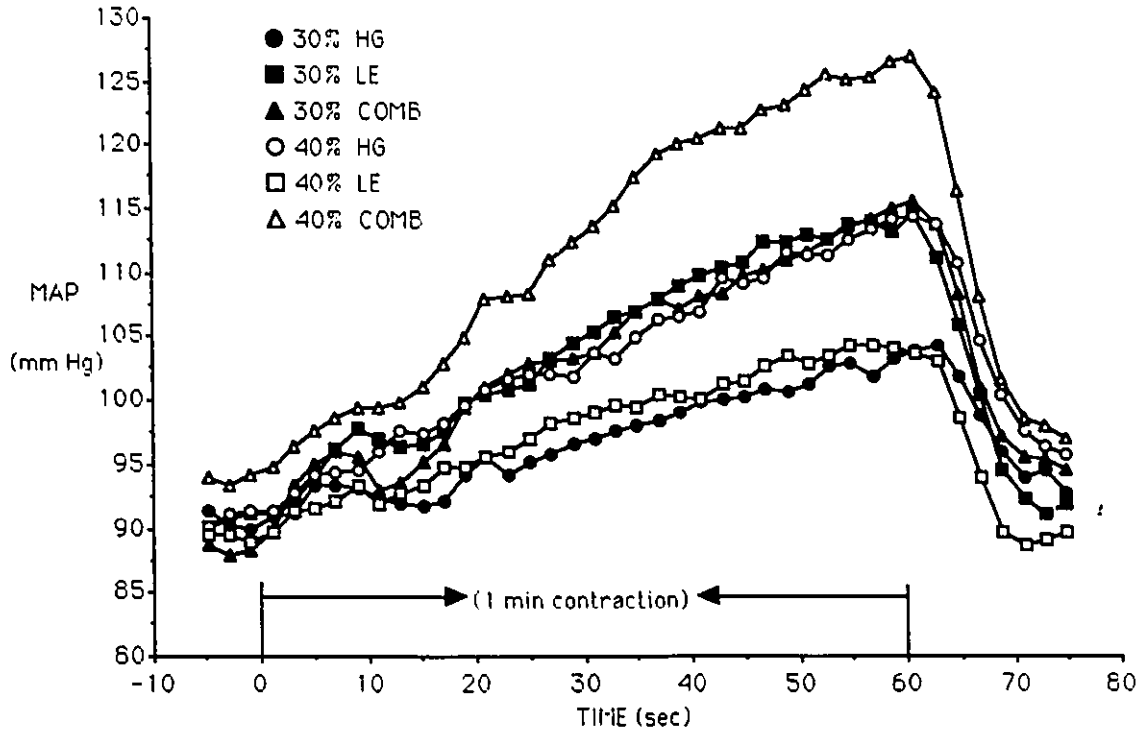

Figure 3: Continuous MAP response (2 sec interval) for isometric contractions (HG, LE and COMB) at 30% and 40% MVC.

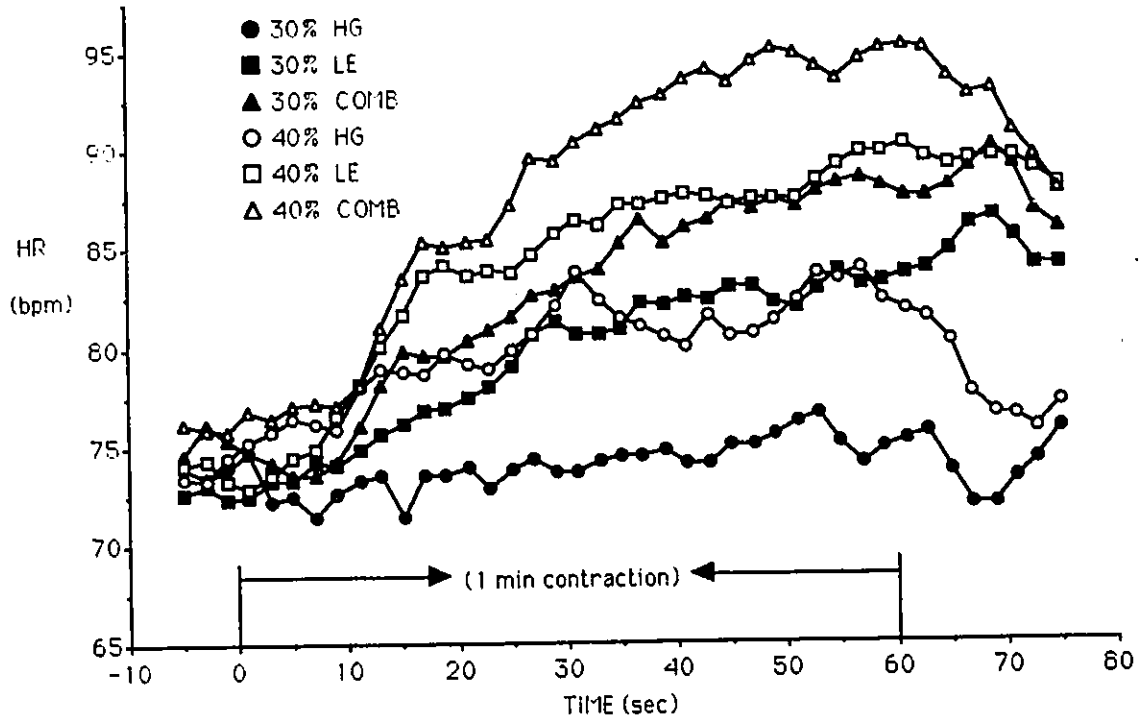

Figure 4: Continuous HR response (2 sec interval) for isometric contractions (HG, LE and COMB) at 30% and 40% MVC.

II

INTRODUCTION

1.1 Introduction

Muscular exercise is accompanied by important changes in cardiovascular function which lead to an increase in blood flow to the working muscles (Perez-Gonzalez, 1981). The nature of these changes depend on whether dynamic or static exercise is being performed. Mitchell (1976) defined isotonic (dynamic) exercise as primarily a change in muscle length with little change in muscle tension. Conversely, Temkin (1983) defined isometric (static) exercise as the development of tension within a muscle without significant change in length of its fibres. Thus, these two types of exercise will lead to different hemodynamic responses. Rhythmic or dynamic muscular activity produces increases in heart rate, stroke volume, cardiac output and systolic blood pressure, while systemic vascular resistance falls dramatically (Lewis, et al., 1985). Diastolic blood pressure remains relatively constant or increases slightly (Kino, et al., 1975). Conversely, sustained or isometric contraction tends to produce less of an increase in cardiac output and heart rate with little or no increase in stroke volume, but with an acute increase in systolic and diastolic blood pressures without an accompanying fall in systemic vascular resistance.

Since the human cardiovascular response during isometric exercise is attributed to both peripheral and central stimuli, a number of studies have been conducted to determine whether the reflex from the working muscle is related to the muscle mass involved in the contraction or the percentage of maximal tension achieved. Lind and McNicol (1967) provided evidence suggesting that the increase in heart rate and blood pressure were similar at the same comparative tensions, irrespective of the size of the muscle mass involved or of the absolute tension generated. Riendl, Gotshall, Reinke and Smith (1977) showed that muscle mass does not influence the cardiovascular response during isometric contraction of large and small muscle groups, but that intensity of exercise is the contributing factor influencing this hemodynamic response. They also concluded a

dissociation in the mechanisms responsible for heart rate and blood pressure since there was an evident divergence between these two responses during static contraction. Likewise, McCloskey and Streatfeild (1975) concluded that the cardiovascular responses to sustained contractions performed by human subjects are determined by the proportion of maximal tension achieved rather than the muscle mass recruited. Furthermore, muscle mass had a less obvious affect on heart rate than blood pressure during isometric exercise. On the other hand, Buck, Amundsen and Nielsen (1980) indicated a significant difference in pressor response between small (finger adductor) and large (handgrip) muscle groups of the upper extremity during static exercise at 40% MVC. The results indicated that larger muscle masses do exhibit a greater pressor response. Similarly, Nagle, Seals and Hanson (1988) showed that the pressor response was indeed a function of the amount of muscle mass being utilized. Their results indicated a progressively greater blood pressure and heart rate response in healthy men as muscle mass increased when performing handgrip, left and right leg extension and dead lift at 30% MVC. Likewise, Mitchell, Payne, Saltin and Schibye (1980) demonstrated a linear increase in blood pressure and heart rate with respect to muscle mass during isometric exercise at 40% MVC as follows: fingers (digits II and III), forearm, knee extensors and combined forearm and knee extensors. They concluded that both pressor response and increased heart rate were dependent upon muscle mass at the same intensity during sustained contraction.

As such, there is a discrepancy in the literature pertaining to the role of muscle mass and intensity of exercise on cardiovascular response to isometric stress. Therefore, further investigation is required to provide insight with respect to the influence of muscle mass and intensity on blood pressure response to isometric exercise.

1.2 Statement of The Problem

The purpose of this study was to determine the effect of different muscle masses and intensities to selected isometric stressors with respect to the pressor response in normotensive males.

1.3 Scope of The Study

Twenty-two subjects ranging in age from 24 to 32 years volunteered to participate in the study. Prior to isometric testing, subjects were requested to participate in the CAFT to ensure that they did not have a predisposition to an exaggerated blood pressure response to exercise. The isometric tests were performed at 30 and 40% MVC using the Martin Vigorometer and Kin-Com 500H Isokinetic apparatus. The tests included one-arm handgrip, one-leg extension and combined one-arm handgrip and one-leg extension. Cardiovascular responses (heart rate, systolic blood pressure, diastolic blood pressure and mean arteriol pressure) were continuously measured using a Finapres 2300BP monitor. Analysis of variance with repeated measurements was utilized to determine the influence of muscle mass recruitment and intensity of exercise on the cardiovascular response to isometric exercise. When a significant F-value was determined, Least Square Means technique for post-hoc analysis was administered to determine significant differences between the means. A linear regression coefficient was used to calculate the difference between the blood pressure slopes (pressor response) of the three muscle masses at 30% and 40% MVC.

1.4 Definition of Terms And Abbreviations

1-RM:

A single repetition maximum.

Afterload:

This is the increased aortic pressure placed on the heart due to the sudden increase in blood pressure without decreases in total peripheral resistance. A common occurrence during isometric exercise.

Central Reflex:

A response elicited by direct action of the central nervous system, including the spinal cord and brain, which descends on the medullary cardiovascular centres.

Isometric (Static) Contraction:

Contraction in which tension is developed without any significant change in the length of the muscle fibre.

Maximum Voluntary Contraction (MVC):

The maximum contraction of a single or group of muscles during an isometric exercise.

Muscle Mass:

The number of muscle fibres recruited for an exercise.

Peripheral Reflex:

The response to exercise is reflexly elicited by afferent neural activity from receptors in the skeletal muscle or joints acting upon the medullary cardiovascular centres.

Total Peripheral Resistance

In the circulatory system the arterioles represent the main source of resistance to the flow of arterial blood. The resistance therefore, is beyond the level of the artery where blood pressure is measured thus it is termed peripheral resistance.

Pressor Response:

The abrupt increase in blood pressure following isometric exercise, primarily due to an increase in cardiac output with little or no change in total peripheral resistance.

III

REVIEW OF LITERATURE

2.1 Introduction

Isometric exercise is physiologically characterized by a dramatic increase in blood pressure (pressor) response on the cardiovascular system. Yet, the exact nature of the mechanisms underlying the pressor response during tetanic muscular activity are unknown. Not until this skepticism is further investigated can these mechanisms, which govern the pressor response to isometric exercise, be understood and appreciated. Many of these mechanisms will be investigated with respect to the progressive recruitment of muscle mass on blood pressure response to isometric exercise in normotensive males.

2.2 Mechanical Hindrance to Blood Flow

Shepherd et al. (1981) suggested that the blood flow to skeletal muscle during tetanic contraction is dependent upon the combined influence of local metabolites, intramuscular pressure and perfusion pressure.

2.2.1 Metabolites

Mitchell, Schibye, Payne and Saltin (1981) stated that the blood pressure response within the periphery of the exercising muscle during static contraction is associated with receptors that are activated by metabolic events related to the muscle contraction. Likewise, Lind and McNicol (1967) suggested that fatiguing isometric exercise is associated with a large pressor response, which is related to a reflex arc stimulated by metabolites released into the interstitial space of working muscle fibres. Alam and Smirk (1937) observed normal men perform repeated leg extension and handgrip while a large sphygmomanometer cuff provided a mechanical occlusion,

whereby arresting the circulation through the legs and the arms and localizing metabolites. The results indicated that when circulation was arrested following exercise, a rise in blood pressure was significantly greater than when the same exercise is performed without the circulation occluded. Saltin, Sjogaard, Gaffney and Rowell (1981) evaluated the relationship between interstitial potassium and lactate metabolic concentrations and alterations in heart rate and blood pressure during static exercise of the human quadriceps muscle. They concluded that elevated lactate is an unlikely mediator of central hemodynamic responses, while potassium demonstrates a temporal and semi-quantitative relationship with blood pressure response. Therefore, if a peripheral chemical does exist for the regulation of the cardiovascular response during static contraction, potassium would be a more likely metabolite than lactate, although other compounds may also be of significance.

2.2.2 Intramuscular Pressure

During isometric exercise, the intramuscular pressure in the contracting muscle will cause a mechanical inhibition to blood flow and therefore elevate the systemic vascular resistance. Saltin et al. (1981) suggested that the proportion to which this resistance will occur depends on the muscle mass recruited as well as the relation between intramuscular and arterial pressure. Also, Sejersted, Hargens, Kardel, Blom, Jensen and Hermansen (1984) reported that intramuscular fluid pressure increased linearly with force up to maximal voluntary contraction. Yet, during prolonged submaximal static contraction, the intramuscular pressure acted independent of the force developed. Sejersted et al. (1984) hypothesized that blood flow is initially impeded deep in the muscle where pressure is the greatest and also in short bulging muscles with enormous fibre curvature compared with long slender fibres. Humphrey and Lind (1963) investigated the blood flow through the active and inactive forearm during sustained handgrip contraction and concluded that increased blood flow went predominantly to the active muscles and that the rate of blood flow increase was associated with tension developed. Also, Humphrey and Lind (1963) concluded that intramuscular pressure cannot occlude the blood supply until the tension has exceeded 70% MVC. Despite the effect of mechanical hindrance during isometric contraction on systemic vascular resistance, the most influential factor effecting blood pressure stems from the reflexly mediated

pressor response (Shepherd, et al., 1981).

2.2.3 Perfusion Pressure

According to Shepherd et al. (1981), perfusion pressure increases with the duration and force of voluntary muscle contraction. Quarry and Spodick (1974) investigated the cardiac response to isometric exercise in normal males with respect to different postures and levels of exertion. The results suggest that the significant changes in cardiocirculatory response are directly related to the level of exertion. Also, Quarry and Spodick found that in order to provide significant cardiocirculatory responses, isometric intensities must be 50 to 100% MVC. Conversely, Lind, Taylor, Humphreys, Kennelly and Donald (1963) indicated significant responses in cardiac output, systemic arterial pressure and heart rate before, during and after sustained handgrip contractions, held 10, 20 and 50% of MVC. It was concluded that the combination of vasoconstriction to inactive muscles and organs and increased cardiac output raised the blood pressure in an attempt to maintain perfusion pressure within the contracting muscle, which was inhibited by mechanical compression.

In conclusion, mechanical hinderance demands a symmetrical relationship between local metabolites, intramuscular pressure and perfusion pressure to efficiently maintain blood flow to the statically contracted muscle. Potassium is a likely metabolite produced during static contraction that stimulates afferent receptors within the active muscle, which in turn produce an increase in cardiac output and muscle blood flow. Intramuscular pressure provides a mechanical hinderance initially within the deepest part of the muscle and is dependent upon the muscle mass and tension generated to increase systemic vascular resistance during isometric exercise. Finally, perfusion pressure is maintained by increases in both vasoconstriction in nonactive muscles and cardiac output. Blood flow within the active muscle is not occluded during static exercise until intensities greater than 70% MVC are attained.

2.3 Peripheral Reflex

Alam and Smirk (1937) showed a blood pressure increase following rhythmic exercise of the forearm and leg muscles by either arresting the circulation through the muscle during contraction, or rapid rhythmic exercise. Both clinical methods were effective in accumulating metabolites within the working muscle and in causing an increase in arterial blood pressure proportional to the intensity of the exercise. All ensuing studies have verified the conclusion established by Alam and Smirk that the prime stimuli of the rise in blood pressure was a reflex originating in the exercising muscle {Figure I} (Shepherd et al., 1981).

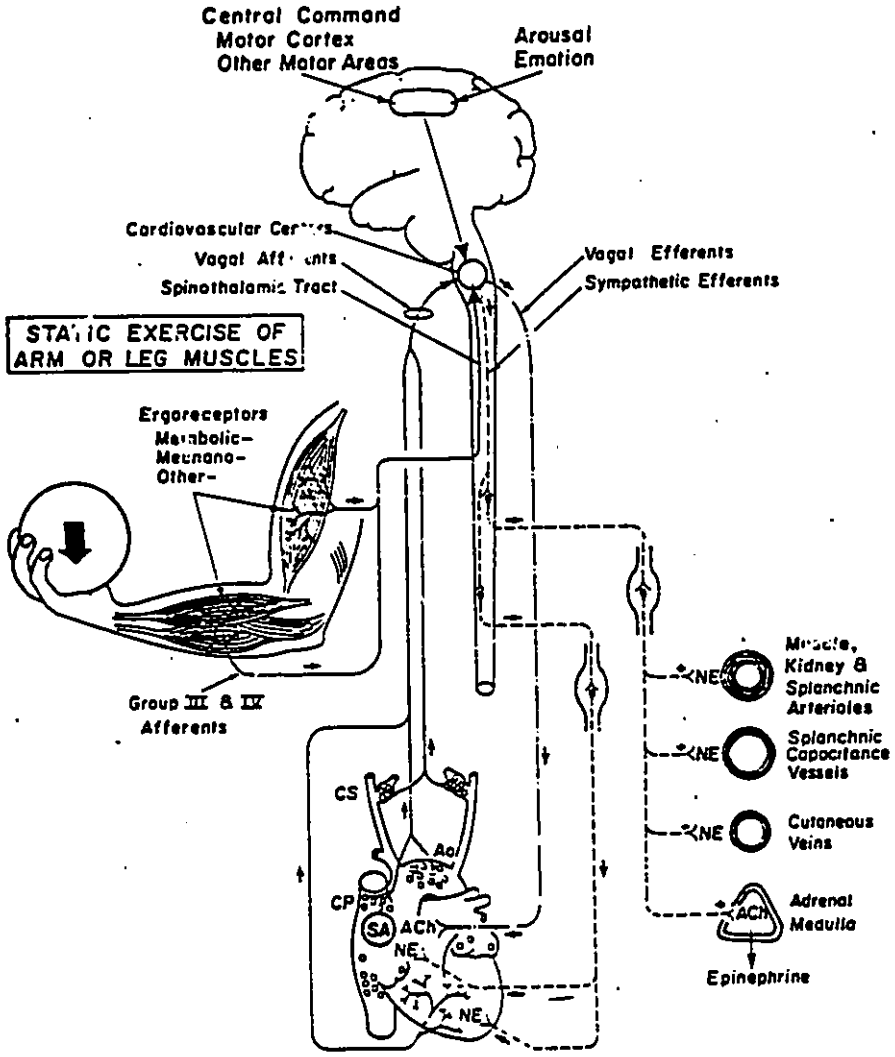

Figure I: Static Exercise:Retrospective And Introspective. (From Shepherd et al., 1981, p. I-181, by permission of the American Heart Association, Inc.)

2.3.1 Metabolites Acting on Afferent Nerve Endings

In young men, the blood pressure response following static contraction remains elevated when the circulation is inhibited with an inflatable cuff (Mitchell et al., 1981). It would appear that peripheral contribution is associated with receptors that are stimulated by metabolic events surrounding the muscle during static contraction. This would suggest that central commands accompanying the exercise, or mechanoreceptors within the muscles, are not entirely responsible for the pressor reflex (Shepherd et al., 1981). Studies were performed in vagotomized, chloralose-anesthetized dogs, whereas stimulation of muscle afferent fibre produced an increase in systemic arterial pressure and cardiac output that appears designed to maintain blood flow through the resistance vessels of the contracting muscle (Nutter & Wickliffe, 1981). Perez-Gonzalez (1981) studied a subject performing both isometric and rhythmic 30% MVC handgrip contractions. When rhythmic contraction was repeated with the brachial artery completely occluded, pressor response was much more prominent and similar to the blood pressure reflex obtained previously during static handgrip.

These studies reviewed suggest that the circulatory response to exercise is modulated by metabolic factors stimulating afferent nerve endings and therefore act as a contributing factor in the pressor response (Shepherd et al., 1981; Perez-Gonzalez, 1981).

2.3.2 Mechanoreceptors III and IV

Mechanoreceptor afferents with conduction velocities greater than 17m/sec (group I and II fibres) do not elicit reflex stimulation of vasomotor sympathetic nerves or cause increases in blood pressure. Therefore, only group III and IV afferents conducting below 15m/sec can elicit a reflex pressor response (Shepherd et al., 1981). A study conducted on adult cats, rats and dogs using electrical stimulation of muscle afferents as well as natural stimulation of stretch, pressure and induced exercise concluded that group III and IV fibres in muscle afferents were most prominently responsible for the cardiopulmonary effects in muscular activity. Kalia, Mei and Kao (1981) confirmed that there are unmyelinated fibres connected to pressure receptors which produce

cardiopulmonary reflex effects.

According to Kniffni, Mense and Schmidt (1981), the majority of afferent nerve fibres in mammalian skeletal muscle are thin myelinated (group III) and unmyelinated (group IV) afferents. The functions concerning these two afferents are to induce cardiovascular and respiratory adjustments during exercise (ergoreceptive function) and to transmit nociceptive information from the muscle to the spinal cord (nociceptive function). Kniffki et al. (1981) concluded that 50% of these group III and IV fibres are excited by noxious stimuli such as chemical, mechanical or thermal (ie. nociceptors), while non-noxious stimuli such as light stretch, contractions and light local touching activate receptive units with fine afferent fibres. These mechanoreceptors act as modulators in the circulatory and respiratory adjustments during exercise (ie. ergoreceptors). Shepherd et al. (1981) summarized the contribution of mechanoreceptors by suggesting that when circulatory occlusion is applied at the end of a period of contraction, the pressor response which is maintained is not the full response that is achieved during the contraction. Intramuscular mechanoreceptors generate part of the reflex drive during the initial static contraction period.

2.3.3 Potassium Ion Influx

Potassium ions from the active muscle may be a contributing factor in the blood pressure response during isometric contraction (Shepherd et al. 1981). Saltin et al. (1981) indicated that changes in potassium concentration of the femoral venous blood closely follow the pressor reflex during and after static exercise, whereas blood pressure had returned to normal values when lactate concentration was at its greatest. Saltin et al. (1981) concluded that afferent impulses originating in the exercising muscle could possibly be responsible for the central hemodynamic response to isometric exercise. Likewise, McCloskey and Mitchell (1972) showed increases in cardiovascular and ventilatory responses following isotonic potassium-chloride injection in cats and dogs during muscular exercise, presumably due to stimulation of small myelinated and unmyelinated afferents in the muscle. It can be concluded from these studies that metabolic and chemical receptors in the working muscle serve a significant role in the pressor response during static exercise. Shepherd et al. (1981) stated that no concrete decision can be reached as to the exact stimulus for the muscle

reflex, nor whether anaerobic exercise is necessary in manufacturing the necessary by-products for the pressor response.

Therefore, peripheral reflex originating in the statically contracted muscle acts as a prime factor in the rise in blood pressure. Studies involving muscle occlusion and vagotomized animals support the contribution of metabolic influence on muscle afferent fibres that stimulate increases in systemic arterial pressure and cardiac output. Mechanoreceptors III and IV have been identified as muscle afferent fibres whose function is to induce cardiovascular and respiratory adjustments as well as to transmit information from the periphery to the spinal cord during isometric exercise. Also, potassium by-products produced during static exercise stimulate small myelinated and unmyelinated metabolic and chemical ergoreceptors in the active muscle which cause a peripheral reflex.

2.4 Central Reflex

Johansson (1894) stated that the cardiovascular response to exercise is regulated by neurogenic "central command" descending from the higher motor centres. Goodwin, McCloskey and Mitchell (1972) investigated the blood pressure response during isometric exercise in human subjects where varying central motor demand was measured. Results indicated that when a similar force and less central command during static exercise was incorporated, then blood pressure and heart rate were lower then when similar force and greater central command were utilized. Goodwin et al. (1972) concluded from their study that the cardiovascular response during static exercise is radiated by descending central commands. Yet, other investigations suggest that central influence is not totally understood (Longhurst and Mitchell, 1979).

2.4.1 Cortical Irradiation

Cortical irradiation of the neurogenic component is defined as the result of a direct message from the motor cortex on the autonomic areas of the brainstem (Krogh and Lindhard, 1913). Mitchell et al. (1981) claimed that central mechanisms are responsible for extremely fast elevations

in blood pressure and heart rate during the initial stages of isometric exercise. Alam and Smirk (1939) observed a decrease in blood pressure by 5-10 mm Hg after the cessation of exercise with circulation occluded. They suggested that the small blood pressure fall is an indication of the cessation of mental contribution associated with the muscles involved in the activity. Shepherd et al. (1981) stated that a portion of the increase in sympathetic outflow and decreased vagal tone associated with the cardiovascular response to isometric exercise is due to stimulation initially in the higher centres of the brain.

2.4.2 Muscle Fibre Recruitment

Mitchell et al. (1980) studied the role of muscle mass recruitment on the cardiovascular response to static exercise in eleven healthy males. Activation of greater motor units and a larger muscle mass generated a larger contribution from both central and peripheral components and thus increased blood pressure and heart rate. The central contribution alone provided interesting values with respect to increases in the number of motor units recruited from different muscle masses (ie. finger, forearm and leg). It is suggested that the number of motor neurons activated to produce a given force with arm muscles may be larger then when using the leg muscles, since the number of muscle fibres in a motor unit is less in the arms than in the legs (Feinstein, Lindegard, Nyman & Wohlfart, 1955). The study conducted by Mitchell et al. (1981) investigated the blood pressure response in young men at different intensities during isometric exercise. Results showed that the central input is related to the quantity of centrally activated motor units or the discharge frequencies

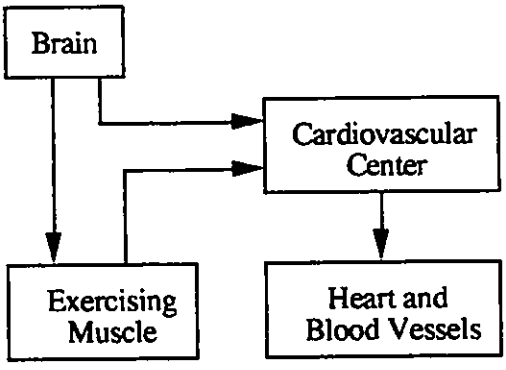

Figure II: Pressor Response To Static Exercise. (From Mitchell et al., 1981, p. I75, by permission of the American Heart Association, Inc.)

in recruited motor units, whereas peripheral input is mediated by receptors activated by metabolic events associated with the exercising muscles (Figure II).

In conclusion, the cardiovascular response to isometric exercise is mediated by a neurogenic central command initiating from the higher motor centres of the brain. Cortical irradiation is considered responsible for the initial increases in blood pressure and heart rate during isometric exercise due to a direct message from the motor cortex to the autonomic areas of the brainstem increasing sympathetic outflow and decreasing vagal tone. Since muscle fibre recruitment is associated with the number of motor units within a muscle, central reflex is responsible for the quantity of centrally activated motor neurons and discharge frequency in recruiting motor units during any given intensity of isometric exercise.

2.5 Muscle Mass and Tension

Research has been undertaken to determine whether the peripheral reflex generated in the active muscle during isometric exercise is due to the muscle mass or percentage of tension achieved. Studies have resulted in a controversy as to the effect of muscle mass and tension in both elevated blood pressure (pressor response) and heart rate (Lind & McNicol, 1967; McCloskey & Streatfeild, 1975; Mitchell et al., 1981; Misner, Going, Massey, Ball, Bemben & Essandoh, 1990). The following studies investigated the contribution of muscle mass and tension on the increases in blood pressure and heart rate during static contraction.

2.5.1 Pressor Response and Elevated Heart Rate

Lind and McNicol (1967) suggested that the magnitude of the increase in heart rate and blood pressure during sustained contraction of the arms and legs in four healthy men is associated with tension developed, and unrelated to the mass of the muscles. The results indicated that when two or more muscle groups contracted isometrically at the same relative tension, the increases in heart rate and blood pressure were the same, whether they were contracted separately or combined. Neither the mass recruited, nor the absolute tension they produced had any bearing on the

cardiovascular response. McCloskey and Streatfeild (1975) sunported these findings in male and female subjects during static contraction of the finger and hand regarding blood pressure response, but suggested that the influence of muscle mass on heart rate is less definite and may influence the increase in heart rate. Likewise, Misner et al. (1990) studied the cardiovascular response to maximal voluntary sustained 2-minute static contraction by three different muscle groups (right hand finger flexors, right leg extensors and both leg extensors) in young adult males and females. Their results showed that blood pressure increased throughout sustained muscular contractions. Conversely, heart rate did not increase throughout exercise under these conditions, suggesting that different mechanisms are contributing to the mediation of blood pressure and heart rate. Notwithstanding, Mitchell et al. (1981) found that the pressor response during sustained contraction at 40% MVC elicited progressive elevations when the muscle mass was increased. Also, following occlusion of the muscle after cessation of contraction, the blood pressure again increased linearly with a larger contribution of muscle mass.

The above studies have indicated that muscle mass and tension are influential in both pressor response and increased heart rate during isometric exercise. Yet, the mechanisms responsible for increases in blood pressure and heart rate may be very different during isometric exercise.

2.5.2 Muscle Fibre Type and Characteristics

Petrofsky, Phillips and Lind (1981) examined the blood pressure response during isometric exercise in the medial gastrocnemius muscle of female cats at sustained tensions from 10 to 100% MVC. By voluntarily recruitment of slow or fast twitch fibres, Petrofsky et al. (1981) discovered that during the initial stages of static contraction when essentially slow twitch fibres are incorporated, blood pressure response was significantly lower than when fast twitch motor units were recruited first. The explanation supporting this observation may be due to the metabolism of slow and fast twitch fibres, or to the density of sensory receptors surrounding these motor units. Asmussen (1981) stated that the tension limit, in which blood flow is completely occluded during sustained exercise, is partially dependent on the muscle fibre type within the muscle. For example,

in the elbow extensor consisting mainly of fast twitch fibres, the limit lies at about 10% MVC. Yet, in the elbow flexor where there are very few fast twitch fibres, the tension limit is much greater at 25% MVC. Asmussen (1981) stated that during a prolonged static exercise, fast twitch fibres are vulnerable to fatigue as compared to slow twitch fibres. Shepherd et al. (1981) suggested that in muscle, a mixed population of fibre types have a varied number of ergoreceptors and that the distribution of these receptors is muscle-specific. Therefore, with regard to the afferent impulses stimulating the cardiovascular centres of the brain during isometric exercise, a large muscle which contains fewer motor units than a small muscle at any given tension, may provide similar central commands (Asmussen, 1981).

In conclusion, muscle mass and tension may play a significant contribution in the peripheral reflex. Studies indicate that they both influence pressor response and increased heart rate, although the mechanism affecting blood pressure and heart rate may be very different. Fast twitch fibres contribute the greatest to blood pressure increases during isometric exercise, yet they limit static exercise due to their nature to fatigue quickly.

2.6 Hemodynamic Response

In healthy normal men, isometric exercise causes increases in sympathetic stimulation and decreased vagal outflow (Shepherd et al., 1981). As a result, cardiac output, heart rate mean arterial pressure, systolic blood pressure and diastolic blood pressure are all increased (Rowell, Freund & Hobbs, 1981; Gonyea, Diepstra, Muntz & Mitchell, 1981) without increases in systemic vascular resistance (Martin, Shaver, Leon, Thompson, Reddy & Leonard, 1974). Increases in cardiac output are due primarily to tachycardia (Asmussen, 1981). Thus, the elevation in blood pressure during static exercise is a reflection of the increase in cardiac output (Shepherd et al., 1981).

2.6.1 Autonomic Influence

The efferent system is controlled by sympathetic and parasympathetic nerve stimulation and has been studied using alpha- and beta-adrenergic antagonists and anticholinergic agents (Goldstraw & Warren, 1985). Martin et al. (1974) investigated the autonomic mechanisms in hemodynamic response to isometric exercise in male subjects. Using selected autonomic blockade with intravenous propranolol, practolol, atropine and combined atropine-propranolol to emulate the role of autonomic nervous system, young male volunteers performed 30% MVC handgrip contraction for 3 minutes. The initial increase in heart rate (30 seconds) was due to vagal withdrawal, since this response was inhibited by atropine. The remainder of the response (increased heart rate) was attributed to sympathetic outflow, since it was not influenced by atropine, but by combined atropine-propranolol blockade. Martin et al. (1974) concluded that vagal withdrawal appears to be the initial mechanism contributing to increases in heart rate followed by increases in sympathetic stimulation during isometric exercise. Likewise, Gonyea, Diepstra, Muntz and Mitchell (1981) studied the cardiovascular response to static exercise training in the conscious cat using propranolol and atropine autonomic blocking agents. Beta-adrenergic receptor blockade with propranolol had no significant effect on the heart rate response, whereas vagal or parasympathetic blockade with atropine suppressed the heart rate increases during the initial isometric testing sessions. Also, no adaptations were observed in intrinsic heart rate following three months of training, suggesting that heart rate reduction at the completion of the training was instrumented by neural mechanisms. Gonyea et al. (1981) also concluded that the initial tachycardia during static exercise is mediated by vagal withdrawal (parasympathetic system), whereas the left ventricular contractility is influenced primarily by the sympathetic nervous system. Clements, Offord, Baron, Brown, Bardsley and Harrison (1984) investigated the cardiovascular hemodynamics of bicycle and handgrip exercise in normal subjects before and after the administration of propranolol. During isometric handgrip, systolic and diastolic arterial pressure increased moderately with only a mild increase in heart rate. The results from this study confirm those of Gonyea et al. (1981) with respect to propranolol, suggesting that beta-adrenergic blockade has a predominant effect on heart rate during isometric exercise. Finally, Perez-Gonzalez, Schiller and Parmley (1981) showed that administration of propranolol diminished the expected increase in

heart rate during isometric handgrip at 30% MVC for three minutes. However, the pressor response was unaffected since it is mediated by alpha-adrenergic stimulation. They concluded that the pressor response seen for static contraction is due to increased cardiac output, which in turn, is primarily influenced by increased heart rate. However, even in the absence of increased heart rate, blood pressure can nevertheless increase dramatically due to alpha constriction of the systemic vascular bed. Nutter and Wickliffe (1981) claimed that vasoconstriction may occur in a number of vascular beds during isometric exercise in order to increase systemic arterial pressure and muscle perfusion. The study by Lind and McNicol (1967) analyzed the hemodynamic response during sustained exercise (2.5 min.) in healthy men during handgrip exercises at 20, 30 and 50% MVC. Heart rate increased during exercise with elevations in blood flow and pressure in the working muscle. The change in blood flow in the exercising forearm represents a compromise between metabolically induced vasodilation, intramuscular pressure and possibly a vasoconstriction due to sympathetic activity. Likewise, Williams, Mudd and Lind (1981) measured forearm blood flow at two seconds following repeated contractions of 4 seconds at 60% MVC, with 8 seconds between exercises. Following the infusion of phentolamine (alpha-blocker), blood flow was significantly reduced, suggesting that increased sympathetic activity reduces blood flow between intermittent contractions.

2.6.2 Effect on Inactive Muscle

Seals (1989) studied the relationship between changes in efferent muscle sympathetic nerve activity to the lower leg and calf vascular resistance during handgrip contraction in humans. He claimed that their is a linear increase with intensity and duration in muscle sympathetic nerve discharge between vascular resistance in active and non-active muscles. Yet, it appears that a vasodilator mechanism is elicited in the forearm that completely counteracts the vasoconstriction in the active muscle and thereby increases blood flow in the forearm. Meanwhile, blood flow to non-active musculature is continually inhibited due to sympathetic nerve activity. Similarly, Humphreys and Lind (1963) studied the blood flow through active and inactive muscles of the forearm during sustained handgrip contractions at various intensities. The results were as expected indicating blood flow increases predominantly to muscles active in the contraction. An interesting

observation discussed by Humphreys and Lind (1963) deals with the blood flow through the contracted muscles when the tension developed was a value greater than 70% MVC. Contraction at this intensity increased intramuscular pressure and impeded blood supply. Likewise, Lind, Dahms, Williams and Petrofsky (1981) examined blood flow through the resting arm during handgrip contraction in twelve volunteer subjects. In the experiment, blood flow and oxygen uptake were measured during a given isometric contraction and calibrated against the electromyographic activity. The working arm then became the contralateral (resting) arm, while the other hand performed isometric exercise. Six of the twelve subjects demonstrated increased muscular activity, blood flow and oxygen uptake in the non-active contralateral arm. Rusch, Shepherd, Webb and Vanhoutte (1981) reported similar results following simultaneous evaluation of the human calf and forearm resistance vessels during contralateral isometric exercise, mental stress and abnormal respiratory movements. During the sustained contraction, vascular resistance increased in the active leg due to noradrenergic outflow and decreased in the resting contralateral forearm. The mechanism responsible for the increased circulation in the forearm has been attributed to metabolic demands due to it's involuntary contraction during isometric exercise of the initial working forearm (Lind et al., 1981) or to vasodilation in the resting limb resulting from cholinergic fibres. An interesting observation in the study by Rusch et al. (1981) was that during emotional stress, dilation occurred in the vessels of the resting forearm and vasoconstricted in those of the calf. This would provoke question as to the importance of central command in triggering a dilator mechanism which offsets the well known vasoconstrictor response.

Thus, hemodynamic responses are affected by autonomic influence in producing the desired reaction to an isometric stress. Studies using drugs designed to block the function of the autonomic system indicate that beta-and alpha-adrenergic stimulation initiate the increases in heart rate and blood pressure, respectively. Blood flow increases in the active musculature during isometric exercise due to a vasodilatory overriding on the sympathetic nerve activity. Vasoconstriction continues to restrict blood flow to non-active muscles throughout static exercise. Increases in blood flow to the contralateral forearm, following cessation of isometric handgrip and emotional stress suggest a metabolic and central influence on blood pressure, respectively.

2.7 Baroreceptor Influence

As blood pressure increases, the stretch of the arterial vessels in the active muscle stimulates the arterial baroreceptors to reflexly decrease heart rate and compensatory dilation of the peripheral vasculature, which inevitably decreases blood pressure (Katch, Katch & McArdle, 1986). According to Abboud, Mark and Thames (1981) the role of arterial baroreceptor during isometric exercise is important because of the characteristic rises in arterial pressure, cardiac filling pressure and an increase in the force of ventricular contraction. Also, numerous cardiovascular disorders such as aortic stenosis, hypertension and myocardial infarction are associated with abnormal responses to exercise and can be influenced by cardiac baroreceptors.

2.7.1 Normal vs. Inhibited Response

The ability of the carotid sinus baroreflex to control blood pressure and heart rate is not affected during isometric exercise and it is unlikely that reduction in arterial baroreceptor sensitivity has a role in the initiation of the heart rate and blood pressure response to static contraction (Shepherd et al., 1981). Conversely, Abboud et al. (1981) investigated the baroreceptor influence on blood pressure and heart rate responses during isometric handgrip at 10 and 20% MVC. The results indicated that carotid baroreflex dampened the sympathetic activation from muscle afferent nerves (mechanoreceptors III and IV) and stimulated vagal afferents. However, when exposed to lower body negative pressure (hypotension), their was an enhanced vasoconstrictor response. This was attributed primarily to an uninhibited sympathetic stimulation and depressed activation of cardiopulmonary baroreceptor response during isometric exercise. Similarly, Smith, Graitzer, Hudson and Raven (1988) studied the baroreflex function in untrained subjects, endurance athletes and weight-trained men in response to progressive incremental phenylephrine infusion and lower body negative pressure. The data indicated that endurance trained subjects had a reduced heart rate-mean blood pressure slope during both phenylephrine infusion and lower body negative pressure compared to untrained and weight-trained subjects. Smith et al. (1988) proposed that the differences could be due to an alteration in the reflex cardiovascular control of the endurance trained (1-2h/day) athletes not seen in the weight-trained subjects. Various levels of lower body

negative pressure were studied in both athletes (football players) and nonathletes to determine to what extent forearm vascular resistance is augmented by cardiovascular baroreflex control (Takeshita, Jingu, Imaizumi, Kunihiko, Koyanagi and Nakamura, 1986). The major observation in this study was that the reflex forearm vasoconstriction in response to lower body negative pressure at -10 mmHg and the slope of the regression line relating alterations in central venous pressure and forearm vascular resistance was steeper in athletes than in nonathletes.

These studies indicate that exercise training can augment the increase of inhibitory influence of the cardiopulmonary receptors in man. The augmentation of cardiovascular response to static exercise can be influenced by decreased activity of carotid receptors or vagal afferents (Rowell, et al., 1981).

2.8 Static vs. Dynamic Exercise

Combined static and dynamic exercise are commonly performed in everyday life, especially in occupational work where pushing, pulling, carrying and handling are frequent occurrences (Kilbom & Persson, 1981). Yet, it is important that one recognizes the hemodynamic differences between static and dynamic exercise.

2.8.1 Hemodynamic Response

Muscular exercise is accompanied by important changes in cardiovascular function which lead to an increase in blood flow to the working muscles (Perez-Gonzalez, 1981). The nature of such changes seem to depend on two basic kinds of exercises being performed. Rhythmic or dynamic muscle activity produces increases in heart rate, stroke volume, cardiac output and systolic blood pressure. No elevation or even decreases in diastolic blood pressure occur causing only small changes in mean arterial blood pressure (Kino, Lance, Shahamatpour & Spodick, 1975). Conversely, static or isometric contraction tends to produces smaller increases in heart rate and cardiac output with little or no increase in stroke volume, and acute increases in both systolic and diastolic blood pressures without any fall in total peripheral resistance. Tuttle and Horvath

(1957) investigated the effects of static (handgrip) and dynamic (bicycle ergometer) work on the blood pressure and heart rate response in men. During bicycle ergometry, a significant increase in systolic blood pressure occurred, followed either by no change or a slight decrease in diastolic pressure. Conversely, static contraction produced sharp rises in both systolic and diastolic arterial pressures. The energy cost of performing handgrip and bicycle ergometry was also different. The average oxygen debt for the dynamic work was 1200 ml, whereas an average debt of only 150 ml was measured following static contraction. Tuttle and Horvath (1957) concluded that the blood pressure responses are a consequence of reflexes which are modified by the metabolic changes associated with the exercise. Similarly, Blomquist, Lewis, Taylor and Graham (1981) studied the hemodynamic response to static and dynamic exercise of small muscle groups in young men. Differences between modes of exercise were a higher oxygen uptake during dynamic one-arm curl compared to handgrip, while the circulatory results were almost identical during both exercises, except for a slightly higher diastolic arterial pressure during handgrip at 50% MVC.

Numerous studies investigating left ventricular function have also indicated significant differences with respect to isometric and dynamic exercise (Shepherd et al., 1981). Keul, Simon and Lehmann (1981) analyzed echocardiographic measurements in successful athletes associated with endurance running and resistance weight training, as well as untrained subjects and patients with hypertension. The endurance trained athletes had an increased left ventricular end-diastolic diameter relative to body weight in contrast to weight trained subjects. Ejection fraction was also greater in endurance runners, whereas weight trained athletes indicated a moderate decrease. Longhurst, Kelly, Gonyea and Mitchell (1981) studied the effects of acute and chronic dynamic and static exercise on the cardiovascular system in endurance runners, weight lifters and initially sedentary subjects. During static handgrip contraction, endurance athletes showed significantly greater end-diastolic and end-systolic volumes when compared to sedentary subjects. Shepherd et al. (1981) concluded, with respect to left ventricular function, that the normal cardiac response to dynamic exercise includes both a Starling effect and an enhanced contractile state. The left ventricular end-diastolic and end-systolic volumes during isometric exercise in sedentary subjects are smaller and tend to be unpredictable. Various studies investigating isometric exercise have demonstrated that end-diastolic and end-systolic dimensions have been known to decrease. Perez-

Gonzalez, Schiller and Parmley (1981) demonstrated no change in end-diastolic diameter before or after the administration of propranolol in five healthy subjects following isometric handgrip, whereas end-systolic diameter increased slightly without the use of drugs. Although decreases in left ventricular dimension do occur following static exercise, the expected elevations have been observed in healthy subjects, heart disease patients (Helfant, Maria, DeVilla & Meister,1971) and aortic valve disease patients (Krayenbuehl, Grimm, Turina & Senning, 1981) following isometric exercise. Helfant et al. (1971) concluded that handgrip isometric exercise imposes an acute afterload on the left ventricle due to increases in systolic and diastolic pressures; this response is not characteristic of dynamic exercise.

2.8.2 Muscle Mass and Mode of Contraction

Blomqvist et al. (1981) stated that muscle mass, irrespective of mode of contraction (static or dynamic) is the determining factor influencing the hemodynamic response. The rate of elevation in mean, systolic and diastolic blood pressures with increasing oxygen uptake was inversely proportional to the active muscle mass, suggesting that a gradual transition from dynamic to static hemodynamic response is evident with decreasing muscle mass. Conversely, Perez-Gonzalez (1981) concluded from his studies on anesthetized cats, when stimulation of ventral roots elicited comparable sensory messages to that of static and dynamic exercise, that a pressor response was observed following isometric and not dynamic sensations. Thus, mode of contraction is not a determining factor of the cardiovascular response to dynamic and static exercise. Lewis, Snell, Taylor, Hamra, Graham, Pettinger and Blomqvist (1985) investigated the role of muscle mass and mode of contraction in the circulatory responses to static and dynamic exercise in healthy males. Increases in mean arterial pressure were similar for each mode of contraction (static/dynamic handgrip and static/dynamic two-knee extension). Cardiac output was greater during dynamic than static, as well as for two-knee exercise over handgrip exercise. Conversely, systemic vascular resistance was lower during dynamic exercise when compared to static exercise. Lewis et al. (1985) summarized their observations by stating that the magnitude of the pressor response was associated with the active muscle mass, but independent of the mode of contraction. Yet, the mode of contraction contributed to the circulatory changes stimulating the pressor response.

In conclusion, the major difference between static and dynamic exercise is that isometric contraction imposes an afterload on the heart, while rhythmic exercise causes a preload on the heart. Also, the blood pressure response to isometric exercise tends to be greater than to dynamic exercise due to metabolic receptor demands. Muscle mass and type of exercise affect blood pressure differently to isometric and dynamic contractions, suggesting functional differences. Finally, dynamic training tends to increase end-diastolic and end-systolic volume more than isometric training in both normal subjects and heart patients.

2.9 Cardiovascular Disease and Training in Isometrics

Numerous studies have been conducted on subjects suffering from cardiovascular disease (Haskell et al., 1981; Jetté et al., 1988; Colombo et al., 1989), as well as dynamic or static exercise training (Longhurst et al., 1980; Keul et al., 1981; Maiorano et al., 1989) using isometric exercise as an influence on hemodynamic response. Also, subjects with cardiovascular disease who performed endurance training have been investigated with respect to their hemodynamic response to isometric exercise (Sullivan, Higginbotham, Frederick & Cobb, 1988). Regardless of the physical condition, the hemodynamic response is still commonly characterized by a dramatic increase in blood pressure during isometric contraction.

2.9.1 Cardiovascular Disease

Haskell et al. (1981) investigated the cardiovascular responses of handgrip exercise in patients following cardiac transplantation. Both cardiac transplant and ischemic heart disease patients were able to increase arterial pressure during isometric exercise of 50% MVC. Haskell et al. (1981) concluded that the pressor response could be attributed primarily to elevated peripheral vascular resistance, since these patients lack the ability to increase heart rate and stroke volume. Similarly, Jetté et al. (1988) assessed left ventricular function during dynamic (supine cycling) and isometric (dual handgrip, 50% MVC) exercise in normal subjects and myocardial infarct patients. Significant increases in heart rate, systemic blood pressure and pulmonary capillary pressure occurred during static contraction in both normal subjects and patients, suggesting that isometric

exercise at 50% MVC is beneficial in evaluating the hemodynamic response, and in particular, the left ventricular function of myocardial infarct patients. Krayenbuehl et al. (1981) reported that isometric handgrip of 30% MVC was effective in uncovering abnormalities of left ventricular function in aortic valve disease patients at the pre- and post-operative catheterization which were not detectable in the unstressed state. Conversely, Colombo et al. (1989) investigated the effect of isometric handgrip at 30% MVC, bicycle ergometry and tyramine infusion on blood pressure and heart rate variations in hypertensive in-patients and normotensive healthy subjects. The results showed that tyramine infusion elicited a significantly greater increase of systolic blood pressure as compared to bicycle ergometry and handgrip, while bicycle ergometry gave rise to higher diastolic blood pressure compared to tyramine infusion and handgrip in hypertensive patients. Colombo et al. (1989) concluded that bicycle ergometry and tyramine infusion are more useful than isometric handgrip for the detection of cardiovascular hyperreactivity in hypertensive patients.

2.9.2 Training

Studies have been conducted to determine the different effects that dynamic and static exercise training have on the cardiovascular system during both modes of exercise (Shepherd et al., 1981). Longhurst et al. (1980) and Keul et al. (1981) similarly investigated the effect of static and dynamic training on the cardiovascular response to static and dynamic training and in particular, left ventricular dimension in successful endurance and power athletes. Results were similar for endurance runners and weight lifters; whereas echocardiography detected increases in absolute left ventricular mass as a consequence of dynamic and static training. Longhurst et al. (1980) also showed a larger myocardial mass relative to body mass, but only in the endurance runners. It was concluded that endurance training can alter both the absolute and relative left ventricular mass, and the response of the cardiovascular system to static exercise. In agreement, Maiorano et al. (1989) found that blood pressures appeared to be lower at rest and during static handgrip at 30% MVC in dynamically trained subjects compared to untrained volunteers. A high correlation between elevated systolic blood pressure and anthropometric measures of obesity in untrained athletes suggested that overweight is a factor contributing to an increase in the activity of the sympathetic nervous system (Maiorano et al., 1989).

Therefore, both cardiovascular disease patients and trained subjects indicate a pressor response to isometric exercise. The former relys predominantly on increases in systemic vascular resistance with isometric exercise to produce the pressor response since cardiac output and stroke volume increase minimally. Static stress at intensities of 30% and 50% MVC are effective in evaluating the pressor response. Endurance training can alter the cardiovascular response to isometric exercise. In particular, blood pressure tends to be less in response to static exercise in the dynamically trained subject, which could be attributed to a smaller sympathetic outflow contribution.

2.10 Summary

The preceeding studies have demonstrated numerous examples of the pressor response during isometric exercise. Many factors have contributed to the hemodynamic changes that occur when the musculature is under sustained contraction. The mechanisms responsible for these changes have been associated with both central and peripheral reflexes. Muscle mass and intensity of exercise are factors of specific interest due to thier involvement with mechanical hinderance of the contracted muscle, autonomic stimulation, mechanoreceptor influence, central command on the motor neurons and adaptations with training. Therefore, the effect of muscle mass and intensity on the blood pressure response to isometric exercise can offer further understanding into the possible mechanisms which contribute to the pressor response.

IV

METHODOLOGY

3.1 Introduction

This study was designed to examine the effect of muscle mass and intensity of exercise on selected isometric exercises with respect to the pressor response in normotensive males.

3.2 Subjects

Twenty moderately active male subjects age 24 to 32 years participated in this study. All subjects gave informed written consent to participate in the study prior to testing. All subjects met the following criteria:

1) male, age 24 to 32 years
2) do not suffer from a physical ailment which could alter their performance
2) non-smoker
3) resting blood pressure lower than 140/90 mm Hg and exercise blood pressure less than 170/100 mm Hg following the initial stage of the CAFT
4) answer "no" to all questions on the modified PAR-Q
5) not taking medication that would alter cardiovascular response or physical performance.

3.3 Study Protocol

The testing protocol was conducted on two consecutive days.

Day I

Screening of Subjects

At the initial testing session subjects were thoroughly briefed as to the nature of the

study and their involvement. Subjects were requested to sign an informed consent (Appendix B) and complete a modified PAR-Q (Appendix A) prior to participation and a health questionnaire (Appendix C). The subjects were then seated for a five minute period. Resting heart rate and blood pressure values were measured using a stethoscope and sphygmomanometer at the end of the rest period. The subjects were then screened to ensure that they did not demonstrate an exaggerated blood pressure response to exercise by participating in the initial stage of the Canadian Aerobic Fitness Test (CAFT) (Jetté, Landry and Sidney, 1990). Participants between the ages of 20 and 29 years started at stage 5, while those between 30 and 39 years started at stage 4. Subjects walked up and down a two-step staircase (20.3 cm) to a six-count recorded musical rhythm for three (3) minutes at a rate equivalent to 65 to 70% of the average aerobic power anticipated in a person in a 10 year older age group than the subject's (CSTF, 1986). Blood pressure and heart rate measurements were monitored using a calibrated aneroid sphygmomanometer and stethoscope prior to exercise. At the completion of the 3-minute exercise, blood pressure was determined immediately following the 10-second post-exercise heart rate measurement {ie, between the 15th and 45th second of post-exercise}(Jetté, Landry and Sidney, 1990). If the subjects heart rate did not exceed as predetermined standard following the initial stage and no physically harmful symptoms were observed, the participants were allowed to proceed to the second and third stages of the CAFT. An exaggerated blood pressure response was considered if the subject's systolic blood pressure exceeded 170 mmHg or Δ 45 mmHg following the initial stage of the CAFT. A predicted maximum aerobic capacity was calculated from the measurements of the last completed stage of the CAFT performed by the subjects using the Jetté procedure (Appendix D). These calculations were used to the determine the subjects maxVO_2 and provide insight into the fitness level of each participant.

Anthropometric Measures

The following anthropometric measurements were then recorded: height, weight, chest, abdomen and gluteal (Jetté, 1983). Body mass index (BMI), chest minus waist (CMW) and waist to hip ratio (WHR) were calculated from the anthropometric measurements. These basic

anthropometric measurements were evaluated to determine whether the subjects represented a normal sample of the population.

Determining MVC (1-RM)

Subjects were instructed on how to properly perform one-arm handgrip using the Martin Vigorometer and one-leg extension using the Kin-Com 500H Isokinetic apparatus (Chattecx Corp.; Chattanooga, TN) while wearing a Finapres 2300 BP monitor (Ohmeda; Louisville, CO). Subjects were then provided with a 20 minute rest while sitting on the Kin Com 500H Isokinetic apparatus before the start of the isometric testing. The subjects were instructed to exhale during isometric exercises so as not to perform a Valsalva manoeuvre. Maximum voluntary contraction (MVC) was determined for handgrip and leg extension exercises from the greatest of three repeated maximal contractions for each exercise. The duration of each maximal repetition (1-RM) was five seconds with a two minute rest between each contraction.

Day II

Pressor Response to 30% and 40% MVC Isometric Exercises

Day two served as the actual testing session to determine the influence of muscle mass and intensity on blood pressure response to isometric exercise. Following a five minute period of inactivity, resting heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were measured from the second index finger on the subject's right hand while wearing the Finapres 2300 BP monitor cuff (Hartmann and Bassenge, 1989). During the examination phase, subjects were seated in an upright position on the Kin-Com 500H Isokinetic apparatus and performed 30% of their predetermined MVC for one minute for one-arm handgrip, one-leg extension and combined one-arm handgrip and one-leg extension tasks. Each of the three tasks was performed once with a five minute rest period between each exercise. The subjects were then provided with a ten minute rest period before being instructed to perform the same three isometric tasks at 40% MVC. The order of isometric exercise was randomly selected for each subject. (Reliability of 0.95 was determined from previous pilot investigations).

3.4 Statistical Analysis

Descriptive analysis of the participants included the following means: age, height, weight, resting heart rate, resting blood pressures, predicted maxVO_2, maximum handgrip strength and maximum leg extension strength.

The hemodynamic response of the three muscle masses during isometric exercise were compared at two different intensities. A 3X2 factorial design analysis of variance (ANOVA) with repeated measures was conducted to determine if there were any significant differences in heart rate and blood pressures with respect to muscle mass and intensity to isometric stressors. The independent variables in this study included three separate muscle masses (1. one-arm handgrip, 2. one-leg extension 3. combined one-arm handgrip and one-leg extension) and two levels of intensity (30% and 40% MVC). The dependent variables measured included heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure at rest and during one minute of static contraction. In the presence of a significant F-ratio, Least Square Means technique was administered for post-hoc analysis in order to localize significant differences between the means. An alpha level of 0.05 was used as a standard for statistical significance.

Linear regression analysis was calculated for both systolic and diastolic blood pressure each of the three isometric exercises at 30% and 40% MVC to determine the slope of the pressor responses. The slope for each of the contractions was calculated using comparison of regression coefficients to determine if thier were any significant differences between the pressor responses for the three respective isometric exercises at both 30% and 40% MVC (Edwards, 1984). An alpha level of 0.05 was used as a standard for statistical significance.

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V

APPENDICES

Appendix A

DEPARTMENT OF HUMAN KINETICS THESIS RESEARCH CONSENT FORM

DEPARTMENT OF HUMAN KINETICS
THESIS RESEARCH CONSENT FORM

Whenever a study involving human subjects is undertaken, the University of Ottawa Human Research Ethics Committee requires written consent of the participants. I, _____, authorize Brent E. Faught (230-1611) of the School of Human Kinetics, University of Ottawa, to conduct the following fitness evaluation and selected isometric tests. This study will be orchestrated under the supervision of Dr. Maurice Jetté, School of Human Kinetics (564-9108). The purpose of this study will be to examine the effect of muscle mass and intensity of exercise on the blood pressure response during isometric exercises in normotensive males age 24 to 32 years.

I understand that the testing protocol will be conducted on two separate days. During the initial session (Day 1), I will be requested to complete a modified PAR-Q form (Jetté form). I will then perform three stages of the Canadian Aerobic Fitness Test (CAFT), whereas I will step up and down a two-step staircase (20.3 cm) for three (3) minutes at a rate equivalent to 65 to 70% of the average aerobic power anticipated of a person in a 10 year older age group than mine. My blood pressure and heart rate measurements will be monitored prior to and upon completion of the test. Anthropometric measurements will then be taken in order to determine my height, weight and girths. After these preliminary tests, I will be shown and instructed how to perform the following required tasks for fifteen (15) minutes on the Kin-Com 500H Isokinetic apparatus and Trerice dynomometer, while wearing the Finapres 2300 BP Monitor:

1. left handgrip
2. dominant leg extension
3. combined left handgrip and dominant leg extension.

I understand that I will be given a ten (10) minute rest before I am instructed to maximally contract during the three respective isometric tasks. The three tasks will consist of three maximal contractions, each of which will be for a duration of five (5) seconds followed by a five (5) minute rest between each contraction. Thus, the total duration of the test will be 2 hours from the initial ten minute rest to the completion of the last isometric task.

On day two (2), I understand that I will wear the Finapres 2300 BP Monitor on my index finger of my right hand throughout the entire examination phase, during which time blood pressure and heart rate will be recorded. Resting values (heart rate and blood pressures) will be monitored, while I am in a sitting position following a 5 minute period of inactivity. Next, I will participate in the actual isometric testing session, During this time, I will be instructed to isometrically contract 30% of my predetermined MVC for one minute for each of the three respective isometric tasks. I will then repeat the same respective isometric tasks at 40% MVC. I understand that I will perform each task only once with a five (5) minute rest period between each exercise. The handgrip will be performed using the Trerice dynomometer, while dominant leg extension will be performed on the Kin-Com Isokinetic apparatus. Throughout the examination, I will be sitting in an upright position on the Kin-Com 500H Isokinetic apparatus.

I understand all information collected will be kept confidential and presented in an anonymous form in the final report. I understand that I will be informed of my performance in relation to this study concerning the effect of muscle mass and intensity on blood pressure response to isometric exercises in normotensive males. I understand that I have the right to withdraw from this study at any time.

Subject: _____

Witness: _____

Date: _____

Appendix B

PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)

Modified Physical Activity Readiness Questionnaire (PAR-Q)

Family Name:_____ Date _____

Given Name: _____ Age: _____

1.	Has a physician ever said you have heart trouble?	Yes	No
2.	Do you frequently have pains in your heart and chest?	Yes	No
3.	Do you often feel faint or have spells of severe dizziness?	Yes	No
4.	Has a physician ever said that your blood pressure was too high?	Yes	No
5.	Do you suffer from any respiratory tract problem such as chronic bronchitis, asthma or emphysema?	Yes	No
6.	Have you ever had or are you now suffering from any nervous disorder?	Yes	No
7.	Do you suffer from any bone or joint problem which either has been or may be irritated by an exercise session?	Yes	No
8.	Do you know of a valid medical reason why you should not be involved in either a regular exercise program or an exercise testing session?	Yes	No
9.	At present, are you taking medication for blood pressure? If yes, please specify:	Yes	No

Reason:_____

Name: _____

Dosage:_____

10.	At present, are you taking any other type of medication, whether are prescribed or "over the counter"? If yes, please specify:	Yes	No

Reason:_____

Name: _____

Dosage:_____

Signature: _____

Date:_____

Jetté, M., Quenneville, J., Sidney, K., Fitness Testingand Counselling in Health Promotion, 1990 ©

Appendix C

HEALTH QUESTIONNAIRE

HEALTH QUESTIONNAIRE

Family Name: _____ Date _____

Given Name: _____ Age: _____

It would be greatly appreciated if you would answer the follow ing questions by checking the appropriate response.

1. Are you at present: a smoker ____
   non smoker ____
   ex smoker ____

2. If you are an ex smoker: cigarettes: no./day ____
   pipe: bowls/day ____
   cigars: no.day ____

3. If you are an ex smoker, please indicate the number of years as an: ex smoker: ____
   smoker: _ _

4. Which one of the following statements best describes your participation in physical activity? Please circle the appropriate number:

   1. Non exerciser; no regular participation in physical activities.
   2. Light exerciser; occasional participation (2 or 3 times per month) in physical activities which cause slight changes above the normal state.
   3. Moderate exerciser; participation in regular physical activities (once or twice a week) which cause perspiration and an increase in breathing above the normal state.
   4. Heavy exerciser; participation in regular physical activities three or more times a week, 20 minutes or more each time and at a level which causes perspiration and heavy breathing

5. With respect to alcohol consumption, which of the following statements best describes your drinking habits? Please circle the appropriate number:

   1. abstain or rarely drink alcohol
   2. drink 1 to 3 drinks per week
   3. drink 4 to 7 drinks per week
   4. drink 8 to 12 drinks per week
   5. drink more than 13 drinks per week

Note:

one drink = beer: 12-oz bottle/can
wine: 4-oz
liquor: 1.5-oz

Appendix D

PREDICTED MAXIMUM AEROBIC CAPACITY EQUATION

Predicted Maximum Aerobic Capacity Equation

Stage Completed

5th $MaxV\dot{O}_2 = 42.5 + \{16.6 \times 2.0066\} - \{0.12 \times \text{weight (kg)}\} - \{0.12 \times HR\} - \{0.24 \times age\}$

6th $MaxV\dot{O}_2 = 42.5 + \{16.6 \times 2.3453\} - \{0.12 \times \text{weight (kg)}\} - \{0.12 \times HR\} - \{0.24 \times age\}$

7th $MaxV\dot{O}_2 = 42.5 + \{16.6 \times 2.7657\} - \{0.12 \times \text{weight (kg)}\} - \{0.12 \times HR\} - \{0.24 \times age\}$

From: Jette, M., Campbell, J., Mongeon, J. and Routhier, R. (1976). The Canadian Home Fitness Test as a predictor of aerobic capacity

Appendix E

PERMISSION FOR COPYRIGHT MATERIAL

May 6, 1991

The American Heart Association,
7320 Greenville Ave.,
Dallas, TX
U.S.A.
75231

To whom it may concern;

I am currently completing my thesis on "The Effect of Muscle Mass and Intensity on The Blood Pressure Response in Normotensive Males" for a Master of Science degree in Kinanthropology at the University of Ottawa. I wish to use two of your figures in my review of literature. I am referring to Figure 12 from Mitchell et al. (1981). Response of Arterial Blood Pressure to Static Exercise in Relation to Muscle Mass, Force Development and Electromyographic Activity in Circulation Research p. I-75, as well as Figure 1 from Shepherd et al. (1981). Static (Isometric) Exercise Retrospective and Introspective in Circulation Research p. I-181.

These figures would be used solely for my thesis and not for publication purposes. Therefore, I am requesting permission to reproduce these two figures in my thesis. Thank you very much for your attention in this matter.

Sincerely,

Brent E. Faught

Brent E. Faught
212 Henderson Ave.
Ottawa, ON
Canada
K1N 7P7

Permission is granted provided the publication is credited as the source and author's permission is obtained.

JUN 4 1991

AMERICAN HEART ASSOCIATION
Scientific Publications Department

Appendix F

RAW DATA

	SBP						Input Column
	low intensity			high intensity			
	HG	LE	HG/LE	HG	LE	HG/LE	
1	138.30	137.03	148.33	141.80	141.57	147.87	
2	156.07	150.70	169.87	158.90	172.87	182.54	
3	129.90	146.33	141.53	127.50	138.03	151.97	
4	109.50	114.97	128.13	113.59	128.47	139.17	
5	137.90	136.55	156.27	153.73	160.07	141.77	
6	132.40	125.63	129.93	134.36	136.63	132.27	
7	125.23	132.10	136.60	122.93	134.00	140.37	
8	136.63	140.00	146.27	141.14	139.83	151.87	
9	113.30	110.67	122.53	120.63	129.47	128.60	
10	133.03	143.20	130.07	138.90	139.23	157.33	
11	141.33	132.27	152.43	157.23	145.90	167.17	
12	136.97	124.86	146.23	135.33	151.63	149.10	
13	125.90	136.80	139.43	143.31	139.33	142.76	
14	136.83	130.79	150.33	135.70	134.60	151.60	
15	124.80	129.38	135.83	134.20	127.80	140.57	
16	143.08	136.53	138.20	142.60	140.63	153.63	
17	131.30	124.87	131.15	144.00	146.70	155.47	
18	146.81	146.47	156.73	151.00	155.83	169.93	
19	134.70	133.57	143.60	147.40	150.27	157.33	
20	153.87	140.96	156.67	138.17	155.55	165.37	

	SBP						Input Column
	low intensity			high intensity			
	HG	LE	HG/LE	HG	LE	HG/LE	
Type:	Real	Real	Real	Real	Real	Real	Real
Source:	User Entered	User Entered	User Entered	User Entered	User Entered	User Entered	User Entered
Class:	Continuous	Continuous	Continuous	Continuous	Continuous	Continuous	Continuous
Format:	Free Format Fi...	Free Format Fi...	Free Format Fi...	Free Format Fi...	Free Format Fi...	Free Format Fi...	Free Format Fi...
Dec. Places:	2	2	2	2	2	2	3
Mean:	134.39	133.68	143.01	139.12	143.42	151.33	•
Std. Deviation:	11.52	10.17	11.95	11.86	11.56	13.20	•
Std. Error:	2.58	2.27	2.67	2.65	2.58	2.95	•
Variance:	132.66	103.43	142.89	140.62	133.63	174.14	•
Coeff. of Variation:	8.57	7.61	8.36	8.52	8.06	8.72	•
Minimum:	109.50	110.67	122.53	113.59	127.80	128.60	•
Maximum:	156.07	150.70	169.87	158.90	172.87	182.54	•
Range:	46.57	40.03	47.34	45.31	45.07	53.94	•
Count:	20	20	20	20	20	20	•
Missing Cells:	0	0	0	0	0	0	•
Sum:	2687.85	2673.68	2860.13	2782.42	2868.41	3026.69	•
Sum of Squares:	363747.41	359393.49	411732.08	389764.84	413927.86	461351.36	•

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Subject	19	12988.123	683.585		
INTENSITY	1	1298.802	1298.802	42.246	.0001
INTENSITY * Subj...	19	584.126	30.743		
EXERCISE	3	10431.385	3477.128	60.501	.0001
EXERCISE * Subject	57	3275.898	57.472		
INTENSITY * EXER...	3	566.335	188.778	6.117	.0011
INTENSITY * EXER...	57	1758.970	30.859		

Dependent: SBP

Table of Epsilon Factors for df Adjustment
Dependent: SBP

	G-G Epsilon	H-F Epsilon
INTENSITY	1.000	1.000
EXERCISE	.580	.634
INTENSITY * EXERC...	.762	.872

Least Squares Means Table
Effect: EXERCISE
Dependent: SBP

	Vs.	Diff.	Std. Error	t-Test	P-Value
HG	LE	-1.795	1.695	-1.059	.2940
	COMB	-10.414	1.695	-6.143	.0001
	REST	12.211	1.695	7.204	.0001
LE	COMB	-8.618	1.695	-5.084	.0001
	REST	14.007	1.695	8.263	.0001
COMB	REST	22.625	1.695	13.347	.0001

Least Squares Means Table
Effect: INTENSITY * EXERCISE
Dependent: SBP

	Vs.	Diff.	Std. Error	t-Test	P-Value
LOW INTENSITY, HG	LOW INTENSITY, LE	.709	1.757	.403	.6882
	LOW INTENSITY, COMB	-8.614	1.757	-4.904	.0001
	LOW INTENSITY, REST	9.847	1.757	5.605	.0001
	HI INTENSITY, HG	-4.728	1.757	-2.692	.0093
	HI INTENSITY, LE	-9.028	1.757	-5.139	.0001
	HI INTENSITY, COMB	-16.942	1.757	-9.644	.0001
	HI INTENSITY, REST	9.847	1.757	5.605	.0001
LOW INTENSITY, LE	LOW INTENSITY, COMB	-9.322	1.757	-5.307	.0001
	LOW INTENSITY, REST	9.139	1.757	5.202	.0001
	HI INTENSITY, HG	-5.437	1.757	-3.095	.0030
	HI INTENSITY, LE	-9.736	1.757	-5.543	.0001
	HI INTENSITY, COMB	-17.650	1.757	-10.048	.0001
	HI INTENSITY, REST	9.139	1.757	5.202	.0001
LOW INTENSITY, COMB	LOW INTENSITY, REST	18.461	1.757	10.509	.0001
	HI INTENSITY, HG	3.885	1.757	2.212	.0310
	HI INTENSITY, LE	-.414	1.757	-.236	.8145
	HI INTENSITY, COMB	-8.328	1.757	-4.741	.0001
	HI INTENSITY, REST	18.461	1.757	10.509	.0001
LOW INTENSITY, REST	HI INTENSITY, HG	-14.576	1.757	-8.297	.0001
	HI INTENSITY, LE	-18.875	1.757	-10.745	.0001
	HI INTENSITY, COMB	-26.789	1.757	-15.250	.0001
	HI INTENSITY, REST	0.000	.	.	.
HI INTENSITY, HG	HI INTENSITY, LE	-4.300	1.757	-2.448	.0175
	HI INTENSITY, COMB	-12.214	1.757	-6.953	.0001
	HI INTENSITY, REST	14.576	1.757	8.297	.0001
HI INTENSITY, LE	HI INTENSITY, COMB	-7.914	1.757	-4.505	.0001
	HI INTENSITY, REST	18.875	1.757	10.745	.0001
HI INTENSITY, COMB	HI INTENSITY, REST	26.789	1.757	15.250	.0001

	DBP						Input Column
	low intensity			high intensity			
	HG	LE	HG/LE	HG	LE	HG/LE	
1	80.00	77.72	84.63	109.00	78.97	102.23	
2	96.40	101.93	110.37	102.63	124.47	122.32	
3	81.03	89.40	86.60	83.03	90.47	97.07	
4	67.07	68.43	77.00	70.30	79.33	81.23	
5	76.00	82.17	88.87	89.03	91.17	81.93	
6	74.67	72.90	78.90	83.39	84.43	79.67	
7	76.20	91.25	82.37	79.39	86.73	88.23	
8	82.30	76.33	86.67	84.53	87.20	96.73	
9	53.90	59.50	61.53	67.47	69.77	73.70	
10	73:37	77.87	76.43	80.57	83.10	95.63	
11	98.43	93.10	97.93	102.07	91.40	101.40	
12	90.72	93.26	96.67	97.69	80.77	98.13	
13	80.03	84.73	88.13	88.47	89.70	91.14	
14	89.23	86.03	99.53	99.50	99.70	108.63	
15	86.83	93.45	92.63	96.27	87.87	99.80	
16	100.35	92.73	95.20	93.23	93.50	105.63	
17	86.73	83.43	86.59	93.63	93.73	99.73	
18	90.83	91.37	98.27	93.48	100.30	108.43	
19	86.87	85.00	89.07	91.27	95.86	99.03	
20	89.67	87.79	94.03	89.37	90.28	106.70	

	DBP						Input Column
	low intensity			high intensity			
	HG	LE	HG/LE	HG	LE	HG/LE	
Type:	Real	Real	Real	Real	Real	Real	Real
Source:	User Entered	User Entered	User Entered	User Entered	User Entered	User Entered	User Entered
Class:	Continuous	Continuous	Continuous	Continuous	Continuous	Continuous	Continuous
Format:	Free Format Fl...	Free Format Fi...	Free Format Fi...	Free Format Fi...	Free Format Fi...	Free Format Fi...	Free Format Fi...
Dec. Places:	2	2	2	2	2	2	3
Mean:	83.03	84.42	88.57	89.72	89.94	96.87	.
Std. Deviation:	11.11	10.04	10.53	10.55	10.96	11.64	.
Std. Error:	2.48	2.24	2.35	2.36	2.45	2.60	.
Variance:	123.50	100.71	110.78	111.25	120.22	135.52	.
Coeff. of Variation:	13.38	11.89	11.88	11.76	12.19	12.02	.
Minimum:	53.90	59.50	61.53	67.47	69.77	73.70	.
Maximum:	100.35	101.93	110.37	109.00	124.47	122.32	.
Range:	46.45	42.43	48.84	41.53	54.70	48.62	.
Count:	20	20	20	20	20	20	.
Missing Cells:	0	0	0	0	0	0	.
Sum:	1660.63	1688.39	1771.42	1794.32	1798.75	1937.36	.
Sum of Squares:	140231.15	144446.54	159001.19	163092.89	164059.24	190243.04	.

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Subject	19	12658.532	666.239		
INTENSITY	1	1050.574	1050.574	47.209	.0001
INTENSITY * Subj...	19	422.817	22.254		
EXERCISE	3	4495.876	1498.625	32.091	.0001
EXERCISE * Subject	57	2661.885	46.700		
INTENSITY * EXER...	3	389.137	129.712	7.289	.0003
INTENSITY * EXER...	57	1014.318	17.795		

Dependent: DBP

Table of Epsilon Factors for df Adjustment
Dependent: DBP

	G-G Epsilon	H-F Epsilon
INTENSITY	1.000	1.000
EXERCISE	.686	.771
INTENSITY * EXERC...	.817	.947

Least Squares Means Table
Effect: EXERCISE
Dependent: DBP

	Vs.	Diff.	Std. Error	t-Test	P-Value
HG	LE	-.805	1.528	-.527	.6005
	COMB	-6.346	1.528	-4.153	.0001
	REST	8.481	1.528	5.550	.0001
LE	COMB	-5.541	1.528	-3.626	.0006
	REST	9.285	1.528	6.077	.0001
COMB	REST	14.826	1.528	9.703	.0001

Least Squares Means Table
Effect: INTENSITY * EXERCISE
Dependent: DBP

	Vs.	Diff.	Std. Error	t-Test	P-Value
LOW INTENSITY, HG	LOW INTENSITY, LE	-1.388	1.334	-1.040	.3025
	LOW INTENSITY, COMB	-5.540	1.334	-4.153	.0001
	LOW INTENSITY, REST	5.138	1.334	3.852	.0003
	HI INTENSITY, HG	-6.685	1.334	-5.011	.0001
	HI INTENSITY, LE	-6.906	1.334	-5.177	.0001
	HI INTENSITY, COMB	-13.837	1.334	-10.372	.0001
	HI INTENSITY, REST	5.138	1.334	3.852	.0003
LOW INTENSITY, LE	LOW INTENSITY, COMB	-4.151	1.334	-3.112	.0029
	LOW INTENSITY, REST	6.526	1.334	4.892	.0001
	HI INTENSITY, HG	-5.296	1.334	-3.970	.0002
	HI INTENSITY, LE	-5.518	1.334	-4.136	.0001
	HI INTENSITY, COMB	-12.449	1.334	-9.332	.0001
	HI INTENSITY, REST	6.526	1.334	4.892	.0001
LOW INTENSITY, COMB	LOW INTENSITY, REST	10.678	1.334	8.005	.0001
	HI INTENSITY, HG	-1.145	1.334	-.858	.3943
	HI INTENSITY, LE	-1.366	1.334	-1.024	.3100
	HI INTENSITY, COMB	-8.297	1.334	-6.220	.0001
	HI INTENSITY, REST	10.678	1.334	8.005	.0001
LOW INTENSITY, REST	HI INTENSITY, HG	-11.823	1.334	-8.863	.0001
	HI INTENSITY, LE	-12.044	1.334	-9.029	.0001
	HI INTENSITY, COMB	-18.975	1.334	-14.224	.0001
	HI INTENSITY, REST	0.000	.	.	.
HI INTENSITY, HG	HI INTENSITY, LE	-.221	1.334	-.166	.8687
	HI INTENSITY, COMB	-7.152	1.334	-5.361	.0001
	HI INTENSITY, REST	11.823	1.334	8.863	.0001
HI INTENSITY, LE	HI INTENSITY, COMB	-6.931	1.334	-5.195	.0001
	HI INTENSITY, REST	12.044	1.334	9.029	.0001
HI INTENSITY, COMB	HI INTENSITY, REST	18.975	1.334	14.224	.0001

	MAP						Input Column
	low intensity			high intensity			
	HG	LE	HG/LE	HG	LE	HG/LE	
1	93.73	90.82	98.50	114.70	93.13	110.97	
2	109.03	110.30	123.50	117.33	135.53	138.64	
3	93.87	102.37	99.60	95.47	101.97	110.63	
4	79.39	81.83	92.23	82.74	91.80	95.93	
5	91.63	95.07	106.23	106.13	108.80	96.97	
6	89.33	87.60	94.20	97.96	97.50	94.13	
7	90.87	99.93	98.50	91.50	100.30	102.23	
8	98.00	98.29	103.33	102.24	101.87	112.27	
9	69.67	74.03	76.67	82.30	84.97	88.53	
10	90.87	94.47	91.40	96.80	98.73	112.60	
11	111.13	103.23	114.03	117.43	106.23	119.93	
12	103.83	107.15	111.87	110.90	100.17	113.93	
13	93.87	99.30	103.00	102.63	103.97	105.41	
14	101.97	99.93	113.43	110.03	110.57	122.43	
15	97.93	104.41	103.77	106.40	98.90	112.77	
16	112.30	108.20	111.13	110.20	110.03	124.07	
17	97.43	94.47	98.15	105.93	105.97	114.17	
18	106.40	106.03	114.57	108.72	116.33	126.80	
19	100.60	99.33	104.23	108.20	112.03	116.63	
20	105.10	101.90	110.63	102.40	105.72	125.10	

	MAP						Input Column
	low intensity			high intensity			
	HG	LE	HG/LE	HG	LE	HG/LE	
Type:	Real	Real	Real	Real	Real	Real	Real
Source:	User Entered	User Entered	User Entered	User Entered	User Entered	User Entered	User Entered
Class:	Continuous	Continuous	Continuous	Continuous	Continuous	Continuous	Continuous
Format:	Free Format Fl...	Free Format Fi...	Free Format Fi...	Free Format Fi...	Free Format Fi...	Free Format Fi...	Free Format Fi...
Dec. Places:	2	2	2	2	2	2	3
Mean:	96.85	97.93	103.45	103.50	104.23	112.21	•
Std. Deviation:	10.43	9.02	10.50	9.98	10.48	12.45	•
Std. Error:	2.33	2.02	2.35	2.23	2.34	2.78	•
Variance:	108.87	81.39	110.16	99.56	109.80	154.94	•
Coeff. of Variation:	10.77	9.21	10.15	9.64	10.05	11.09	•
Minimum:	69.67	74.03	76.67	82.30	84.97	88.53	•
Maximum:	112.30	110.30	123.50	117.43	135.53	138.64	•
Range:	42.63	36.27	46.83	35.13	50.56	50.11	•
Count:	20	20	20	20	20	20	•
Missing Cells:	0	0	0	0	0	0	•
Sum:	1936.95	1958.66	2068.97	2070.01	2084.52	2244.14	•
Sum of Squares:	189657.24	193363.88	216124.86	216138.72	219347.38	254752.09	•

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Subject	19	12003.818	631.780		
INTENSITY	1	1177.713	1177.713	66.458	.0001
INTENSITY * Su...	19	336.703	17.721		
EXERCISE	3	6110.400	2036.800	50.134	.0001
EXERCISE * Sub...	57	2315.752	40.627		
INTENSITY * EX...	3	428.043	142.681	8.848	.0001
INTENSITY * EX...	57	919.167	16.126		

Dependent: MAP

Table of Epsilon Factors for df Adjustment
Dependent: MAP

	G-G Epsilon	H-F Epsilon
INTENSITY	1.000	1.000
EXERCISE	.650	.723
INTENSITY * EXERCISE	.777	.892

Least Squares Means Table
Effect: INTENSITY * EXERCISE
Dependent: MAP

	Vs.	Diff.	Std. Error	t-Test	P-Value
LOW INTENSITY, HG	LOW INTENSITY, LE	-1.085	1.270	-.855	.3962
	LOW INTENSITY, HG/LE	-6.601	1.270	-5.198	.0001
	LOW INTENSITY, REST	6.352	1.270	5.002	.0001
	HIGH INTENSITY, HG	-6.653	1.270	-5.239	.0001
	HIGH INTENSITY, LE	-7.378	1.270	-5.810	.0001
	HIGH INTENSITY, HG/LE	-15.360	1.270	-12.095	.0001
	HIGH INTENSITY, REST	6.352	1.270	5.002	.0001
LOW INTENSITY, LE	LOW INTENSITY, HG/LE	-5.515	1.270	-4.343	.0001
	LOW INTENSITY, REST	7.438	1.270	5.857	.0001
	HIGH INTENSITY, HG	-5.567	1.270	-4.384	.0001
	HIGH INTENSITY, LE	-6.293	1.270	-4.956	.0001
	HIGH INTENSITY, HG/LE	-14.274	1.270	-11.241	.0001
	HIGH INTENSITY, REST	7.438	1.270	5.857	.0001
LOW INTENSITY, HG/LE	LOW INTENSITY, REST	12.953	1.270	10.200	.0001
	HIGH INTENSITY, HG	-.052	1.270	-.041	.9675
	HIGH INTENSITY, LE	-.778	1.270	-.612	.5428
	HIGH INTENSITY, HG/LE	-8.759	1.270	-6.897	.0001
	HIGH INTENSITY, REST	12.953	1.270	10.200	.0001
LOW INTENSITY, REST	HIGH INTENSITY, HG	-13.005	1.270	-10.241	.0001
	HIGH INTENSITY, LE	-13.731	1.270	-10.813	.0001
	HIGH INTENSITY, HG/LE	-21.712	1.270	-17.097	.0001
	HIGH INTENSITY, REST	0.000	.	.	.
HIGH INTENSITY, HG	HIGH INTENSITY, LE	-.725	1.270	-.571	.5700
	HIGH INTENSITY, HG/LE	-8.707	1.270	-6.856	.0001
	HIGH INTENSITY, REST	13.005	1.270	10.241	.0001
HIGH INTENSITY, LE	HIGH INTENSITY, HG/LE	-7.981	1.270	-6.285	.0001
	HIGH INTENSITY, REST	13.731	1.270	10.813	.0001
HIGH INTENSITY, HG/LE	HIGH INTENSITY, REST	21.712	1.270	17.097	.0001

	HR						Input Column
	low intensity			high intensity			
	HG	LE	HG/LE	HG	LE	HG/LE	
1	73.63	70.10	80.40	80.07	79.70	90.13	
2	77.73	89.27	97.37	88.50	108.50	108.14	
3	71.23	79.97	78.03	73.40	78.60	82.67	
4	60.21	62.67	65.27	59.81	68.63	67.80	
5	71.30	74.00	71.37	72.90	71.60	71.87	
6	60.90	72.80	82.13	65.46	73.03	80.07	
7	57.97	71.57	70.53	67.79	79.17	77.80	
8	88.13	92.36	92.63	89.69	95.87	96.57	
9	67.93	71.83	72.43	72.67	79.77	77.20	
10	64.57	75.50	81.33	72.20	81.40	85.77	
11	86.60	89.13	90.13	96.57	94.20	102.43	
12	76.28	79.11	82.80	82.69	85.00	89.73	
13	79.63	83.93	85.67	85.47	86.90	86.97	
14	86.67	90.83	100.17	95.53	99.83	95.60	
15	77.90	80.93	85.40	91.97	84.93	91.43	
16	80.96	84.30	87.77	81.00	84.70	94.57	
17	73.97	75.03	77.63	75.90	77.20	83.63	
18	64.88	75.53	76.17	79.97	69.83	79.47	
19	81.43	81.70	82.50	85.80	85.17	88.80	
20	77.13	84.93	85.20	85.73	94.24	106.50	

	HR						Input Column
	low intensity			high intensity			
	HG	LE	HG/LE	HG	LE	HG/LE	
Type:	Real	Real	Real	Real	Real	Real	Real
Source:	User Entered	User Entered	User Entered	User Entered	User Entered	User Entered	User Entered
Class:	Continuous	Continuous	Continuous	Continuous	Continuous	Continuous	Continuous
Format:	Free Format Fl...	Free Format Fi...	Free Format Fl...	Free Format Fi...	Free Format Fi...	Free Format Fi...	Free Format Fi...
Dec. Places:	2	2	2	2	2	2	3
Mean:	73.95	79.27	82.25	80.16	83.91	87.86	•
Std. Deviation:	9.01	7.88	8.89	10.06	10.43	10.83	•
Std. Error:	2.01	1.76	1.99	2.25	2.33	2.42	•
Variance:	81.11	62.13	79.03	101.20	108.72	117.27	•
Coeff. of Variation:	12.18	9.94	10.81	12.55	12.43	12.33	•
Minimum:	57.97	62.67	65.27	59.81	68.63	67.80	•
Maximum:	88.13	92.36	100.17	96.57	108.50	108.14	•
Range:	30.16	29.69	34.90	36.76	39.87	40.34	•
Count:	20	20	20	20	20	20	•
Missing Cells:	0	0	0	0	0	0	•
Sum:	1479.05	1585.49	1644.93	1603.12	1678.27	1757.15	•
Sum of Squares:	110920.59	126869.46	136791.36	130422.49	142895.13	156606.88	•

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Subject	19	11499.351	605.229		
INTENSITY	1	676.794	676.794	43.545	.0001
INTENSITY * Su...	19	295.307	15.542		
EXERCISE	3	3165.746	1055.249	37.787	.0001
EXERCISE * Sub...	57	1591.789	27.926		
INTENSITY * EX...	3	238.076	79.359	10.114	.0001
INTENSITY * EX...	57	447.260	7.847		

Dependent: HR

Table of Epsilon Factors for df Adjustment
Dependent: HR

	G-G Epsilon	H-F Epsilon
INTENSITY	1.000	1.000
EXERCISE	.743	.846
INTENSITY * EXERCISE	.909	1.076

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

Least Squares Means Table
Effect: INTENSITY * EXERCISE
Dependent: HR

	Vs.	Diff.	Std. Error	t-Test	P-Value
LOW INTENSITY, HG	LOW INTENSITY, LE	-5.322	.886	-6.008	.0001
	LOW INTENSITY, HG/LE	-8.294	.886	-9.363	.0001
	LOW INTENSITY, REST	.632	.886	.714	.4781
	HIGH INTENSITY, HG	-6.204	.886	-7.003	.0001
	HIGH INTENSITY, LE	-9.961	.886	-11.245	.0001
	HIGH INTENSITY, HG/LE	-13.905	.886	-15.697	.0001
	HIGH INTENSITY, REST	.632	.886	.714	.4781
LOW INTENSITY, LE	LOW INTENSITY, HG/LE	-2.972	.886	-3.355	.0014
	LOW INTENSITY, REST	5.955	.886	6.722	.0001
	HIGH INTENSITY, HG	-.881	.886	-.995	.3239
	HIGH INTENSITY, LE	-4.639	.886	-5.237	.0001
	HIGH INTENSITY, HG/LE	-8.583	.886	-9.689	.0001
	HIGH INTENSITY, REST	5.955	.886	6.722	.0001
LOW INTENSITY, HG/LE	LOW INTENSITY, REST	8.927	.886	10.077	.0001
	HIGH INTENSITY, HG	2.091	.886	2.360	.0217
	HIGH INTENSITY, LE	-1.667	.886	-1.882	.0650
	HIGH INTENSITY, HG/LE	-5.611	.886	-6.334	.0001
	HIGH INTENSITY, REST	8.927	.886	10.077	.0001
LOW INTENSITY, REST	HIGH INTENSITY, HG	-6.836	.886	-7.717	.0001
	HIGH INTENSITY, LE	-10.593	.886	-11.959	.0001
	HIGH INTENSITY, HG/LE	-14.537	.886	-16.411	.0001
	HIGH INTENSITY, REST	0.000	.	.	.
HIGH INTENSITY, HG	HIGH INTENSITY, LE	-3.757	.886	-4.242	.0001
	HIGH INTENSITY, HG/LE	-7.701	.886	-8.694	.0001
	HIGH INTENSITY, REST	6.836	.886	7.717	.0001
HIGH INTENSITY, LE	HIGH INTENSITY, HG/LE	-3.944	.886	-4.452	.0001
	HIGH INTENSITY, REST	10.593	.886	11.959	.0001
HIGH INTENSITY, HG/LE	HIGH INTENSITY, REST	14.537	.886	16.411	.0001

variable	x2	xy	y2	b	(xy)2/x2	(y-y)2	df	F (3.10)
SBP								
C/40	8990.00	6489.06	4857.67	0.72	4683.86	173.81	28.00	
LE/40	8990.00	4272.77	2085.74	0.48	2030.76	54.98	28.00	
HG/40	8990.00	3333.46	1269.45	0.37	1236.04	33.41	28.00	
						262.20	84	3.12
pooled	26970	14095.3	8212.86	0.52	7366.60	846.26		
					846.26	584.06	2	292.03
								93.56
C/30	8990.00	4700.62	2546.22	0.52	2457.82	88.40	28.00	
LE/30	8990.00	2898.36	954.15	0.32	934.43	19.72	28.00	
HG/30	8990.00	2074.46	549.21	0.23	478.69	70.52	28.00	
						178.65	84	2.13
pooled	26970	9673.44	4049.58	0.35867	3469.61	579.97		
						401.32	2	200.66
								94.35

variable	x2	xy	y2	b	(xy)2/x2	(y-y)2	df	F
DBP								
C/40	8990.00	4825.38	2665.3	0.54	2590.02	75.28	28.00	
LE/40	8990.00	3280.02	1230.42	0.36	1196.72	33.70	28.00	
HG/40	8990.00	3131.14	1100.45	0.35	1090.55	9.90	28.00	
						118.88	84	1.42
pooled	26970	11236.5	4996.17	0.42	4681.49	314.68		
						195.80	2	97.90
								69.18
C/30	8990.00	3322.54	1264.31	0.37	1227.95	36.36	28.00	
LE/30	8990.00	1983.96	459.03	0.22	437.83	21.20	28.00	
HG/30	8990.00	1713.76	339.88	0.19	326.69	13.19	28.00	
						70.75	84	0.84
pooled	26970	7020.26	2063.22	0.26	1827.37	235.85		
						165.11	2	82.55
								98.02

PostHoc													
SBP 30		b1-b2	(xy)2/x2	(y2-e)	(xy2)/x2	(y2-g)	f+h	2df-4	i / j	1/8990	(k*l)1/2	d/m	t= 2.05
	A: HG vs LE	0.09	478.69	70.52	934.43	19.724	90.248	56	1.61	0.0002	0.0189	4.84	sd
	B: C vs HG	0.29	2457.82	88.40	478.69	70.52	158.92	56	2.84	0.0002	0.0251	11.63	sd
	C:C vs LE	0.20	2457.82	88.40	934.43	19.724	108.12	56	1.93	0.0002	0.0207	9.67	sd
DBP 30													
	A: HG vs LE	0.03	326.69	13.19	437.83	21.20	34.39	56	0.61	0.0002	0.0117	2.57	sd
	B: C vs HG	0.18	1227.95	36.36	456.63	13.19	49.55	56	0.88	0.0002	0.014	12.75	sd
	C:C vs LE	0.15	898.67	365.64	437.83	21.20	386.84	56	6.91	0.0002	0.0392	3.80	sd
DBP 40													
	A: HG vs LE	0.02	1090.55	9.90	1196.72	33.70	43.60	56	0.78	0.0002	0.0132	1.26	ns
	B: C vs HG	0.19	2590.02	75.28	1090.55	9.90	85.18	56	1.52	0.0002	0.0184	10.24	sd
	C:C vs LE	0.17	2590.02	75.28	1196.72	33.70	108.98	56	1.95	0.0002	0.0208	8.26	sd
SBP 40													
	A: HG vs LE	0.25	2030.76	54.98	4683.86	173.81	228.79	56	4.09	0.0002	0.0301	8.177	sd
	B: C vs HG	0.35	1236.04	33.41	4683.86	173.81	207.23	56	3.7	0.0002	0.0287	12.23	sd
	C:C vs LE	0.10	1236.04	33.41	2030.76	54.977	88.387	56	1.58	0.0002	0.0187	5.576	sd